Scholars Journal of Applied Medical Sciences
Abbreviated Key Title: Sch J App Med Sci
ISSN 2347-954X (Print) | ISSN 2320-6691 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Pediatrics

Anemia in Indian Children and Management

P. John Solomon[1], Margaret Chellaraj[2], A. Priya Margaret[3*]

[1]Professor and Director of Pediatrics, Sree Balaji Medical College, Chennai – 600044, Formerly Professor and H.O.D, Pediatric Hematology & Oncology, Government Stanley Medical College, Chennai – 600001
[2]Professor and H.O.D of Hematology, Madras Medical College, Chennai – 600003
[3]Assistant Professor of Pediatrics, Sree Balaji Medical College, Chennai – 600044

DOI: 10.36347/sjams.2021.v09i07.012 | **Received:** 06.06.2021 | **Accepted:** 09.07.2021 | **Published:** 17.07.2021

***Corresponding author:** A. Priya Margaret

Abstract **Review Article**

Anemia is defined as hemoglobin level of less than the 5th percentile for age [1]. It is a common condition in children seen in everyday practice. It has to be detected early to prevent complications. Anemia is more common in children below 2 years of age. Most children with mild anemia are asymptomatic. Hence, in many countries where the prevalence of anemia is very high it is common practice to screen for anemia at 1 year of age. Most cases of anemia in our country are caused by nutritional deficiency of iron, and or vitamins. Simple investigations and treatment are adequate to diagnose and manage most of the cases of anemia. Medical practitioners should have a thorough knowledge about the common causes of anemia and their management. Referral to a Pediatric Hematologist is required only in some cases. Before arriving at a conclusion we should compare the blood results with normal values for that age. Anemia in the newborn may be due to blood loss, hemolysis caused by blood group incompatibilities or abnormalities of the cell membranes of the RBCs or its enzymes. During infancy and childhood the commonest cause of Anemia is nutritional, of which iron deficiency is more common. Folic acid and B12 deficiency can also cause anemia. Thalassemia major and severe forms of hereditary spherocytosis cause anemia and jaundice requiring blood transfusion and iron chelation and folic acid therapy. Autoimmune hemolytic anemia is not common in children. In some inherited diseases like Thalassemia Hematopoietic Stem Cell Transplantation offers the best hope for cure.

Keywords: Deficiency, Transfusion, Hemolysis, Cardiac failure, Incompatibility, Hemoglobin.

INTRODUCTION

Anemia is defined as Hemoglobin level of less than the 5th percentile for age [1]. It can also be defined as a reduction of the hemoglobin concentration or Red Blood Cell volume below the range of values occurring in healthy persons [2].

The normal hemoglobin (Hb), Packed Cell Volume (PCV), and MCV values vary with age as follows.

Table 1

Variable	28 weeks	34 weeks	40 weeks	24 hrs	72 hrs	Range at term	1 month
Hemoglobin (g/dl)	14.5	15	16.8	18.4	17.8	15-20	14
Packed cell volume (%)	45	47	53	58	55	48-60	43
MCV (fl)	120	118	107	108	99	96-112	104
Reticulocytes (%)	10	10	7	7	3	3-10	1

Table-2

Age in years	Hemoglobin (g/dl)		PCV (%)		MCV (μM^2)	
	Mean	Lower limit	Mean	Lower limit	Mean	Lower limit
0.5 – 1.9	12.5	11	37	33	77	70
2 – 4	12.5	11	38	34	79	73
5 – 7	13	11.5	39	35	81	75
8 - 11	13.5	12	40	36	83	76
12 yrs	13.5	12	41	36	85	78

