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Original article

Relationship between Maternal Obesity and Maternal and Neonatal Iron Status

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Abstract

Background: Maternal iron condition influences the iron status of neonates since iron transferred from the mother is the only source for fetal iron. The aim of this study was to examine the association of obesity with inflammatory markers (CRP) and iron status in both mother and infant. **Methods:** This comparative cross sectional study was conducted on 100 pregnant women and their neonate from the attendance of Agouza Police Hospital and Benha University Hospitals. **Results:** Our results shows that there was no statistically significant difference found between obese and non-obese pregnant women regarding neonatal HB, TLC, Platelets count, Serum iron in cord, TIBC in cord and CRP in cord. There was a negative impact of maternal BMI on neonatal iron status. The maternal obesity was inversely associated with cord ferritin concentrations, poorer iron status determined by cord ferritin were evident in the neonates of obese women, compared with those born to normal weight women. Maternal CRP, maternal hepcidin and maternal serum transferrin receptor were greater in obese compared with normal weight pregnant women. **Conclusion:** There is a negative impact of maternal BMI on neonatal iron status. The maternal obesity was inversely associated with cord ferritin concentrations, poorer iron status determined by cord ferritin were evident in the neonates of obese women, compared with those born to normal weight women. The concentrations of CRP was greater in obese compared with normal weight pregnant women. Serum hepcidin was elevated in obese pregnant women compared with the normal weight women in this study.

Key words: Maternal Obesity - Maternal Iron Status - Neonatal Iron Status

Introduction:

Obesity is a growing health problem worldwide. World Health Organization (WHO) defines obesity as abnormal or excessive fat accumulation that may impair health with body mass index (BMI) of 30 kg/m2 or more as obese among adults. In 2013, the American Medical Association classified obesity as a disease. Prevalence of obesity among women of reproductive age is increasing worldwide, with current estimates of 20–36%. As the prevalence increases among the women of reproductive age group, so it does among pregnant females ***(1).***

Normal fetal growth and development is dependent upon maternal iron sufficiency during pregnancy. Pregnant women have an increased requirement of iron to support fetoplacental development, expansion of maternal red blood cell mass, and to compensate for intrapartum blood loss; to meet this need, absorption of dietary iron is enhanced concomitant with increased utilization of existing iron stores. (**2)**

Iron deficiency (ID) is common in young women of reproductive age; in Europe, a recent estimate suggests that the prevalence of ID is 10 to 32%. Maternal ID is a major cause of maternal morbidity, and is linked to a higher risk of preterm delivery, low birth weight, and adverse effects on infant neurodevelopment. In infancy, ID has long-term effects on cognitive function.(**3)**

Rates of obesity in pregnancy have significantly increased in recent years, with UK women reported to have the highest prevalence in Europe (25.2%), and maternal obesity is associated with most major adverse maternal and fetal outcomes. **(4)**

The aim of this work was to examine the association of maternal obesity with inflammatory markers (CRP) and iron status in both mother and infant.

Patients and Methods

Subjects:

Study group:

Comparative cross sectional study was conducted on 100 pregnant women and their neonate from the attendance of Agouza Police Hospital and Benha University Hospitals .

Inclusion criteria:

All pregnant women at Agouza police hospital and Benha University Hospitals, Obese women (body mass index (BMI) ≥ 30 kg/m2) and normal weight (BMI < 25 kg/m2) pregnant women, and their neonates.

Exclusion criteria:

- Women with pre-existing diseases as :
  - Autoimmune disorders

- Blood clotting disorders
- Cancer
- Epilepsy and seizure disorders
- Gastrointestinal disorders

- antepartum hemorrhage, pre-eclampsia.
- Those who were unable or unwilling to give written informed consent.
- Preterm birth due to underlying medical conditions
- Intrauterine fetal death
- Still birth baby
- Neonate with congenital anomalies.
- Twins neonate.

