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A RETROSPECTIVE STUDY OF MORPHOLOGICAL & NUTRITIONAL ANEMIA IN SEVERE ANEMIC CHILDREN IN TERTIARY CARE RURAL HOSPITAL OF WESTERN MAHARASHTRA.

Paediatric Medicine

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ABSTRACT

Background- Nutritional anemias develop when the hematopoietic nutrients required for hemoglobin synthesis and/or maintenance are insufficient to meet the demands of an individual. The vast majority of nutritional anemias are due to the deficiency of iron, vitamin B12 (cobalamin, Cbl) and/or folic acid. Additionally, nutritional anemias have a deleterious impact on the physical strength of the individuals as well as decreased productivity and economic loss to the country. **Methods:** The present retrospective study was conducted in department of Paediatrics, Dr.VitthalraoVikhe Patil Pravara Rural hospital Loni, Maharashtra, India for a period of 2 years (September 2015 to September 2017).All patients in the age group 6 months to 12 years with hemoglobin<7 gm /dl were included in the study. Previously diagnosed hemolyticanemias were excluded from the study. **Results:** Out of total 300 cases of severe anemia, nutritional deficiency anemia is predominant which is in79.3% cases, hemolyticanemia in 14.3% cases while in 6.3% cases had varied etiology. Among Nutritional anemia cases in this study, predominantly iron deficiency anemia is in 48.3%, followed by dimorphic anemia and Megaloblastic anemia in 21.8% and 20.6% respectively.There is statistically no significant ($p>0.05$) difference of the type of Nutritional anaemia in between male and female cases. **Conclusion:** Children are more prone to nutritional anemia especially iron deficiency anemia with no sex preponderance.A combination of nutritional supplementation and food fortification programmes, as well as maternal anemia reduction efforts may translate into optimal improvement in the hemoglobin levels of the children.

KEYWORDS

Nutritional anemia, Iron-deficiency anemia, paediatric

INTRODUCTION

Anemia is a very common hematological disorder in infancy. It is ranked as the chronic malady of mankind affecting approximately 30-40% of the world population. In jfldia and other developing countries, incidence of nutritional anemia is as high as 60- 80% of the childhood population.[1]

The normal hemoglobin distribution varies with age and gender. There is also evidence of gender influence. The correct interpretation of hemoglobin or hematocrit values require the consideration of modulating factors in selecting appropriate cut off values. The WHO guidelines of hemoglobin and hematocrit levels below which anemia are present in a population is given as:-

Age or gender group		Hb.gm.	Hematocrit2
1.	Children 6month to59month	llgm%	68.3%
2.	Children 5years to 1 1 years.	11.5gm%	71.3%
3.	Children 12years to 14years.	12gm %	74.5%

Hemoglobin level bellow 7gm/dl is considered severe anemia, 7 to 9.9gm/dl moderate and 10 to 10.9gm/dl as mild anemia.[3]

Iron deficiency is the most common cause of anemia in infancy, while inherited disorders of hemoglobin synthesis, the Hemoglobinopathies form, by far the largest group of genetically determined anemia.[4]There is considerable sex difference in iron status during infancy. Boys are at 10 time higher risk of being classified as having iron deficiency anemia at 9 months.[5]

Iron deficiency anemia (IDA) is the most frequent hematological disorder of childhood and adolescence and the most common form of anemia, with an incidence in industrialized countries of 20.1% between 0 and 4 years of age and 5.9% between 5 and 14 years (39 and 48.1% in developing countries) [6]

Iron is an essential nutrient for the development of the fetus, infant, and child [7] The body's iron content is dependent on its intake and absorption with nutrition. The homeostasis of this nutrient is determined by the balance between its uptake and release from the cells where it is stored and recycled [8,9] Iron is released into the circulation, where it is carried by the plasma protein transferrin, into the duodenum by enterocytes that absorb dietary iron, and by macrophages which recycle senescent erythrocytes and liver reserves [8].

If iron levels in the body are inadequate, its intestinal absorption is enhanced [10]; in case of excess, it is stored in the enterocytes as ferritin and the liver, spleen, and bone marrow as hemosiderin [11]. The release of free iron ions in the plasma, essential for the maintenance of its homeostasis, is mediated by ferroportin, whose expression is subordinated to the activity of hepcidin [12]

Causes of iron deficiency anemia (IDA).