Rakhi Dandona has stated that in 2017 the prevalence of Anemia in India was 60% in children younger than 5 years [3] Lalitha Kailas *et al*. have stated in their article that in India the prevalence of Iron Deficiency Anemia in Children between 6 and 59 months is reported as 56% [4] According to the World Health organization (WHO) anemia is one of the ten most serious health problems in the World [7]. In 2011 WHO estimated that about 273.2 million children aged from 6-59 months in the world were suffering from anemia, with an overall prevalence of 42.6% [7]. In an article published by Sant-Rayer Pasricha of the Walter and Eliza Hall Institute of Medical Research it was reported that more than 75% of Indian toddlers are anemic [4]. In a study done in Bangalore it was observed that among 882 children of age group 6 months to 12 years 642 were anemic (72.79%) of which only 13 (2%) were hemoglobinopathies and 629 (98%) had other causes [11]. In a study done in US, among the 2446 children with Crohn's disease and Ulcerative colitis 85% had iron deficiency [12].

Anemia can be classified as mild (Hb < 11gm/dl), moderate (Hb < 9gm/dl) and severe (Hb< 7gm/dl) [9]. This article has been prepared with the main aim of educating and guiding the practitioners involved in primary care, and busy practice in various parts of India. Hence only the commonly seen anemias are discussed in some detail.

Anemia in Newborns

In the newborn period the following are the important causes of anemia

1. Blood loss

a) Causes: Iatrogenic, placental hemorrhage, injury to placental or umbilical vessels, fetomaternal transfusion, fetoplacental transfusion, twin to twin transfusion, obstetric trauma etc. The most common cause is blood loss due to repeated blood sampling [3, 2]. Hence it is important that the senior most specialists should make the decision on what tests to be done, and an expert in intravenous lines should draw the blood.
b) Clinical presentation: Pallor, tachypnoea, irritability, shock
c) Laboratory investigations: CBC (Hb low, MCV normal), reticulocyte count increased, positive Kleihauer Betke test in fetomaternal transfusion.

2. Increased RBC destruction

a) Causes: Immune destruction, congenital hemolytic anemia. Immune destruction: ABO incompatibility, Rh incompatibility.
b) Clinical presentation: Jaundice, anemia and in severe cases hydrops fetalis. ABO incompatibility is the most common cause of hemolytic disease of the fetus and new born (HDFN) [2].

Rh incompatibility

It is the severe form because of the highly immunogenic RhD antigen (mother Rh negative and fetus / baby Rh positive).

Antenatal diagnosis

If Rh negative mother has RhD antibody titers of more than 1:16 at any time during the subsequent pregnancy, the severity of fetal anemia should be monitored by Doppler ultrasonography of the middle cerebral artery.

Post-natal diagnosis

Blood from the umbilical cord or the infant should be tested for ABO blood group, Rh type, Hb, PCV, Reticulocyte count, Serum Bilirubin and the Direct Antiglobulin Test (DAT). A positive DAT indicates that there are maternal antibodies on the infant's RBCs.

Treatment

In utero, if the fetus is severely anemic or if there is hydrops, O negative, CMV negative, leukocyte poor and irradiated PRBC transfusion should be given into the umbilical vein of the fetus. The post transfusion PCV should be around 45 to 55%.

At birth a Cord Hb level of less than 10gm/dl or serum bilirubin level of more than 5mg/dl, and supportive factor of a reticulocyte count of ≥ 15% and prematurity are indications for exchange transfusion. Subsequently the Hb level and serum bilirubin level should be assessed every 6 hours to know the extent of hemolysis. If the serum bilirubin level is more than 20 mg / dl in a term infant, exchange transfusion should be done. IVIG can prevent the hemolysis and reduce the need for exchange transfusion. Rh immunoglobulin (Rho GAM) should be given to all mothers who are at risk for Rh alloimmunisation, within 72 hours of delivery of Rh+ve infant.

ABO incompatibility

In this condition usually the mother is O group and the infant is either A or B group. Rarely the mother is a group and the infant is B group or vice versa. The first born may be affected in ABO incompatibility unlike in Rh incompatibility where usually the 2nd born is affected. Less than 10% of at risk infants only develop clinical manifestation of hemolysis because the naturally occurring maternal antibodies against ABO blood group antigens are almost always IgM and hence do not cross the placenta. Rarely group O mothers can produce IgG antibodies which cross the placenta and produce hemolysis. IVIG administration may reduce the rate of hemolysis and hence the need for exchange transfusion rarely arises. When exchange is needed we should use group `O` Rh compatible blood.