All pregnant women in both groups were subjected to the following:

- **Maternal history include:**
  - Age.
  - Body mass index (divide weight of mother in kilograms by her height in meters squared).
  - Parity.
  - Smoking or not.
  - Educational level.
  - History of Gestational Diabetes.
  - History of Pre-eclampsia.
- **Neonatal history include:**
  - Gestational age at delivery.
  - Sex of baby.
  - Large or small for gestational age.
  - Weight at delivery.
- **Maternal Investigations were done At 15 weeks' gestation for all groups including:**
  - CBC.
  - Serum ferritin.
  - Serum transferrin receptor.
  - Total iron binding capacity.
  - Hepcidin.
  - CRP.
- **Neonatal investigations were done at the time of birth for all groups including:**
  - Serum ferritin.
  - CRP.
  - CBC.
- **Data and Sample Collection:**

At 15 weeks' gestation sociodemographic and anthropometric measurements including objectively measured weight, were collected. Maternal blood samples were collected and stored at −80◦C, and for the infants, cord blood was collected following delivery . Information on maternal and neonatal outcomes was collected throughout pregnancy and at delivery.

Statistical analysis:

Data were collected, revised, coded and entered to the Statistical Package for Social Science (IBM SPSS) version 20. The qualitative data were presented as number and percentages while quantitative data were presented as mean, standard deviations and ranges when their distribution found parametric.

The confidence interval was set to 95% and the margin of error accepted was set to 5%. So, the p-value was considered significant as the following:

P > 0.05 = non-significant (NS).

P < 0.05 = significant (S).

P < 0.001 = highly significant (HS).

Results:

There was no statistically significant difference found between two groups regarding Age, and gravidity & parity, and there was highly statistically significant difference found between two groups regarding Education level Table (1).

The previous table shows that there was no statistically significant difference found between two groups regarding Sex of baby, Large or small for GA and birth weight (KG), and there was highly statistically significant difference found between two groups regarding Gestational age at delivery Table (2).

The previous table shows that there was no statistically significant difference found between two groups regarding maternal HB, TLC, PCV, Platelet count, Serum iron, TIBC, UIBC and Serum ferritin (mg/L) and there was highly statistically significant difference found between two groups regarding CRP and Transferrin receptor, and Hepcidin. (Table 3).

The previous table shows that there was no statistically significant difference found between two groups regarding neonatal HB, TLC, Platelet, Serum iron in cord, TIBC in cord and CRP in cord,, and there was highly statistically significant difference found between two groups regarding cord ferritin Table (4).

Table (1): Comparison between Mother Obese and Mother Non Obese regarding Age, Education level, Smoking, History of gestational diabetes, History of pre-eclampsia, gravidity & parity.

		Obese No.= 37	Non Obese No.= 63	Test value	P-value	Sig.
Age(years)	Mean ± SD	26.54 ± 2.83	26.84 ± 2.80	-0.516•	0.607	NS
	Range	19 – 31	19 – 31			
Education level	High	16 (24.0%)	50 (75.0%)	6.113	0.003	HS
	Low	20 (58.8%)	14 (41.1%)			
Smoking	No	37 (100.0%)	63 (100.0%)	–	–	–
	Yes	0 (0.0%)	0 (0.0%)			
History of gestational diabetes	No	37 (100.0%)	63 (100.0%)	–	–	–
	Yes	0 (0.0%)	0 (0.0%)			
History of pre-eclampsia	No	37 (100.0%)	63 (100.0%)	–	–	–
	Yes	0 (0.0%)	0 (0.0%)			
Gravidity & parity	G1P1+0	7 (18.9%)	15 (23.8%)	7.607*	7.607	NS
	G2P1+0	1 (2.7%)	0 (0.0%)			
	G2P2+0	5 (13.5%)	7 (11.1%)			
	G3P2+1	2 (5.4%)	1 (1.6%)			
	G3P3+0	8 (21.6%)	10 (15.9%)			
	G3P3+1	1 (2.7%)	0 (0.0%)			
	G4P2+2	3 (8.1%)	10 (15.9%)			
	G4P3+1	0 (0.0%)	1 (1.6%)			
	G4P4+0	0 (0.0%)	1 (1.6%)			
	PG	10 (27.0%)	18 (28.6%)			

P-value >0.05: Non significant (NS); P-value <0.05: Significant (S); P-value< 0.01: highly significant (HS)
•: Independent t-test, ß: Mann-Whitney test, *: Chi-square test

Table (2): Comparison between Obese Mother and Non Obese Mother regarding Gestational age at delivery, Sex of baby, Large or small for GA and Brith wt. (KG)

