MATERNAL AND FOETAL PLASMA ZINC CONCENTRATION AND CONGENITAL MALFORMATIONS

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SUMMARY

Plasma zinc levels were estimated within 24 hours of delivery in 28 women who gave birth to congenitally malformed infants; and cord blood collected in 20 malformed infants; and the results were compared with normal controls.

Plasma zinc level in women who had delivered malformed babies were significantly low - 48.28 mgm/dl ± 19.38 than in women who had delivered normal infants - 81.7 mgm/dl ± 20.47, P<0.01. Cord blood zinc levels were also low in malformed infants - 47.28 mgm/dl ± 22.4 than in normal infants 79.46 mgm/dl ± 18.47, P<0.01.

A strong correlation was found between low maternal plasma zinc level and congenital anomalies - specially neural tube defects and zinc deficiency is responsible for malformation by still illunderstood mechanism.

Introduction

The subject of congenital malformation is of paramount importance as it contributes considerably to perinatal mortality. The cause of most of the congenital malformations is unknown; though both hereditary and environmental factors are considered to be important.

Zinc is an important metabolic substance for mother and foetus. Zinc deficiency appears to have a marked effect on growing on proliferating tissues. Zinc deficiency in pregnant rats resulted in high rate of embryonic death, severe intrauterine growth retardation and high incidence of congenital malformations, affecting every organ system (Hurley et al., 1966). Solton and Jenkins (1982) reported mean maternal plasma zinc levels to be significantly lower in women who gave birth to congenitally malformed infants than in controls. This study was carried out to establish the relationship between maternal plasma zinc levels and congenital anomalies in newborn.

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Material and Method

The study was carried out the Government Medical College, Nagpur. Plasma zinc levels were estimated within 24 hours of delivery in 28 women who gave birth to a congenitally malformed infant (Group I); and when cord blood collection was possible plasma zinc concentration was done in 20 congenitally malformed babies (Group II). Plasma zinc levels were studied in 20 mothers who gave birth to normal babies (Group III) and cord blood zinc estimation of these normal babies was also carried out (Group IV).

Detailed history was taken to exclude any major illness, history of bleeding per vaginum, drug intake, radiation and any infection during early weeks of pregnancy were noted. Blood sugar estimation was done in all cases.

Plasma zinc levels were estimated by Atomic absorption spectrophotometric analysis. Absorption was plotted against particles per million (PPM) and concentration was calculated.

Observations

Distribution of patients in control and study group were compared as regards age and parity, socio-economic status. One patient had septran (Trimethoprim 80 mg + methoxazole 40 mg) at 8 weeks of gestation congenital anomalies in this neonate were, encephalocele with polydactaly and syndactaly; while other women who had chloroquin at 12 weeks of gestation in this case neonates had cleft lip and cleft palate. Two mothers had fever at 8 weeks and 10 weeks of gestation, anomalies recorded were omphalocele and facial palsy with rudimentary ear respectively. Associated hydramnios was present in 21% of cases.

TABLE - I
VARIOUS CONGENITAL ANOMALIES DETECTED AT BIRTH

	Anomaly	*No. of cases*
1.	Anencephaly	7
2.	Hydrocephalus	5
3.	Meningocele	2
4.	Spina bifida	1
5.	Cleft lip and palate	4
6.	Omphalocele	1
7.	Encephalocele with Syndactaly and Polydactaly	2
8.	Talipes equinovarus	1
9.	Imperforate anus	1
10.	Microcephaly	1
11.	Facial palsy with rudimentary ear	1
12.	Single umbilical artery	1
13.	Achondroplasia	1
		28

Above table shows various abnormalities encountered during study. Central nervous system anomalies were prevalent (64%). Multiple congenital anomalies were observed in 10 percent of cases.

TABLE - II
MALE FEMALE RATIO

Sex	*No. of cases*	*Percentage*
Male	19	67.85
Female	9	32.14

Maximum number of malformed babies i.e. 67..85% were males as shown in above table. Live births were 72 percent as compared to stillbirth i.e. 28%.

The lowest value of plasma zinc was found in anencephaly baby 18.5 mgm/dl and the highest value 110.4 mgm/dl in cleft lip and palate.

TABLE - III
ZINC CONCENTRATION IN VARIOUS GROUPS

Group	*Plasma zinc in mgm/dl mean ± S.D.*		*P*
	Group		
Mother -	Study I	48.28 ± 19.38	<0.01
	Control III	81.73 ± 20.47	
Infants	Study II	47.28 ± 18.47	<0.01
	Control IV	79.46 ± 22.41	

Plasma zinc level was lower i.e. almost half in mother who gave birth to congenital malformed babies as compared to mothers who had normal babies.

Discussion

In 1869 Raulin showed that zinc was essential for the growth of fungus Aspergillus niger but it was not until 1934 when Todd et al. demonstrated that zinc was an essential nutrient in mammalian species of rats. The need for an adequate zinc intake by the breeding hen to ensure normal embryonic development was confirmed in the experiment by Blamberge and Unabelle in 1960.

Hurley (1966) have shown that zinc deficiency appears to have a most marked effect on the growing or proliferating tissue. Almost all the full term foetuses showed gross congenital anomalies involving skeletal, brain, eye, heart, lung and urogenital system.

Laurence et al (1980) concluded that women receiving adequate diets have a lower incidence and recurrence of foetal neural tube defects than women receiving poor diets.

Wynn and Wynn (1981) reported epidemics of congenital malformations particularly following wars and famines.

Soltan and Jenkins (1982) noted significantly low plasma zinc levels in women who had delivered congenitally malformed infants than in normal controls. 9.03 ± 2.16 and 10.36 ± 2.04 micromoles/litre respectively.

Buamah in 1984 showed that serum zinc concentrations were lower in the anencephalic pregnancy than in normal control subjects.

Relationship between low maternal plasma zinc and congenital malformations in newborns is confirmed in present study.

Plasma zinc values in cord blood samples correspond to their maternal blood samples in both the groups thus indicating zinc is transferred passively from the mother to foetus across the placenta. Plasma zinc values in congenital anomalous babies and their mothers are low by about 30 mgm/dl. When compared to normal babies and their mothers.

Significantly lower values of plasma zinc are recorded in CNS malformation 43.2 mgm/dl as compared to other malformations 59.1 mgm/dl.

Zinc deficiency may operate directly by impairment of DNA, RNA synthesis and thus in nucleic acid metabolism and metalloenzyme synthesis. This deficiency may be a part of generalized dietary deficiency or may be caused by chronic infections or inherited defects in metabolic handling of zinc. It seems probable that both dietary phytate and fiber may contribute to the occurrence of zinc deficiency in poorly nourished group that subsist largely on cereals namely wheat.

Marginal zinc deficiency at or before conception may become severe with the demands of pregnancy specially if com-

bined by anorexia and vomiting. Low zinc levels during conceptions or during early embryogenesis may operate as a cause of congenital malformation, suggest zinc supplement in early pregnancy or preconceptional therapy as a prophylaxis, Dietary counselling may be advised to women with history of congenital malformation in previous pregnancy. Brown flour, egg yolk, meat, cabbage contain zinc and yeast oysters are rich sources of zinc.

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