3. Congenital hemolytic anemia

a) Causes: Hereditary spherocytosis (HS), G6PD Deficiency, Pyruvate Kinase deficiency, etc.
b) Clinical presentation: Anemia, jaundice, splenomegaly
c) Laboratory investigations: Peripheral smear examination is very useful to identify the cause of hemolysis. In Hereditary spherocytosis we can see microspherocytes, and in G6PD deficiency bite cells and Heinz bodies.
d) Treatment: Packed red cell transfusion (PRBC) to maintain Hb ~ 10 gm%

4. Decreased RBC Production

a) Causes: Anemia of prematurity, Rubella / CMV / Parvovirus B19 infection, Diamond Blackfan Anemia etc.
b) Clinical Presentation: Pallor, irritability.
c) Laboratory investigation: CBC (MCV normal), Reticulocyte count (low)
d) Treatment: Each condition needs different treatment. Preterm babies may require PRBC transfusion.

Anemia beyond the Newborn period

The normal levels of Hb, PCV and MCV beyond neonatal period are given in Table 2. Ideally all the children should be screened for anemia at 1 year of age. If the Hemoglobin is low, further evaluation must be done to determine the cause of anemia. Most children are asymptomatic when Hb is >7 gm%

The first line investigations are CBC, reticulocyte count and Peripheral smear examination. CBC provides important information like Hemoglobin level, Packed cell volume, White cell count, Platelet count, Mean Corpuscular Volume (MCV) and others.

Reticulocyte count reflects the capacity of bone marrow to compensate for the anemia.

$$\text{Reticulocyte index} = \text{Reticulocyte \%} \times \frac{\text{Observed hematocrit}}{\text{Normal hematocrit}} \times \frac{1}{\mu}$$

where μ is maturation factor of 1 – 3 related to the severity of anemia. The normal reticulocyte index is 1.

Physiologic Anemia of Infancy

The hemoglobin drops to 9 to 11gm/dl at 6 to 8 weeks in term infants and 7 to 9 gm/dl in preterm infant due to decrease in HbF and increase in Hb A and decrease in erythropoietin production. The life span of RBCs in preterm infants is less than 60 days. Preterm infants may occasionally require transfusion. Recombinant human erythropoietin is an alternative.

Anemia can be classified based on the size of RBCs as follows:

Microcytic (MCV low = < 70)		**Normocytic (MCV Normal=70- 90)**		**Macrocytic (MCV high = > 90)**	
Retic count low	Retic count high	Retic count low	Retic count high	Retic count Low	Retic count high
1.Iron deficiency. 2.Thalassemia trait. 3.Chronic disease	Thalassemia Major/Intermedia	1.Chronic disease. 2.Pure red cell aplasia. 3.Malignancy. 4.Hypersplenism. 5.Infection 6.Transient erythroblastopenia of childhood	1.Hereditary spherocytosis. 2.G-6-P-D deficiency.	1.Folate, B12 deficiency. 2.Aplastic Anemia. 3.Fanconi anemia. 4.Diamond Blackfan syndrome 5.Drug induced. 6.Hypothyroidism. 7.Liver disease.	Autoimmune hemolytic anemia

Microcytic Anemia

In this anemia the size of the red blood cells are small (MCV= < 70 fl). Commonest cause of microcytic anemia in children is iron deficiency.

Iron deficiency anemia (1DA)

Iron deficiency is the most common micronutrient deficiency worldwide affecting 25% of the world's population according to World Health Organisation. Young children are at high risk for developing IDA because of the poor bioavailability of iron in their diet and the increased demand. During the most active period of brain development of the young children if they develop iron deficiency, the risk for irreversible brain damage adds to the concern [14].

