

The Influence of Maternal Iron Deficiency Anaemia on the Haemoglobin Concentration of the Infant

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Summary

Serum ferritin, iron, and haemoglobin (Hb) values of 27 pregnant women who did not receive oral iron therapy during pregnancy, and Hb of their normal full-term babies were determined. Maternal blood samples were obtained at 16 and 34 weeks of pregnancy and infants' blood samples were obtained at the first day and 3 months of life. Mothers were divided into two groups according to their serum ferritin values. By analysing the results we were not able to detect any correlation between maternal ferritin, Hb and newborn gestational age, and Hb and birth weight. The same was found when the groups were compared by a maternal serum ferritin above and below 12 ng/ml.

Introduction

The benefit of routine iron supplementation during pregnancy is questionable.¹ Administration of iron to mothers was not associated with any important improvement for perinatal outcome such as low birth weight, prematurity, infant mortality, and serious maternal mortality.² Maternal iron deficiency anaemia do not cause anaemia in the infants soon after birth.^{3,4} Hb levels of newborn infants were not influenced by the presence of depleted iron stores or latent iron deficiency of the mothers.⁵

This study was performed in Sivas, Turkey, where the socio-economic conditions are poor, and most of the mothers have inadequate nutrition and multiparities. We have investigated the effect of maternal iron deficiency anaemia on the Hb level of the infant during the first 3 months of life.

Materials and Methods

Twenty-seven mothers with a median age of 24.1 years (range 18–34 years) having less than 16 weeks of pregnancy who did not receive iron, although recommended, were included in the study. None of the mothers included in the study, had any infection and malignancy or hepatic disease. Weight gain, health, and nutritional status of these mothers were carefully followed up and recorded.

All infants (11 boys and 16 girls) were born after a 38–42-week period of gestational age calculated from the first day of mother's last menstrual period and assessed clinically by Dubowitz criteria within 24 h of birth.

Maternal serum ferritin, iron, Hb, and transferrin saturation levels were measured at 16 and 34 weeks of pregnancy, and after delivery. Hb concentration was measured in all infants in the first 24 h, and at 3 and 6 months of age.

Serum ferritin concentrations were determined by EIA (biomerix-69080) kit.

Students *t*-test, regression and correlation analyses were used for statistical analyses.

Results

Mothers were divided into two groups: the first, consisting of women whose mean serum ferritin was <12 ng/ml and the second with ferritin levels ≥ 12 ng/ml or equal at 16 and 34 weeks of pregnancy. The mean of serum ferritin in the first group (7.57 ± 1.02 ng/ml and 5.53 ± 0.71 ng/ml) was significantly lower than that in the second group (32.9 ± 3.55 ng/ml and 22.41 ± 3.02 ng/ml, $P < 0.01$). The mean Hb concentration of the low serum ferritin group was found significantly lower than that of the higher ferritin group at 16 weeks of pregnancy ($P < 0.01$). Seven mothers, previously clinically non-anaemic, would become clinically anaemic in the late stage of their pregnancies. Serum ferritin and Hb values of mothers are summarized in Table 1.

Table 2 shows the characteristics of newborns whose mothers have serum ferritin values of <12 ng/l and ≥ 12 ng/l.

Discussion

In recent years it was reported that maternal anaemia frequently results in premature birth, and affects perinatal mortality and morbidity.⁶ Hemminki and Starfield had shown that administration of iron did not affect this condition.² Our results are in harmony with those.

Serum ferritin concentration is accepted as a reliable indicator of whole body iron storage in healthy

TABLE 1
Maternal serum ferritin and haemoglobin levels

Pregnancy weeks		n	Haemoglobin (g/dl)		Serum ferritin (ng/ml)	
			$\bar{x} \pm Sx$	t	$\bar{x} \pm Sx$	t
16 weeks	<12 ng/ml	7	9.3 ± 0.22	$t=8.95$	7.57 ± 1.02	$t=6.84$
	≥12 ng/ml	20	11.95 ± 0.19	$P<0.01$	32.9 ± 3.55	$P>0.01$
34 weeks	<12 ng/ml	14	11.02 ± 0.25	$t=0.39$	5.53 ± 0.71	$t=5.57$
	≥12 ng/ml	13	11.16 ± 0.23	$P>0.01$	22.41 ± 3.02	$P<0.01$

TABLE 2
Infants characteristics according to maternal serum ferritin values at 16 and 34 weeks of gestation

Maternal serum ferritin concentration	n	Gestational age (weeks) $\bar{x} \pm Sx$	At birth			3 Months		
			Weight (g) $\bar{x} \pm Sx$	Haemoglobin (g/dl) $\bar{x} \pm Sx$	t	Haemoglobin (g/dl) $\bar{x} \pm Sx$	t	
16 weeks	<12 ng/l	7	39 ± 0.30	3278.57 ± 99.36	16.7 ± 0.59	$t=0.79$	12.75 ± 0.23	$t=1.85$
	≥12 ng/l	20	39.42 ± 0.15	3225 ± 85.12	17.25 ± 0.38	$p>0.01$	12.2 ± 0.19	$P>0.01$
34 weeks	<12 ng/l	14	39.35 ± 0.19	3210.71 ± 128.14	17.2 ± 0.36	$t=0.21$	12.4 ± 0.14	$t=1.14$
	≥12 ng/l	13	39.2 ± 0.20	3234.6 ± 119.12	17.06 ± 0.54	$P>0.01$	12.18 ± 0.17	$P>0.01$

subjects and pregnant women. The effect of mother's iron storage on the fetus is an interesting subject, and this interest increased by the utilization of serum ferritin estimations.⁷

