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Prevalence Of Zinc Deficiency In Breasted New Born Babies

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Abstract:

Introduction: Zinc is an essential trace element, mainly acting as a cofactor in enzymes and nucleic acids. It helps in the regulation of gene expression by binding with high affinity to the correct region of DNA via Zinc Finger Motifs. Adequate zinc is essential for optimum growth, immune competence and neurobehavioral development, but limited information on population zinc status hinders the expansion of interventions to control zinc deficiency. Zinc crosses the placenta and accumulates in the fetus during the last trimester and hence premature infants are at high risk of Zinc deficiency.

Aims and objectives: To find the prevalence of Zinc deficiency in the preterm and term newborn infants by measuring serum Zinc in the first week of life.

Material and methods: This was a prospective observational study conducted from July 2013 to May 2015 in SKIMS Srinagar, a tertiary care hospital in north India, involving 100 otherwise normal babies. Serum Zinc was determined during the first week of life in both term and preterm babies who came in the contact during their routine follow up visits. The babies were not having any obvious disease and were exclusively breastfed.

Results: A total of 100 babies were studied, out of which 68 were term and 32 were preterm. Among the 100 babies 45 babies were deficient in Zinc, defined as serum Zinc less than 60 mcg/ dl. Prevalence of zinc deficiency was more in preterm babies (58%) compared to term babies (38%). Mean serum Zinc was 69.38±14.51 mcg/dl in the term babies and 57.43±15.98 mcg/dl in the preterm babies.

I. INTRODUCTION

Zinc is an essential trace element with atomic number 30. It is a component of thousands of metalloenzymes and nucleic acids. Amongst its properties, the first to be identified were the catalytic properties. A major advance in our understanding of biology of zinc was the identification of proteins that contain a 'zinc finger motif'. Structurally a ZMF is a recurring pattern of amino acids with conserved residues of cysteine and histidine at the base to which zinc binds in a tetrahedral arrangement. It helps in regulation of gene expression by binding with high affinity to the correct region of DNA.

Zinc crosses the placenta and accumulates in the fetus during the last trimester at around 250mcg/kg/24hr.The amount of zinc provided by 200ml of human milk per kg per day falls from 1665mcg on the first day of lactation to 160mcg after 4 months. Infants are at risk of zinc deficiency during the first 6 months of age. Adequate intake during first 6 months of life is 2 mg/day.

Adequate Zinc intake is critical for health. The production and potency of several cytokines is perturbed even in mild Zinc deficiency leading to early apoptosis. It plays a role in the maintenance of integrity of epitheliums as well as acts as an antioxidant. Thus, in the case of diarrhea multiple functions of the zinc may help to maintain the integrity of the gut

mucosa to reduce or prevent fluid loss. Notably, these responses can occur within 48 hours, much more readily than the direct effects of zinc on the cellular development.

Asymptomatic Zinc deficiency is defined as serum Zinc less than 60 mcg/ dl with no clinical features. Mild Zinc deficiency causes nonspecific adverse effects on health including but not restricted to impaired growth, increased incidence if infectious diseases and adverse pregnancy outcomes. Most severe form of Zinc deficiency is acrodermatitis enteropathica, characterized by vesiculobullous or psoriaform skin lesions symmetrically distributed over peri-oral, acral and perineal areas The hair often has a peculiar reddish tint and alopecia of some degree is characteristic. Associated manifestations include chronic diarrhea, stomatitis, paronychia, nail dystrophy, growth retardation, intercurrent bacterial infections and super infection with candida albicans.

Zinc is used for the treatment of zinc deficiency, acrodermatitis enteropathica, Wilson's disease and acute diarrhea. Some evidence now indicates that as many as 25% cases of diarrhea and 40% cases of pneumonia can be prevented by preventing Zinc deficiency. In populations at risk of Zinc deficiency, preventive Zinc supplementation reduces the incidence of premature delivery, decrease morbidity from childhood diarrhea and lower respiratory infections, lowers all-cause mortality, and increases the linear growth and weight gain among infants and young children. Among the most urgent remaining issues are the identification of the population at most risk of zinc deficiency and identification of culturally appropriate and sustainable strategies to reduce the prevalence of Zinc deficiency through the changes in the dietary practices.