In a study done in Kerala, among the 160 children belonging to upper middle class evaluated 30% were anemic; of which 85.4% had IDA [5]. In a study done in Israel it was observed that children with extremely low red meat consumption had 4 fold higher rates of iron deficiency than those who consumed that 2 times per week [6].

a) Causes

Diet poor in iron content, gastrointestinal blood loss, hook worm infestation, cow's milk protein allergy, low birth weight, prematurity, obesity, etc.

b) Clinical manifestation

Most of the children with mild IDA are asymptomatic. Infants with IDA have delayed

cognitive, motor and affective development that may be long lasting [13]. There is also an increased risk of breath holding spells, pica, seizures and strokes and decreased linear growth due to defective IGF-1 secretion [13].

c) Laboratory investigations

CBC, Reticulocyte count and Peripheral smear study. The Hb, PCV, MCV, MCH, MCHC, RBC count etc. are reduced. There may be reactive thrombocytosis. RDW is increased because the cell size is variable.Serum ferritin (iron storage protein) is low. Then serum iron level decreases, Iron binding capacity (serum transferrin) increases, and the transferrin saturation falls below normal. There is increased soluble transferrin receptor and decreased reticulocyte hemoglobin content (RHC). Hb estimation fails to detect early iron deficiency because it reflects iron content of previous 120 days [1]. Reticulocytes survive in the periphery for 1-2 days and hence RHC of less than 27.5 pg is 83% sensitive in detecting iron deficiency, and is a better real time measurement of bone marrow iron stores [1]. Bone marrow aspiration for iron staining involves an invasive procedure and hence it is not recommended.

IDA should be differentiated from Thalassemia trait as follows.

IDA	**Thalassemia trait**
RDW increased	RDW normal
Mentzer index >13	Mentzer index <13 Ref [1]

Mentzer index is the ratio of MCV (fl) divided by RBC count (x 10^6 per mm^3) [1]. In Thalassemia trait there is erythrocytosis unlike in iron deficiency and hence the ratio is reduced. Anemia of chronic disease can also present as microcytic anemia. Here the iron binding capacity is not increased.

d) Prevention

1. Encourage exclusive breast feeding upto 6 months of age (less iron but absorption is 3 times more compared to other milks). Preterm breastfed infants should receive an iron supplement from 4 weeks of age. Additional source of iron should be given to infants starting from 4 months of age, first as an iron supplement, followed by iron fortified foods. Partially breastfed and non-breastfed infants should consume exclusively iron fortified formulas [15].
2. Infants who are not breast fed can be given iron fortified formula for the 1st year. After 1 year cow`s milk should be limited to less than 24 ounces per day [1]. Heme iron (meat and fish) is more bioavailable than non heme iron (vegetables and cereals). Hence heme iron is absorbed better [15].
3. Since 1mg of iron is required daily and 10% of the dietary iron is absorbed, about 10mg of iron should be available daily in food.
4. Delay clamping of the umbilical cord for 1-3 minutes. This helps to prevent anemia in the neonatal period and early infancy.

e) Treatment

Oral ferrous salts are given (eg Ferrous sulfate) if possible with vitamin C or fruit juice to enhance absorption. Avoid tea and diet rich in calcium and fibre which decrease the iron absorption. If there are gastrointestinal complaints the dosage can be reduced. The therapeutic dose of elemental iron is 3-6mg/kg in 2 divided doses. Ferrous sulfate has 20% of elemental iron, and it should be given in between meals. Intravenous preparations like iron sucrose, ferricarboxymaltose may be given if there is malabsorption. High doses of iron may worsen infection with gram negative bacteria or yersinia. Iron from heme sources is 10 times more bioavailable than from non heme sources. After starting iron, there will be reticulocytosis within 96 hours, and the hemoglobin will increase by 0.1-0.4g/dl/day. In 1 month time the rise of hemoglobin should be at least 1gm/dl. Iron stores should be replenished by continuing iron therapy for 3 months after the hemoglobin reaches normal levels.