		Obese No.= 37	Non Obese No.= 63	Test value	P-value	Sig.
Gestational age at delivery(weeks)	**Mean ± SD**	39.7 ± 1	40± 1	11.410	0.000	HS
	Range	38.7– 40.9	39.3– 41			
Sex of baby	**Female**	16 (43.2%)	28 (44.4%)	0.073*	0.787	NS
	Male	21 (56.8%)	35 (55.5%)			
Large or small for GA	**Large**	11 (29.5%)	10 (15.8%)	2.701*	0.259	NS
	Small	9 (24.5%)	18 (20.3%)			
	normal	17 (45%)	35 (55.5%)			
Brith wt (KG)	**Mean ± SD**	1.75 ± 0.40	1.82 ± 0.42	-0.883•	0.379	NS
	Range	1.1 – 3	1.16 – 3.32			

P-value >0.05: Non significant (NS); P-value <0.05: Significant (S); P-value< 0.01: highly significant (HS)
•: Independent t-test, ß: Mann-Whitney test, *: Chi-square test

Table (3): Comparison between Obese Mother and Non Obese Mother regarding maternal Hb, TLC, PCV, PLATELET COUNT, Hepcidin, CRP, Serum iron, TIBC, UIBC, Serum ferritin, Transferrin receptor.

		Obese No.= 37	Non Obese No.= 63	Test value	P-value	Sig.
HB(g/dL)	**Mean ± SD**	11.31 ± 1.17	11.35 ± 1.12	-0.186•	0.853	NS
	Range	10 – 14.6	10 – 14.6			
TLC	**Median (IQR)**	8400 (6800 – 10000)	8900 (7500 – 10900)	-1.868 ß	0.062	NS
	Range	3100 – 11200	3100 – 11700			
PCV	**Mean ± SD**	34.78 ± 4.36	33.88 ± 4.65	0.955•	0.342	NS
	Range	25 – 44	22.4 – 44			
PLATELET COUNT	**Mean ± SD**	215135.14 ± 59026.44	237888.89 ± 52241.10	-2.004•	0.048	NS
	Range	106000 – 345000	106000 – 345000			
Hepcidin(ng/mL)	**Mean ± SD**	10.48 ± 2.56	8.82 ± 2.64	9.375•	0.013	S
	Range	4.60 – 12.50	4.40 – 14.20			
CRP(mg /L)	**Mean ± SD**	6.56 ± 1.53	5.11 ± 1.68	4.316•	0.000	HS
	Range	1.4 – 8.4	1.4 – 8.3			
Serum iron(µg/dL)	**Mean ± SD**	114.97 ± 40.21	102.49 ± 37.46	1.565•	0.121	NS
	Range	63.2 – 303.09	42.75 – 303.09			
TIBC(µg/dL)	**Mean ± SD**	307.96 ± 79.38	335.06 ± 72.57	-1.741•	0.085	NS
	Range	118.9 – 525	118.9 – 525			
UIBC(µg/dL)	**Mean ± SD**	234.74 ± 75.42	263.00 ± 93.18	-1.567•	0.120	NS
	Range	113 – 485.3	113 – 485.3			
Serum ferritin(ugl/L)	**Median (IQR)**	19.8 (16.1 – 43)	22.15 (16.8 – 34)	-0.738 ß	0.461	NS
	Range	8.7 – 67.8	7.9 – 65.6			
Transferrin receptor(nmol/L)	**Median (IQR)**	1.3 (0.91 – 1.51)	0.98 (0.52 – 1.32)	-2.682 ß	0.007	HS
	Range	0.42 – 1.59	0.38 – 1.55			

P-value >0.05: Non significant (NS); P-value <0.05: Significant (S); P-value< 0.01: highly significant (HS)

•: Independent t-test, ß: Mann-Whitney test

Table (4): Comparison between Obese Mother and Non Obese Mother regarding neonatal Hb, TLC, Plt , Serum iron in cord, Serum ferritin in cord, TIBC in cord and CRP in cord.