Prematurity
Low birth weight
Celiac disease
Helicobacter pylori infection
Cow milk protein intolerance

Clinical Features of Iron Deficiency

Mild to moderate iron deficiency, not associated with anemia, can be asymptomatic or lead to fatigue and/or poor tolerance to exercise [13]. Typical clinical presentation of moderate to severe iron deficiency includes the usual signs of anemia, such as pallor and fatigue. More specifically, restless leg syndrome, mucus trophic lesions (stomatitis, glossitis), pica, frequent infections, mood, and behavior disorders with a decline in school performance may occur.

Megaloblastic anemia:-

is caused by a deficiency of vitamin B12 and folate in 95% of cases. , Rarely, it can be caused by inborn errors of metabolism of vitamin B12 and folate ,It is named because of the classical presence of megaloblasts in bone marrow and macrocytes (large, structurally abnormal, and immature red blood cells) in the blood. These are formed as a result of asynchronous maturation between the nucleus and cytoplasm due to defective deoxyribonucleic acid (DNA) synthesis[14]

Clinical Features:-

Lethargy, easy fatigability, anorexia, Pallor and sallow complexion, Glossitis, Hyperpigmented nail beds and knuckles, Neurological manifestations—infantile tremor syndrome, hypotonia, paresthesias, seizures, and delayed development; Petechiae and other hemorrhagic manifestations, Hepatosplenomegaly (30–40% of cases) mimicking acute leukemia Clinical Features,Thrombosis may occur in cases with hyperhomocysteinemia[15,16]

METHODS:

The present retrospective study was conducted in department of Paediatrics, Dr.VitthalraoVikhe Patil Pravara Rural hospital Loni, Maharashtra, India for a period of 2 years (September 2015 to September 2017). All patients in the age group 6 months to 12 years with hemoglobin<7 gm /dl were included in the study. Previously

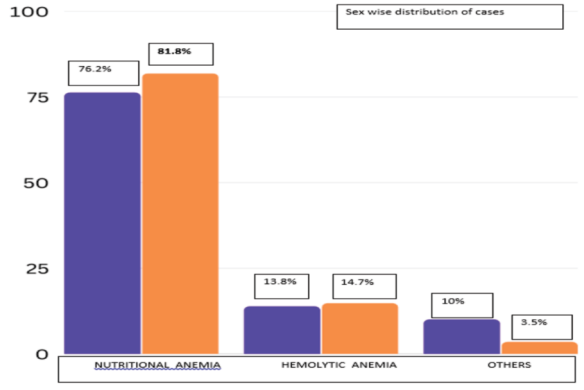
diagnosed hemolyticanemias were excluded from the study.

Data collection for this study included a retrospective review of hospital medical records of all children aged 6months to 12 years who were admitted to paediatric ward . Laboratory investigations reports such as complete hemogram, peripheral blood smear, reticulocyte count, osmotic fragility, sickling test, Hb - electrophoresis, etc. and renal function tests, liver function tests were noted. Various data on demographic, nutritional status and clinical profile were noted.

RESULTS

Distribution according to Physiological types

Sex	Physiological Type			
	Nutritional	Haemolytic	Others	Total
Male	99(76.2%)	18 (13.8%)	13 (10%)	130
Female	139(81.8%)	25(14.7%)	6(3.5%)	170
Total	238 (79.3%)	43(14.3%)	19(6.3%)	300

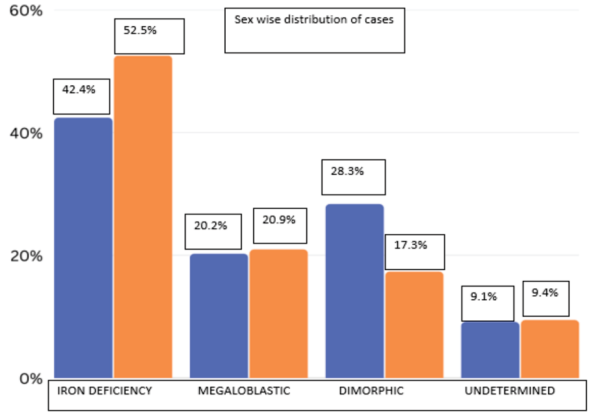


- Out of total 300 cases nutritional deficiency anemia is predominant which is in 79.3% cases, hemolytic anemia in 14,3% cases while in 6.3% cases had varied etiology of anemia like physiological anemia of infancy, Anemia of prematurity hemolytic uremic syndrome, malaria.