Only if there is congestive heart failure or imminent failure, packed red cells can be infused very slowly in quantities of 3-5ml/kg. If the patient takes iron regularly, the stool will be slightly dark coloured. So, if the patient says that the colour of the stool is normal while on oral iron therapy, we have to suspect noncompliance. The teeth may also be stained with iron, and it will disappear sometime after discontinuing the drug.

Normocytic anemia

In these patients the RBCS have normal size (MCV 70 – 90 fl). A low reticulocyte count in normocytic anemia indicates infection, anemia of chronic disease, transient erythroblastopenia of childhood, leukemia etc. A peripheral blood smear examination is important to rule out leukemia. A high reticulocyte count indicates hemolysis. Eg. Congenital hemolytic anemia due to membrane disorders, enzyme deficiency, etc.

a) Anemia of chronic disease

It was observed in a study in U.S that anemia was secondary to infection in about half of the cases [8]. In Chronic infection/ inflammation the anemia is usually normocytic normochromic, but it can be microcytic and hypochromic as well. In these cases the RBC survival is reduced by 30%. Also there is ineffective bone marrow response and decreased production of erythropoietin.[9] The other mechanisms are that the absorption of iron from the intestine decreases. Moreover the macrophages easily destroy the red cells. They also bind to the iron and thus prevent the bone marrow from utilising the iron.

Treatment

Management of the underlying disease will lead to correction of the anemia in about 3 months. Recombinant human erythropoietin can be administered.

b) Transient Erythroblastopenia of Childhood

Here there is moderate to severe normocytic anemia with reticulocytopenia following a viral infection in a child 6 months to 3 years of age. Bone marrow aspirate will show reduced RBC precursors. RBC adenosine deaminase levels are normal (in congenital DBA it is increased). Spontaneous recovery in 2 months is the usual outcome. Steroids are not helpful.

Macrocytic anemia

In these children the RBC size is large (MCV > 90 fl).

a) Causes

Vitamin B12/ folic acid deficiency

Vitamin B12/ folic acid deficiency, Diamond Blackfan syndrome, Liver disease, Hypothyroidism, intake of anticonvulsants like phenytoin and phenobarbitone, chronic hemolytic anemia, drugs like Valproic acid, pyrimethamine, methotrexate, trimethoprim etc. Aplastic anemia, and post splenectomy reticulocytosis can also present with macrocytosis. B12 and Folic acid deficiency can be caused by dietary and other factors. Drugs like phenytoin, valproic acid, pyrimethamine, methotrexate, trimethoprim, etc can cause folic acid deficiency. In a study in Mexico it was observed that there was preponderance of other cause related anemia over iron deficiency related anemia [10]. Cobalamine is synthesized only by microorganisms, and human beings have to rely on animal products like meat, eggs, fish and milk.

b) Clinical presentation

Pallor is the usual complaint. Neurological manifestations may also be there such as paresthesia, sensory deficits, hypotonia, seizures, developmental delay, and regression of mile stones, and neuropsychiatric changes which may occur in the absence of any hematological abnormalities.

c) Laboratory investigations

Macroovalocytosis of RBCs, and hypersegmented large sized polymorphs are usually seen in the peripheral smear. Neutropenia and thrombocytopenia can also occur mimicking aplastic anemia. Serum B12 and folate assay, and RBC folate assay can help us to come to the correct diagnosis. LDH is increased. Pernicious anemia can be ruled out by detection of antiparietal antibodies and antiintrinsic factor antibodies. Bone marrow aspiration will show megaloblasts.

d) Treatment

Folic acid should not be given in undiagnosed megaloblastic anemia unless vitamin B12 is administered concurrently, otherwise neuropathy may be precipitated (Sub acute combined degeneration of the Spinal cord). From 1 month to1 year of age the dose is 500 microgram/kg once daily for 4 months. From 1 year to 18 years 5 mg of oral folic acid once daily is given for 4 months. In hemolytic anemias the dosage is 5 mg once daily [16].