		Obese No.= 37	Non Obese No.= 63	Test value	P-value	Sig.
Neonatal Hb (g/dL)	Mean ± SD	16.34 ± 1.73	16.27 ± 2.06	0.173•	0.863	NS
	Range	13.2 – 22	12 – 22			
Neonatal TLC	Median (IQR)	11.3 (9.5 – 15)	11.4 (9.4 – 15.3)	-0.107 ß	0.915	NS
	Range	8.2 – 316	8.2 – 25			
Neonatal Plt	Mean ± SD	253.14 ± 64.86	263.07 ± 76.32	-0.663•	0.509	NS
	Range	18.3 – 368	18.3 – 519			
Serum iron in cord(µg/dL)	Median (IQR)	65.5 (43.7 – 77.4)	58.7 (27.8 – 106)	-0.139 ß	0.889	NS
	Range	10.8 – 187.6	10.8 – 203.5			
Serum ferritin in cord(ugl/L)	Median (IQR)	34.70 (32.0 – 55.80)	109.80 (32.80 – 143.20)	9.167 ß	0.003	HS
	Range	12.80 – 149.20	12.60 – 183.10			
TIBC in cord(µg/dL)	Median (IQR)	348 (223 – 465)	376.3 (264 – 465)	-0.925 ß	0.355	NS
	Range	123 – 545	170 – 556			
CRP in cord	Median (IQR)	1.18 (0.89 – 2)	1.21 (0.39 – 2.23)	-0.004 ß	0.997	NS
(mg /L)	Range	0.02 – 3.3	0.02 – 3.3			

P-value >0.05: Non significant (NS); P-value <0.05: Significant (S); P-value< 0.01: highly significant (HS)

•: Independent t-test, ß: Mann-Whitney test

Discussion

Prevalence of obesity among women of reproductive age is increasing worldwide, with current estimates of 20–36%. As the prevalence increases among the women of reproductive age group, so it does among pregnant females

Regarding to the maternal history there was no statistically significant difference found between two groups in maternal age, and gravidity & parity, and there was highly statistically significant difference found between two groups regarding education level, the obesity prevalence in pregnant women increase with decreasing years of education, there was 66 highly educated mothers (16 obese and 50 non obese) and 34 mothers with low education (20 obese and 14 non- obese).

An inverse association between obesity and education level among the elderly was revealed, which means that obesity prevalence and the odds of being obese increase with decreasing years of education (5).

All infant characteristics (Sex of baby, Large or small for GA and birth weight) were similar between obese and normal weight women, apart from gestational age at delivery, which was greater in non-obese pregnant women, the mean gestational age in obese pregnant mothers 39.7 with SD.± 1 and 40.3 with SD±1 in non-obese pregnant mothers.

Similar to our results, is a study performed previously on 245obese women and 245 non-obese women showed that there is association between obesity and gestational age at delivery, which was greater in non-obese pregnant women (6).

Regarding neonatal Hb, our results shows that the mean of neonatal Hb was 16.29 g/dl with SD±1.94, the median of neonatal TLC was 18.80 with IQR (9.40 – 15.25) and the Mean of neonatal Plt. was 259.40 with SD ± 72.11.

Our study shows that there was no statistically significant difference found between obese and non-obese pregnant women regarding neonatal *HB, TLC, Platelet , Serum iron in cord, TIBC in cord and CRP in cord.*

Our study showed that there was a negative impact of maternal BMI on neonatal iron status. The maternal obesity was inversely associated with cord serum ferritin concentrations, poorer iron status determined by cord ferritin were evident in the neonates of obese women, compared with those born to normal weight women.

In agreement with the present study results, it was found that cord ferritin was lower in the neonates born to obese women, and more neonates born to obese women had depleted iron stores (Ferritin < 76 μg/L) compared with neonates born to non-obese pregnant women **(6)**.

It was found that compared to non-obese pregnant women (BMI < 30 kg/m^2), obese women delivered offspring with poorer iron status, as assessed using cord ferritin **(7)**.

In an analysis of the SCOPE-BASELINE pregnancy and birth cohort study, it was shown that maternal obesity at 15 weeks of gestation was inversely associated with cord ferritin concentrations **(8)**.

It was shown that poorer iron status determined by serum ferritin in the cord was an evident in the neonates of obese women, compared with those born to normal weight women **(9)**.

Our study show that maternal CRP was greater in obese compared with normal weight pregnant women and Serum hepcidin was elevated in obese pregnant women compared with the normal weight pregnant

women but We found no relationship between CRP and hepcidin .