'In relation of sex, in male cases there is predominant nutritional anemia in 76.2%; hemolytic anemia in 13.8% and 10% had other cause for anemia. Within 170 females, predominantly 81.8% had nutritional anemia and 14.7% had hemolytic anemia.

There is statistically no significant (p>0.05) difference of type of anaemia in between both the sex in the study.

Sex	Nutritional Anaemia				
	Iron deficiency	Megaloblastic	Dimorphic	Undetermined	Total
Male	42 (42.4%)	20(20.2%)	28 (28.3%)	9(9.1%)	99
Female	73 (52.5%)	29 (20.9%)	24(17.3%)	13(9.4%)	139
Total	115 (48.3%)	49(20.6%)	52 (21.8%)	22 (9.2%)	238



In Nutritional anemia cases in study, predominantly iron deficiency anemia is in 48.3%, followed by dimorphic anemia and Megaloblastic anemia in 21.8% and 20.6% respectively.

- Within the sex of cases, similar predominance of Iron deficiency anemia is in 42.4% were Male and 52.5% female followed by dimorphic anemia in 28.3% Males and 17.3% Females while megaloblastic in 20.2% male and 20.9% females.The cause of Nutritional anemia is undetermined in 9.1% male and 9.4% female cases.There is statistically no significant (p>0.05) difference of the type of Nutritional anaemia in between male and female cases.

DISCUSSION:

Study	Iron deficiency	Megaloblastic	Dimorphic	Undetermined
Hazra B.R., Hazra S.C et al100	54.1%	14%	10%	-
Bramhagen A.C. and Axelsson I et al101	17%	-	-	-
Viswanath D., Hedge R et al20	89%	7%	2%	-
PRESENT STUDY	115 (48.32%)	49 (20.59%)	52 (21.85%)	22 (9.24%)

Out of 300 cases 238 cases were of nutritional anemia and out of this iron deficiency was the cause in 115 patients (48.32%). Combined nutritional deficiency presenting as dimorphic anemia constituted 52 cases (21.85%) followed closely by megaloblastic anemia 49(20.59%). In remaining 22 cases (9.24%) cause of nutritional anemia could not be found out.

Various studies have also found that iron deficiency anemia is the commonest nutritional anemia. Hazra B.R., Hazra S.C. et al[19] in their study of 560 cases of anemia found that 54.1% of all anemias were due to iron deficiency. Bramhagen A.C. and Axelsson I.[101] In their study of iron status of children in Sweden found that 7% of the children had iron deficiency anemia and 10% had iron deficiency without anemia. Findings in our study are in concordance with that of, study of frequency cause and significance of iron deficiency for the children of central Asia by Scrimshaw Nevin.[17]

Viswanath D., Hedge R.[18] found iron deficiency anemia in 89% of cases. This high percentage of IDA as compared to present study may be because of smaller i.e. 6 months to 5 years age group and larger sample size in the above study, as compared to present study of children.

Majority of cases of anemia were due to nutritional deficiency remaining cases were of hemolytic variety and others which included septicemia, leukemias, aplastic anemia, pure red cell aplasia and those whose cause could not be established.

Amongst nutritional anemia, iron deficiency was the commonest, followed by dimorphic and megaloblastic anemia.

CONCLUSION:-

Anemia is still prevalent among children in India and requires urgent attention. Iron and nutritional supplementation are necessary but not enough to significantly produce the necessary reduction in the incidence of childhood anemia. On the contrary, a combination of nutritional supplementation and food fortification programmes, as well as maternal anemia reduction efforts with alleviation of family poverty may translate into optimal improvement in the hemoglobin levels of the children.

Older infants (>6 months) and toddlers are at high risk of nutritional anemia due to inadequate and poorquality complementary diet. Therefore, screening for anemia using hemoglobin estimation should be done at 9-12 months when these infants visit for measles vaccination. This contact point should be utilized to assess the adequacy of complementary food intake and provide appropriate counselling if required. Further, daily iron supplementation in a dose of 1-2 mg/ kg/day can be considered in them individually, especially where dietary intake is inadequate. In young children and adolescents, additional screening may be done at any time if there is clinical suspicion or risk factor for anemia secondary to nutritional or a nonnutritional cause. Additionally, siblings of any child diagnosed with IDA must also be assessed for ID and managed accordingly. Daily iron supplementation is also suggested for adolescent children, especially girls (60-100 mg elemental iron), to replenish the iron stores for pubertal growth and development .