In B12 deficiency, hydroxocobalamin can be given intramuscularly. From 1 month to 18 years of age the dosage is 250 microgram to 1 mg 3 times a week for 2 weeks, then 250 microgram once weekly until blood count is normal and then 1 mg every 3 months [16]. The maintenance treatment is usually given lifelong depending upon the cause.

Diamond Blackfan Anemia

a) Clinical presentation: This is a congenital pure red cell aplasia. Usually anemia is seen before 6 months of age. Many structural abnormalities such as hypertelorism, high arched palate, skeletal abnormalities, cardiac defects etc are also seen in this condition.
b) Laboratory investigations: The MCV is high. There is reticulocytopenia, and reduced or absent RBC precursors in the bone marrow aspirate. The HbF may be slightly elevated. RBC adenosine deaminase activity is also increased.
c) Treatment: PRBC transfusion may be given until 1 year of age. After that low dose steroids can be given. Hematopoietic stem cell transplantation offers cure from this illness. The sibling donor is screened to make sure that he does not carry the DBA gene.

Hemolytic Anemia

a) Causes:

1. Intra corpuscular defect - RBC membrane, enzyme or haemoglobin defects.
2. Extra corpuscular defects – eg. Autoimmune haemolytic anemia.

b) Clinical presentation: Anemia, jaundice, hepatosplenomegaly, gallstones, growth retardation.

c) Laboratory investigations: CBC, reticulocyte count (↑), peripheral blood smear examination, direct antiglobulin test, bilirubin (indirect ↑), serum LDH, Haptoglobin. A thorough examination of peripheral smear is important. The following may be observed in the smear.

Observation	**Diagnosis**
Microcytic RBCs	Thalassemia
Microspherocytes	Hereditary Spherocytosis
Sickle cells	Sickle Cell Anemia

Bite cells	G6PD deficiency
Schistocytes	TTP, Microangiopathy

Among all the hemolytic anemias seen in India, thalassemia and hereditary spherocytosis are common.

Thalassemia syndromes

This is an autosomal recessive disease. β Thalassemia major is a severe form of the disease, and they need early intervention with blood transfusion. β Thalassemia intermedia clinically takes an intermediary course and does not require frequent transfusion like the major cases. β Thalassemia trait cases are asymptomatic, but may have mild microcytic hypochromic anemia.

a) Clinical presentation

Pallor, mild jaundice and hepatosplenomegaly are usual presentations. In thalassemia major pallor appears usually around 6 months of age (normally around 6 months of age Hemoglobin a level goes up and HbF level goes down, but in β thalassemia major it does not happen). Maxillary hyperplasia, flat nasal bridge, frontal bossing, pathological bone fracture etc may be seen in patients who are not adequately transfused for a long period.

b) Laboratory investigations

The Hb is low; MCV & MCH are also low. Nucleated RBCs may be seen in the peripheral blood smear, and the reticulocyte count is around 5-8%. For the degree of anemia it is low because of ineffective erythropoiesis. Hemoglobin electrophoresis will confirm the presence of high level of Fetal haemoglobin (HbF) and low level of Adult haemoglobin (HbA). HbA2 may be slightly raised.

c) Treatment

Packed red blood cell transfusion (PRBC) is given usually every 3 - 4 weeks so that the Hb is maintained at 9-10 mg/dl prior to transfusion, and iron chelation if the serum ferritin level goes above 1000 ng/ml, and hence it has to be started after 12-15 transfusions. The PRBCs should be CMV Negative, leukocyte poor, and irradiated. Quantitative measurement of liver iron and cardiac iron by MRI is possible. Oral iron chelators like deferazirox or deferiprone can be given. In patients with severe iron overload, chelation with more than one drug may be required. Desferioxamine can be given intravenously or subcutaneously with the help of an infusion pump. Subcutaneous administration is for 8 hours in the night daily for 5-6 days a week. Deferiprone is given 3 times a day and it easily enters the cardiac tissue and reduces cardiac hemosiderosis. Deferazirox is given once a day. Hydroxyurea may be useful in some cases of thalassemia intermedia. All the patients should receive oral folic acid.