Three indicators of population risk of Zinc deficiency have been recommended: (1) the percentage of population with serum Zinc concentration below an appropriate cut off, (2) the prevalence of usual dietary Zinc intakes below the Estimated Average Requirement (EAR), and (3) the percentage of children less than 5 years of age with height-for-age Z scores less than -2 SD with reference to WHO child growth standards. Currently Zinc status in community is best measured by serum Zinc and hence we used it as a marker of Zinc deficiency.

II. MATERIAL AND METHODS

This was a prospective observational study conducted from July 2013 to May 2015 in SKIMS Srinagar, a tertiary care hospital in north India. A total of 100 babies were studied. The study was approved by IEC SKIMS Srinagar. Both term and preterm babies who were in their first week of life were included. The babies were exclusively breastfeeding and apparently free from any disease

Baseline parameters were calculated at the beginning of the study. 2ml of venous blood samples during morning hours were obtained for serum zinc determination by trace mineral-free syringes having stainless steel needles. The samples were collected in Zinc-free vacutainer plastic tubes.

We used Colorimetric method for the estimation of the serum Zinc. Zinc Quantification is convenient with this assay in which Zinc binds to a ligand that will be developed and detected at an absorbance at 560 nm. The assay can be used with biological samples such as serum, plasma, CSF or urine with detection sensitivity 0.2 µg/ml (~1– 3 µM). Upon arrival, kit was stored at +4°C and protected from light.

The cells were harvested with an EDTA-free lysis buffer. It was spun down to get rid of cell debris and transfer supernatant to clean tubes. Lysates generally contain significant amounts of protein, so they were deproteinized by adding 50 µl of the 7% TCA solution per 50 µl of the sample (by adding 1x volume TCA to 1x volume sample).

Statistical analysis was done by SPSS software (version 20; SPSS Inc Chicago, IL). Categorical variables were analyzed by using $\chi 2$ /Fishers test. Continuous variables were analyzed by using independent t-test for parametric data and Mann Whitney U test for non-parametric data. Normalcy of the data was checked by Shapiro Wilk test and by examining Q-Q plots.

III. RESULTS

A total of 100 babies were entered into the study; 68 were term and 32 were preterm & 53 babies were male and 47 were female. Majority of the babies were of the birth order 1 or 2. The mean birth order in was 2.06± 0.63. All, except one were born at hospital. Majority (56%) were born at trust hospital. 44 were born by LSCS and 16 by NVD. The average age of the neonates was 3.5±1.73 days. The average age of the mothers was 30.50±3.17 years. The average gestational age of the babies was 37.59±3.04. The average birth weight of the neonates in the study group was 2499±783. The average length of the neonates was 47.33±4.34. The average head circumference of the neonates was 33.00±2.43 centimeters.

About 45% of the babies were Zinc deficient at birth. The average serum zinc at birth of the neonates was 69.38±14.51mcg/dl. Serum Zinc levels were lower in preterm babies; 57.43±15.98 mcg/dl and the prevalence of Zinc deficiency was 58% in them. Serum Zinc concentration less than 60 mcg/dl was considered to be deficiency.

Baseline parameter	Value
AGE OF THE BABY	3.50±1.73 days
MOTHERS AGE	30.50±3.17 years
GESTATIONAL AGE	37.59±3.04 weeks
WEIGHT AT BIRTH	2499±783 grams
LENGTH AT BIRTH	47.33±4.34 cms
HEAD CIRCUMFERENCE AT BIRTH	33.00±2.43 cms
SERUM ZINC AT BIRTH	69.38±14.51 mcg/dl
BREAST MILK ZINC	0.51±0.09 mcg/dl

Table 1

IV. DISCUSSION

Zinc is an essential element in body, its deficiency leads to nonspecific involvement of any organ or process of body as well as some specific deficiency symptoms notably acrodermatitis enteropathica. Worldwide prevalence of Zinc deficiency is around 4-73 % with an average of 31 %, Prevalence of in South East Asia is 34-73 %. A metanalysis

indicated that daily zinc supplementation can reduce the incidence of pneumonia by 41% and diarrhea by 18%. Zinc deficiency affects about 2.2 billion people around the world. About 25 % of the world population is at risk of Zinc deficiency because of inadequate intake of Zinc and excess of phytate. Even in the United States, about 12% of the population is at risk of Zinc deficiency, and perhaps as many as 40 percent of elderly. Using the food balance sheets, it was estimated that about 71.6 % of total population in Southeast Asia was at risk of developing zinc deficiency. On a conservative estimate 40 % of Indian children suffer from zinc deficiency. According to a study conducted in Delhi 73.3% of the children were zinc deficient (plasma zinc <70%). A high prevalence of zinc deficiency (41%) has been reported among pregnant women from Haryana, India. Given its magnitude of prevalence, it becomes essential to find the prevalence of its deficiency in each geographical area so as to take appropriate steps for its prevention and correction.