Mild to moderate macrocytic anemia due to dietary vitamin B12 deficiency can be treated with oral vitamin B12. However, patients

with neurological manifestations, those with pancytopenia, severe anemia, or malabsorption warrant parenteral vitamin B12 therapy.

Providing conditions conducive to reducing the occurrence of inflammatory and infectious processes, improving nutritional status by ensuring that the intake of essential nutrients is adequate for growth, and guaranteeing wider access to health programs and services can help to lower the risk of nutritional anemia in other case have serious consequences for the pediatric population.

REFERENCES-

1. Lokeswar MR , Shah Nitin et al. Approach to a child with anemia. Pediatric clinics of India; Jan 1996. 51 - 62.
2. WF10/NHD/01.3. Iron deficiency anemia, assessment prevention and control. A guide for programme manager 33.
3. DeMaeyer EM, Dallamn P et al. Preventing and controlling Iron deficiency anemia through primary health care. Geneva. World Health Organization, 1989; pp 8 - 9.
4. Fleming AF. Hematological diseases in the Tropics. /?? Gordon C(ed); Manson's Tropical diseases. W.B. Saunders.London.20th ed.1996; pp. 118-130.
5. Domellof et al. Sex differences in iron status during infancy; Pediatrics Vol. 110 No. 3 September 2002; 545 - 552.
6. *Nutritional Anaemias: Tools for Effective Prevention and Control.* WHO; Geneva, Switzerland: 2017. [(accessed on 25 February 2022)]. Available online: https://www.who. int/publications/i/item/9789241513067 [Google Scholar]
7. Cerami C. Iron Nutriture of the Fetus, Neonate, Infant, and Child. *Ann. Nutr.Metab.* 2017;**71**:8–14. doi: 10.1159/000481447. [PMC free article] [PubMed] [CrossRef] [Google Scholar]
8. Sangkhae V., Nemeth E. Regulation of the Iron Homeostatic Hormone Hepcidin. *Adv. Nutr. Int. Rev. J.* 2017;**8**:126–136. doi: 10.3945/an.116.013961. [PMC free article] [PubMed] [CrossRef] [Google Scholar]
9. Donovan A., Roy C.N., Andrews N. The Ins and Outs of Iron Homeostasis. *Physiology.* 2006;**21**:115–123. doi: 10.1152/physiol.00052.2005. [PubMed] [CrossRef] [Google Scholar]
10. Gkouvatsos K., Papanikolaou G., Pantopoulos K. Regulation of iron transport and the role of transferrin. *Biochim. Biophys. Acta (BBA) Gen. Subj.* 2012;**1820**:188–202. doi: 10.1016/j.bbagen.2011.10.013. [PubMed] [CrossRef] [Google Scholar]
11. Saito H. Metabolism of iron stores. *Nagoya J. Med. Sci.* 2014;**76**:235–254. [PMC free article] [PubMed] [Google Scholar]
12. Wang Y., Wu Y., Li T., Wang X., Zhu C. Iron Metabolism and Brain Development in Premature Infants. *Front. Physiol.* 2019;**10**:463. doi: 10.3389/fphys.2019.00463. [PMC free article] [PubMed] [CrossRef] [Google Scholar]
13. Joo E.Y., Kim K.Y., Kim D.H., Lee J.E., Kim S.K. Iron deficiency anemia in infants and toddlers. *Blood Res.* 2016;**51**:268–273. doi: 10.5045/br.2016.51.4.268. [PMC free article] [PubMed] [CrossRef] [Google Scholar]
14. Hariz A, Bhattacharya PT. Megaloblastic Anemia. Treasure Island (FL): StatPearls Publishing; 2021. ;
15. Lanzkowsky P. Manual of Pediatric Hematology and Oncology, 5th edition. Amsterdam, Netherlands: Elsevier; 2005. ;
16. Lokeshwar MR. IAP Speciality Series Book on Paediatric Haematology and Oncology. Mumbai, India: Indian Academy of Pediatrics; 2006.
17. Scrimshaw NS. Frequency Cause and significance of iron deficiency for the children of central Asia. 47-59
18. Viswanath et al. Red cell distribution width in the diagnosis of iron deficiency anemia; Indian J Pediatr 2001; 68 (12): 1117-1119.
19. Hazra B R, Hazra S C. Relative incidence of Iron deficiency amongst all anemias and its common cause; Ind J. Physiol and Allied Sci. 1998; Vol-52. No. 3.