Hematopoietic stem cell transplantation is the only hope for cure in a case of thalassemia major. With this procedure, the overall survival is more than 90% if we get a suitable sibling donor with complete HLA match.

Splenectomy may be required in patients who develop hypersplenism showing pancytopenia and Splenomegaly, requiring more frequent transfusions. But atleast 2 weeks before doing splenectomy vaccinations for Meningococcus, Haemophilus influenza & Pneumococcus (capsulated organisms) should have been completed. If possible splenectomy may be postponed until 6 yrs of age. After splenectomy patient should be on prophylaxis with penicillin lifelong to prevent post splenectomy sepsis.

d) Prevention

Avoid consanguineous marriages. Affected mothers can be given genetic counselling, and should be told about the possibility of antenatal diagnosis during future pregnancies if they want to have more babies (amniocentesis, cordocentesis or chorionic villous sampling).

Hereditary Spherocytosis

a) Clinical presentation

This is the most common inherited abnormality of the red blood cell membrane usually having autosomal dominant inheritance. Family history of gall stones may be there. This may present with chronic pallor, jaundice and splenohepatomegaly. The severity varies and there are mild, moderate and severe cases, presenting at various ages. Severe cases may present in the newborn period itself with pallor and jaundice.

b) Laboratory investigations

Hb is low. MCV is normal, but MCHC is high. Retic count is increased. Indirect hyperbilirubinemia may be present. Peripheral blood smear will show microspherocytes. Osmotic Fragility of RBCs is increased. Other tests available are flowcytometric study of binding of florescence labelled EMA (eosin-5-maleimide) to band 3, which is decreased in HS erythrocytes, and cryohemolysis test. Acidified glycerol lysis test and osmotic gradient ektacytometry have increased sensitivity for hereditary spherocytosis.

c) Treatment

Oral folic acid 5 mg daily, and PRBC transfusion to maintain the Hb level at about 10gms%. Splenectomy may be required in moderate and severe cases getting frequent hypoplastic or aplastic crisis, poor growth or cardiomegaly or requiring very frequent transfusions with hypersplenism. Partial splenectomy is preferable. At least 2 weeks before splenectomy the patient should have completed taking vaccines for Pneumococcus, Meningococcus, and Hemophilus B influenza. After splenectomy they should be on lifelong

prophylaxis with penicillin. As far as possible splenectomy should be postponed until 6 years of age.

Autoimmune Hemolytic anemia

a) Clinical presentatation: Children present with fever, pallor, jaundice, and splenomegaly usually following a viral infection.
b) Laboratory investigations: Hb is low, and there is reticulocytosis. Peripheral blood smear will show spherocytes which are large sized. Indirect bilirubin will be high. Direct antiglobulin test (coomb's test) is usually positive
c) Treatment: Blood transfusion may save lives. It may not be possible to get a complete cross match because of the interference by antibodies, and hence we have to transfuse the least incompatible blood. Corticosteroids may be lifesaving.

Sickle cell anemia

This is rare in India.

a) Clinical Presentation: This usually presents with painful crisis due to occlusion of blood vessels in the hands and feet following exposure to hypoxic situations (eg. dehydration, hypoxia). They can present with chest pain and pneumonia following infection.
b) Laboratory investigations: CBC, Peripheral smear study (Sickle cells+) and Hemoglobin Electrophoresis (Hb S).
c) Treatment: Avoiding the precipitating factors for painful crisis (Dehydration, hypoxia etc). Hydroxyurea can reduce the frequency of painful episodes. Chest pain and pneumonia require adequate hydration, antibiotics and oxygen.