Prevalence of Zinc deficiency in our part of world in new born babies was found to be 45%. The average serum zinc at birth of the neonates was 69.38±14.51mcg/dl. Serum Zinc levels were lower in preterm babies; 57.43±15.98 mcg/dl and the prevalence of Zinc deficiency was 58% in them. Serum Zinc concentration less than 60 mcg/dl was considered to be deficiency. Very high prevalence of Zinc deficiency was found in our region probably because of high intake of phytate by mothers as well as because our region is a developing nation. Because of developing nature, people here take more of vegetables and pulses containing an excess of phytate which reduced the bioavailability of Zinc. Polished rice as a staple food can be one more contributory factor as is lesser intake of animal products.

Although rare; characteristic features of zinc deficiency are found only in severe zinc deficiency and typically in acrodermatitis enteropathica. Acrodermatitis enteropathica is a rare autosomal recessive disorder caused by an inability to absorb sufficient zinc from diet. The genetic defect is in the intestinal zinc specific transporter gene SLC39A4. The disease affects nearly every system of the body. Cutaneous involvement manifests as vesiculobullous, eczematous or psoriaform skin lesions symmetrically distributed over peri-oral, acral and perineal areas and on the cheeks, knees and elbows. The hair often has a peculiar reddish tint and alopecia of some degree is characteristic .Ocular manifestations include photophobia, conjunctivitis and corneal dystrophy. Associated manifestations include chronic diarrhea, stomatitis, glossitis, paronychia, nail dystrophy, growth retardation, irritability, delayed wound healing, intercurrent bacterial infections and super infection with candida albicans. These children typically died in later infancy before the therapeutic effects of oral zinc were recognized and routinely applied .Although subtle and nonspecific; mild to moderate zinc deficiency is the most significant type of zinc deficiency because of its high prevalence. Mild zinc deficiency leads to growth retardation, adverse pregnancy/fetal outcomes and increased predisposition to infections especially respiratory tract infection, diarrheas and malaria. Worldwide high prevalence of mild zinc deficiency leads to preventable death of around 0.8 million children every year. The burden of zinc deficiency is so huge that it has been estimated that more deaths can be prevented by preventing zinc deficiency than by absolute sanitation of whole world. So, it is high time to prevent as well as treat zinc insufficiency.

Five interventional strategies can be used to prevent Zinc deficiency: (1) Adding Zinc to soil, called agronomic biofortification, which both increases crop yields and provides more dietary Zinc. (2) Adding zinc to food, called fortification. (3) Taking Zinc rich foods. Foods rich in Zinc are animal proteins. Although cereals and pulses are rich in zinc but that has low bioavailability because of chelating phytates. (4) Oral repletion as liquid or tablets. (5) Oral repletion as multivitamins/minerals containing zinc. In order to improve our understanding of zinc deficiency in newborn babies, we need to find the zinc content of the breast and the prevalence of zinc deficiency in mother's milk. To assess the effect of zinc supplementation on our babies we need to conduct a randomized controlled trial as well so as the proper decision about zinc supplementation can be taken. A study to find the effect of zinc supplementation in newborn babies during first 6 months on the growth and incidence of respiratory and gastrointestinal infections during first 5 years of life is strongly recommended.

To conclude; zinc is an essential trace element of human body, there is very high prevalence of zinc deficiency in our part of world, leading to preventable morbidity and mortality. Importance of zinc in health has been ignored too long now. We recommend that special programs be implemented to effectively tackle zinc deficiency as are made for iron, iodine and vitamin A.

WHAT IS ALREADY KNOWN?

Globally high risk of Zinc deficiency (4-73%) in general population based on the zinc deficiency in diet.

WHAT THIS STUDY ADDS?

High prevalence of actual zinc deficiency (45%) in newborn babies in a specific geographical area.

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