No difference was found between the initial and last ferritin values of mothers who were followed up to delivery (Table 1). When serum ferritin levels were examined, it was found that in anaemic mothers having less than 12 ng/ml, ferritin levels were low in seven mothers at 16 weeks and in 14 mothers at 34 weeks. During pregnancy, serum ferritin concentration decrease and from 35 weeks of pregnancy onwards concentrations are significantly lower in women who have not received iron. The early fall in serum ferritin concentration is probably due to increased maternal erythroid activity, but in later half of pregnancy the declining ferritin concentrations reflect the transfer of iron to the fetus.^{8,9} This was confirmed by serum ferritin concentration estimation in our study. The mean hemoglobin concentration of the low serum ferritin group was found significantly lower than that of the higher ferritin group (Table 1). Therefore, we think that serum ferritin concentration at 16 weeks of gestation cannot be a reliable prognostic sign of future anaemia in the infant.

There are controversial findings in the literature about the effect of maternal iron deficiency anaemia on

the haemoglobin level of the infant. Placenta and fetus have a special affinity to iron in the mother's circulation and iron is transported through the placenta in spite of concentration gradient.¹⁰ This mechanism provides enough iron for fetal erythropoiesis, even if maternal iron storages are depleted. Consequently, Hb concentration of the newborn is not affected by maternal iron depletion or latent iron deficiency anemia.^{11,12} In this study, mean ferritin levels of mothers at 16 and 34 gestational weeks (serum ferritin levels <12 ng/ml and ≥12 ng/ml) are compared with infants' Hb concentrations at delivery and third month. The difference between these groups were insignificant ($P>0.05$). In other words, infants' Hb level is not affected by maternal ferritin levels. Most of iron in the human body is found in haemoglobin and iron metabolism is largely concerned with the synthesis and breakdown of this protein. Because of this, although Hb levels in the infants do not directly show body stores of iron it may reflect iron status.

We can conclude that, extremely unusual conditions excluded, maternal iron deficiency does not give rise to iron deficiency anaemia in the infant in the period from birth to the third postnatal month. On the contrary, in recent years it was shown that high Hb levels in pregnant mothers caused placental infarcts, which in turn causes intra-uterine growth retardation and

perinatal death.¹³ Iron prophylaxis is applied in iron administration is not justified in

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Haemopoiesis

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Amanita phalloides is a deadly mushroom, well as hepatic failure. Three brothers beginning 12 months of the liver enzyme patients under conservative treatment. The life span is short.

Amanita phalloides is a deadly mushroom, about 90 per cent of patients succumb. Fifty to 100 mg of the mushroom include the amatoxins which are mainly responsible for the liver failure.

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perinatal death.¹³ When the serum ferritin concentration falls below about 50 ng/ml, the initiation of iron prophylaxis is appropriate.¹⁴ We think that routine iron administration to non anemic pregnant mothers is not justified in normal conditions.

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Haemoperfusion in *Amanita phalloides* Poisoning

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Summary

Amanita phalloides is responsible for about 90 per cent of all fatal cases of mushroom intoxication. The amatoxins, the main toxic component of these fungi, are responsible for gastro-intestinal symptoms, as well as hepatic and renal failure.

Three brothers with *Amanita phalloides* poisoning were admitted with gastro-intestinal symptoms beginning 12 h after ingestion. Jaundice, hepatomegaly and neurological symptoms were not present, but the liver enzymes were moderately increased. Alfa-amanitin was detected in sera of all patients. All patients underwent charcoal hemoperfusion and two of them had additional haemodialysis along with conservative therapy. Liver enzymes that showed marked increase on the second day of therapy decreased to normal levels on the 28th day. All of our patients survived.

The life saving role of early haemoperfusion in *Amanita phalloides* poisoning is emphasized.

Amanita phalloides (*A. phalloides*) is responsible for about 90 per cent of all fatal cases of mushroom poisoning. Fifty to ninety per cent of untreated cases succumb.¹⁻³ The main toxic components of these fungi include the amatoxins and the phallotoxins. The amatoxins which are cyclic heptapeptides (MW 1000) are mainly responsible for the early acute gastro-

intestinal symptoms, and the later liver and kidney damage. The phallotoxins, on the other hand, are inactive when taken orally and are not normally involved in human poisoning, but they are potent toxins when given by other routes.^{1,3,4} Amatoxins interfere with the activity of RNA polymerase B thereby inhibiting protein synthesis resulting in cytolysis of hepatocytes and loss of hepatic function.⁵ The toxin also causes damage to the intestinal tract which is thought to be due in large part to enterohepatic

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