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Prevalence of Nutritional Anemia in 'Brain Growth Period'

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Abstract

As many as 4-5 billion people, 66-80% of the world's population, may suffer from reduced learning ability and work capacity due to iron deficiency. Iron deficiency, and specifically iron deficiency anemia, is one of the most severe and important nutritional deficiencies in the world. As early as in 1967, Routh & Agarwal (Indian J Med Res) found that 65% healthy rich population in Delhi, India had no hepatic iron. Preschool children and women of reproductive age are at a highest risk. More than 2 billion people, > 30% of the world's population, are anemic. It is estimated that more than half of the pregnant women in developing countries are anemic [1].

Keywords: Anemia; Iron; Pregnancy; Lactation; Fetal; Placental transfer; Brain; Neurotransmitters

Introduction

Several studies have been carried out to estimate the prevalence of anemia, using the cyanmethemoglobin method. Agarwal, et al studied in the Indian states of Uttar Pradesh and Bihar; the national surveys done by the Indian Council Medical Research (ICMR)- covering 11 states; Showed that the prevalence of anemia in pregnant rural women was 87.6% (hemoglobin levels being <10.9g/dl). In 1992, these anemic women were given different doses of iron 60, 120 and 180 mg with 500 ug folic acid daily for 90 days in 6 states; however, 62% of them, in spite of taking iron-folate therapy, continued to be anemic. Thus, indicating that the short-term treatment as recommended then in the National anemia control program may not be sufficient to control anemia in pregnancy. However, it was observed that the birth weight of the babies born to these women improved, and the low-birth-weight deliveries reduced significantly [2]. The Nutrition Foundation of India in 2002-2003 found the prevalence of anemia in pregnancy and lactation in the rural areas (villages) in 7 Indian states (Assam, Himachal Pradesh, Haryana, Kerala, Madhya Pradesh, Orissa, Tamil Nadu). The prevalence in pregnancy in these villages was 84% (severe anemia Hb <7.0g/dl-in 9.2%) and prevalence in 3 month's Lactation was 92.2 % (Hb <7.0g/dl in 7.3%). These data support the findings of the Indian Council of Medical Research (ICMR) 1999-2000, data collected in 11 states 19 districts in the District Nutrition Survey found the pregnancy anemia prevalence of 84.6% (severe anemia Hb <7.0 g/dl-in 9.9%). 90% adolescent girls also had anemia [3] and >87% of children below 3 years of age were iron deficient [4]. Presently, after 2 decades, the National Family Health Survey (NFHS-5), showed that 54.3 % of the pregnant women are anemic in the rural areas in India as compared to 45.7 % in the urban areas. NFHS-5 and NFI studies used Hemocue and Cyanmetheglobin methods respectively, the former estimates higher level of hemoglobin. The above studies clearly showed that prevalence as well as severity of anemia during pregnancy and lactation remains grave. This is the period when child receives iron for brain (Table 1).

Maternal Death risk in child birth due to anemia

The condition is prominent in Southeast Asian countries where about half of all global maternal deaths are due to anemia and India contributes to about 80% of these in South Asia [5].

Effects of maternal iron deficiency on feto placental unit

- Normally Placental Iron transfer to fetus becomes 3 to 4 times during 20-37 wk of gestation.

a) Cord serum iron and hemoglobin were reduced in preterm as well as fullterm. There is an increased gradient in presence of maternal iron deficiency for transport of iron from mother to fetus but the transport remains proportionate to the degree of maternal hypoferriemia. Placenta plays an important role in maintaining iron transport to fetus. This process of iron transport is purely a placental function over which mother and fetus have no control, as placenta continues to trap iron even when fetus is removed in animals [6]. In spite of this efficient protective mechanism the placental iron content reduces significantly in maternal hypoferriemia [7]. In contrast,Vahlquist [8] in Swedish and Rios et al [9] in American women had shown that cord iron does not change in iron deficient pregnant women.

Table 1: *Percent prevalence of anemia (Hb<11g%) among pregnant women.*

	NFI Study	NFHS – 5	
	Rural	Rural	Urban
India	84	54.3	45.7
Assam	93.9	55.9	41.4
Haryana	91	57.2	54.6
Himachal Pradesh	68.1	43.9	*
Kerala	57.8	27.1	35.4
Madhya Pradesh	96.8	54.9	45.1
Orissa	97	62.2	59.5
Tamil Nadu	91.5	53.1	42.6

b) The placenta of anemic women showed quantitative decrease in villous surface area, volume of villi and length of blood vessel, while surface area and volume of intervillous space was increased. These placental changes in anemia did not normalise on rehabilitation- suggesting "Maturational arrest" [10,11].

b) Fetal Liver iron stores are reduced significantly in maternal hypoferremia. Normally bigger the infant and more advanced the gestational age higher was the amount of iron in fetal liver, spleen and kidney. The tissue iron content increases steeply in last 8 weeks of gestation. Infant born before 36 weeks of gestation, had half the iron content in hepatic reserve [12].

- Fetal brain iron content and neurotransmitters are reduced and their receptors are altered [13,14].

Rat Fetal Brain Iron Content in Maternal Latent Iron deficiency, Neurotransmitters

- Iron decreased 'IRREVERSIBLY' in all brain parts except medulla oblongata and pons [15,16].
- Susceptibility to Iron deficiency showed reduction in different parts of the brain:- corpus striatum-32%, midbrain 21%, hypothalamus 19%, cerebellum 18%, cerebral cortex 17% and Hippocampus 15%.
- Alterations in brain iron content also induced-Significant alterations in Cu, Zn, Ca, Mn, Pb and Cd.