Similar results were founded that **t**he concentrations of CRP were greater in obese compared with normal weight pregnant women **(10)**.

In a group of studies it was shown in their result that the inflammatory markers as CRP as one of a pro-inflammatory cytokine, is frequently elevated in obese pregnant women compared with non- obese pregnant women due to many environmental factors which have been known to elicit the chronic elevation of CRP independently of genetic factors. These environmental insults include infections, tissue injury, pollutions , social economic status (SES), dietary content ,stress, unhealthy lifestyles, and physical activities **(11, 2 and 12).**

Serum hepcidin was elevated in obese pregnant women compared with the normal weight women with the evidence of a higher grade of obesity was associated with increased hepcidin. (**10)**

Inflammation, such as obesity in pregnant women, may lead to hepcidin excess and decreased iron transfer to the fetus (**6**)

In a healthy pregnancy, hepcidin is reduced, enabling increased iron transfer to the fetus. It follows that obesity in pregnancy may lead to hepcidin excess and decreased iron transfer to the fetus. The relationship between maternal obesity, inflammation, and iron status has not been extensively examined, with recent conflicting results. **(13)**

While there was no difference in serum hepcidin concentration between obese and normal weight women, the authors conclusion that a high degree of obesity may be necessary to elicit a significant increase in hepcidin may explain the lack of hepcidin increase in Flynn study, in which the majority of obese women were Class I obese. (**6)**

In an exploratory analysis, we found no relationship between CRP and hepcidin in either obese or normal weight women. This might indicate that the association between inflammatory mediators and hepcidin is not extant in pregnancy. (**10)**

No relationship between CRP and hepcidin in obese or normal-weight women was found. It might indicate that the association between inflammatory mediators and hepcidin is not extant in pregnancy **(6)**.

CRP is frequently elevated in obesity, has been shown to induce expression of hepcidin, a negative regulator of intestinal iron absorption, macrophage iron efflux and mobilization of hepatic iron stores (**14).**

Our result found that sTfR is high in obese pregnant women more than non- obese pregnant women.

A longitudinal analysis of Chinese women, reported higher serum transferrin receptor in obese women at mid-gestation (approximately 20 weeks' gestation) compared with normal weight women, though the increase in serum transferrin receptor from mid to late gestation was less pronounced among obese women.**(15)**

In 23 obese and 25 normal weight women, a study (16) showed that serum transferrin receptor concentration increased more in the obese group over the gestational period. The current study confirms previous observations and reports that the association of obesity with maternal iron status is evident from as early as 15–18 weeks' gestation. (**16)**

One potential biological mechanism that could explain why higher iron status may be detrimental to the fetus is non–transferrin bound iron. Iron is typically carefully chaperoned around the body, predominately by transferrin. Unbound iron can result when the rate of iron influx into plasma exceeds the rate of iron acquisition by transferrin (**17).**

It is therefore possible that higher iron status may lead to oxidative stress via unbound iron, which may result in lipid peroxidation and DNA damage of placental cells and impair the systemic response to infection, compromising the growth of the fetus. There is some evidence that a modest increase in plasma non–transferrin bound iron can occur after iron supplementation in non-pregnant women, although it is unclear whether this is relevant in our study population. (**18)**

Conclusion

There is a negative impact of maternal BMI on neonatal iron status. The maternal obesity was inversely associated with cord serum ferritin concentrations, poorer iron status determined by cord ferritin were evident in the neonates of obese women, compared with those born to normal weight women. The concentrations of CRP was greater in obese compared with normal weight pregnant women. sTfR is high in obese pregnant women more than non-obese pregnant women. Serum hepcidin was elevated in obese pregnant women compared with the normal weight women in this study.

Recommendation:

It is recommended to do this study on wide scale on large number of patients to support our results and to investigate the effect of obesity on disturbances to iron metabolism in both mothers and infants**.**

Obesity is a growing health problem worldwide; Prevalence of obesity among women of reproductive age is increasing

worldwide so it does among pregnant females

Normal fetal growth and development is dependent upon maternal iron sufficiency during pregnancy, so it is better to Calculate the body mass index to all the pregnant women in our hospital, measure maternal iron status to obese pregnant mother at 15 weeks gestational age and measure serum ferritin in the cord to neonates delivered from obese pregnant mother at time of delivery.

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