CONCLUSIONS

Anemia is not a diagnosis, but a manifestation which warrants further evaluation. With increased awareness among the public and the health personnel and improved knowledge and expertise in this area, the mortality and morbidity due to anemia can be brought down markedly in the near future. It is important for the practitioner to have atleast the basic knowledge about the common causes of anemia and their presentation and management. Delay in diagnosis can cause significant morbidity including neurocognitive dysfunction. In this article we have discussed only the common causes of anemia and their management. Any difficult cases may be referred to a Pediatric Hematologist without any delay.

REFERENCES

1. Janus, J., & Moerschel, S. K. (2010). Evaluation of anemia in children. *American family physician*, *81*(12), 1462-1471.
2. Nelson Text book of Pediatrics, 21st edition, International edition, Volume 2, Copy right © 2020 Elsevier Inc.
3. Dandona, R. (2020). Addressing different types of anaemia in Indian children and adolescents. *The Lancet. Child & adolescent health*, *4*(7), 483-484.
4. Kailas, L., Umair, C. H., & Sankar, V. H. (2020). An Untold Tale of Iron Deficiency Anemia. *Indian pediatrics*, *57*(6), 575-576.
5. John, J. J., Mohan, G., Ajitha, K., & David, A. (2019). Iron deficiency anemia among preschool children belonging to affluent families in Kerala, India. *Journal of Current Research in Scientific Medicine*, *5*(1), 23.
6. Moshe, G., Amitai, Y., Korchia, G., Korchia, L., Tenenbaum, A., Rosenblum, J., & Schechter, A. (2013). Anemia and iron deficiency in children: association with red meat and poultry consumption. *Journal of pediatric gastroenterology and nutrition*, *57*(6), 722-727.
7. Nambiema, A., Robert, A., & Yaya, I. (2019). Prevalence and risk factors of anemia in children aged from 6 to 59 months in Togo: analysis from Togo demographic and health survey data, 2013–2014. *BMC public health*, *19*(1), 1-9.
8. Baty, J. M. (1947). Classification of anemia in infants and children. *Journal of the American Medical Association*, *134*(12), 1002-1003.
9. George, R. (1996). Buchanan, The Pediatric clinics of North America, *Pediatric Hematology*, *43*(3).
10. Onyeneho, N. G., Ozumba, B. C., & Subramanian, S. V. (2019). Determinants of childhood anemia in India. *Scientific reports*, *9*(1), 1-7.
11. Saba, F., Poornima, S., Balaji, P. A. R., Varne, S. R. R., & Jayashree, K. (2014). Anemia among hospitalized children at a multispecialty hospital, Bangalore (Karnataka), India. *Journal of family medicine and primary care*, *3*(1), 48.
12. Miller, S. D., Cuffari, C., Akhuemonkhan, E., Guerrerio, A. L., Lehmann, H., & Hutfless, S. (2019). Anemia Screening, Prevalence, and Treatment in Pediatric Inflammatory Bowel Disease in the United States, 2010–2014. *Pediatric gastroenterology, hepatology & nutrition*, *22*(2), 152-161.
13. Soliman, A., De Sanctis, V., & Elalaily, R. (2014). Nutrition and pubertal development. *Indian journal of endocrinology and metabolism*, *18*(Suppl 1), S39.
14. Siegel, E. H., Stoltzfus, R. J., Khatry, S. K., Leclerq, S. C., Katz, J., & Tielsch, J. M. (2006). Epidemiology of anemia among 4-to 17-month-old children living in south central Nepal. *European journal of clinical nutrition*, *60*(2), 228-235.
15. Jelena, R., & Ksenija, S. (2018). Iron deficiency Anemia in children, Current topics in Anemia, Intech open 69774, February 7th 2018.
16. BNF for children
17. Henry, L. Halliday, Garth McClure & Mark Reid, Hand book of Neonatal Intensive care, 3rd edition.
18. Pasricha, S. R., Black, J., Muthayya, S., Shet, A., Bhat, V., Nagaraj, S., ... & Shet, A. S. (2010). Determinants of anemia among young children in rural India. *Pediatrics*, *126*(1), e140-e149.