Brain 'Glutamate metabolism'-(GAD, GDH, and GABA-T)

a) Marked reduction in levels of brain GABA, L glutamic acid and enzymes for biosynthesis of GABA and L-glutamate like glutamate decarboxylase and glutamate transaminase were also reduced.

b) Binding of GABA receptor increased by 143%, but glutamate receptor binding decreased by 63%.

- Brain 'TCA-cycle' enzymes-mitochondrial NAD+ linked dehydrogenase significantly reduced
- Brain 'Catecholamine metabolism'- Whole brain-dopamine, neonephrine, tyrosine and TAT significantly reduced; Corpus striatum – same as in whole brain, except TAT increased.
- Brain '5-HT metabolism'- Tryptophan, 5-HT, 5-HIAA significantly reduced.

The whole brain and corpus striatum showed reduction in catecholamine, dopamine, nor-epinepherine, tyrosine and monoamino oxidase, while tyrosine amino transferase increased in corpus striatum, inspite of reduction in whole brain suggesting that latent iron deficiency induced irreversible neurotransmitter alterations. These changes were specific to iron deficiency as neurotransmitter alterations in fetal brain due to malnutrition get normalised partially or completely on rehabilitation. The significant effects on neurotransmitter receptors (glutamate mediators) during early stages of iron deficiency clearly indicate the deficits in both excitatory and inhibitory pathways of the central nervous system [17,18].

Anemia and Brain -MRI studies

Iron deficiency and thalassemia; both are clinical conditions with anemia but former has no or low iron content and the later excess of iron in body tissues.

- The iron content in globus pallidus, caudate and dentate nuclei was similar in both the clinical conditions.
- There was an increase in creatinine and aspartate and reduction in choline concentration. These are very significant findings as choline is synthesized in the brain in very small amounts; its uptake is Na + dependent, which requires oxygen. Such changes are also observed in Hutington's chorea and Alzeimher's disease.
- In contrast in anemia the changes are due to anoxia, irrespective of body iron status.

References

1. Agarwal KN, Agarwal DK, Sharma A, et al. Prevalence of anemia in pregnant and lactating women in India. Indian J Med Res. 2006; 124: 173-184.
2. Agarwal KN, Agarwal DK, Mishra KP. Impact of anemia prophylaxis in pregnancy on maternal hemoglobin, serum

ferritin and birth weight. Indian J Med Res. 1991; 94: 277-280.

3. Teoteja GS, Singh PS, Dhillon BS, Saxena BN. Prevalence of anemia among pregnant women and adolescent girls in 16 districts of India. Food Nutr Bull. 2006; 27: 311-315.
4. Kapur D, Agarwal KN, Agarwal DK. Nutritional anemia and its control. Indian J Pediatr. 2002; 69: 607-616.
5. Agarwal KN. The effects of maternal iron deficiency on placenta and foetus. In advances in inter maternal child health. 1984; 4: 26-35.
6. Singla PN, Chand S, Agarwal KN. Cord serum and placental tissue iron status in maternal hypoferreimia. Am J Clin Nutr. 1979; 32: 1462-1465.
7. Fletcher J, Suter P. The transport of iron by the human placenta. Clin Sci. 1969; 36: 209-220.
8. Vahlquist BC. Das serumerrisen, eine paediatrisch kinische und experimental studies. Acta Paeditr. 1941; 28: 41-48.
9. Rios E, Lipschitz DA, Cook JD, Smith NJ. Relation of maternal and infant iron stores as assessed by determination of plasma ferritin. Pediatr. 1975; 55: 694-699.
10. Agarwal KN, Krishna M, Khanna S. Placental morphological and biochemical studies in maternal anemia before and after treatment. J Trop Paediatr. 1981; 27: 162-165.
11. Agarwal RMD, Tripathi AM, Agarwal KN. Cord blood hemoglobin iron and ferritin status in maternal anemia. Acta Paediatr. Scand. 1983; 72: 545-548.
12. Singla PN, Gupta VK, Agarwal K N. Storage iron in human fetal organs. Acta Paediatr. Scand. 1985; 74: 701-706.
13. Agarwal KN. Iron and the brain: neurotransmitter receptors and magnetic resonance spectroscopy. Br J Nutr. 2001; 85: 147-150.
14. Shukla A, Agarwal KN, Chansuria JPN, Taneja V. Effect of latent iron deficiency on 5-hydroxytryptamine metabolism in rat brain. J Neurochem. 1989; 52: 730-735.
15. Shukla A, Agarwal KN, Shukla GS. Effect of latent iron deficiency on metal levels of rat brain regions. Biol Trace Elem Res. 1989; 22: 141-152.
16. Shukla A, Agarwal KN, Shukla GS. Latent iron deficiency alters gamma-amino butyric acid and glutamate metabolism in rat brain. Experentia. 1989; 45: 343-345.
17. Shukla A, Agarwal KN, Shukla GS. Studies on brain catecholamine metabolism following latent iron deficiency and subsequent rehabilitation in rat. Nutr Res. 1989; 9: 1177-1186.
18. Mittal RD, Pandey A, Mittal B, Agarwal KN. Effect of latent iron deficiency on GABA and glutamate receptors. Indian J Clin Biochem. 2002; 17: 1-6.