Maternal and Neonatal Iodine Nutrition In Cairo

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Citation

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Abstract

Background Aims:
A neonatal screening program for hypothyroidism began in Egypt in 2000. A high percentage of transient congenital hypothyroidism was detected in some Governorates[1] in addition to a high prevalence of iodine deficiency in some rural areas[2].
This study was conducted to assess iodine nutritional status of Egyptian pregnant women and their newborns in Abbassia district.
Methods:
Measurement of urinary iodine, serum fT3, fT4 and TSH and thyroid ultrasound were done to 113 Egyptian healthy pregnant mothers and their newborns on the 3rd day after delivery.

Results:
Iodine deficiency was detected in 29.2% of mothers and 19.46% of newborns. There were positive correlations ($p<0.05$) between maternal and neonatal values of urinary iodine and thyroid volumes. The percentage of newborns with blood TSH >5mU/L was 8.85%. Forty six % of mothers consumed iodized salt with increase in severity of iodine deficiency with lack of salt iodization ($p<0.05$).
Conclusion:
Increasing dietary iodine intake during pregnancy is mandatory. The campaign of salt iodization in Egypt must be reinforced.

INTRODUCTION

Iodine deficiency is a global health problem [3,4]. Goiter has been known to exist in Egypt since Ancient times, Papyrus dating since 1500 BC reported thyroidectomy and there are suggestions that Cleopatra had goiter. The first scientific report of goiter in Egypt was in 1924 by Dolbey and Omar[5].

Iodine deficiency is the world`s single most important cause of preventable brain damage and mental retardation. It manifests as goiter and a range of physical and mental handicaps which are collectively included in the term Iodine Deficiency Disorders (IDD)[6].

The best known indicators for assessment of IDD are: urinary iodine concentration, thyroid size (preferably by ultrasound) and TSH determination[7]. So, it is important to assess these parameters in pregnant women being vulnerable to iodine deficiency[6] .It is well known that urinary iodine measures the current dietary intake of iodine while prevalence of goiter gives an idea of the past history of iodine nutrition[8] .

Iodine supplements during pregnancy together with proper salt iodization relieve the stress of iodine deficiency of both maternal and fetal thyroid function, together with prevention of the frightening fetal brain damage that results from maternal iodine deficiency [9] .

This study was undertaken to assess the iodine nutritional status of Egyptian pregnant women and their newborns in Abbassia district.

METHODS

This cross-sectional study was conducted in Cairo Governorate in Abbassia district (West Cairo) during the period from March 2006 till December 2006. The target population was 113 healthy pregnant women (mean age: 28.97± 4.95 years) and their newborns (mean gestational age: 38.68 ± 1.82 weeks) among whom 47.4% were males and 52.6% females. Mothers were admitted for delivery at

Maternity Unit, Ain Shams University Hospitals, Abbasia district. They gave birth to apparently healthy singleton full term newborns. A written consent was obtained from all mothers. Screening was done by random sampling technique[10].

On the 3rd day after delivery, all mothers and their newborns were subjected to the following:

Full medical history including socioeconomic status according to El-Bouhy (1988)[11], history of intake of iodized salt and its type and exclusion of any thyroid disease or intake of any thyroid modifying drugs.

Collection of spot urine samples for iodine estimation (ug/dl) according to the method of Dunn etal, 1993[12]. In mothers, urinary iodine >10 ug/dl was considered adequate, from 5-9.9 ug/dl was considered mild deficiency, from 2-4.9 ug/dl moderate and < 2 ug/dl severe. In newborns, values >5ug/dl were considered adequate, from 3.1-5 ug/dl were considered mild deficiency , from 3-1.5 ug/dl moderate and values <1.5 ug/dl severe deficiency[13] .

Free T_3, free T_4 and TSH assay using Immulite Automated Immunoassay Analyzer [14].

Thyroid ultrasound using Acuson Computed Tomography device with a 7.5 mHZ, 6.25 cm linear transducer to determine thyroid volume. The volume of each lobe (ml) was calculated according to the formula of prolate ellipsoid and thyroid volume was calculated as the sum of volumes of the 2 lobes[15]. Goiter was considered in pregnant women if thyroid volume was >18 ml[13] and in newborns if thyroid volume was >1.5 ml [16] .

STATISTICAL ANALYSIS AND DATA MANAGEMENT

The data were statistically analyzed using SPSS statistical soft ware package, Echo soft corporation, USA, 2004. Description of quantitative variables was in the form of mean ± SD and range while that of qualitative variables was in the form of frequency and percentage. Chi-square test with cross tabulation was used to compare 2 categorized quantitative data. Pearson correlation coefficient (r-test) was used to rank different variables against each other. A p value of < 0.05 was considered significant.

RESULTS

Among pregnant mothers, 29.2% (33/113) were iodine deficient with mild degree being most frequent (21/113, 18.5%) followed by moderate (10/113, 8.84%) and lastly the severe deficiency in 1.77 (2/113). On the other hand, 19.46 % (22/113) of their newborns were iodine deficient with 14.15% (16/113) being mild, 4.42% (5/113) moderate and 0.88% (1/113) severe. Also, there was a positive correlation (r = 0.56, p<0.05) between maternal and neonatal urinary iodine values (Figure1). Goiter was detected by ultrasound in 10.6% of mothers (12/113) and in 7.07% of newborns (8/113) with a positive correlation between maternal and neonatal thyroid volumes (r = 0.68, p<0.05).

Figure 1

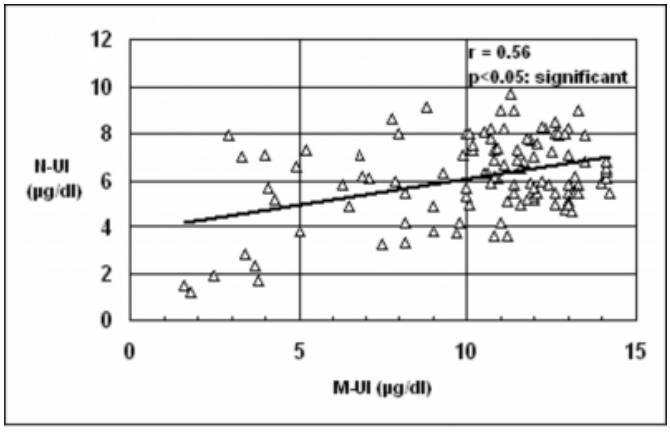
Table 1: Mean urinary iodine, thyroid function tests and thyroid volume among pregnant mothers and their newborns.

Parameter	Pregnant women Mean±SD (range)	Newborns Mean±SD (range)
Urinary iodine (ug/dl)	10.29 ± 3.11 (1.6 – 14.2)	6.13 ± 1.72 (1.2 – 9.7)
TSH (mU/L)	2.76 ± 2.17 (0.5-11.8)	3.87 ± 3.19 (0.56 – 16.6)
FT3 (pg/ml)	3.35 ± 0.68 (1.8 – 6.31)	3.57 ± 1.17 (0.72 – 7.01)
FT4 (ng/dl)	1.26 ± 0.28 (0.4 – 1.72)	1.89 ± 0.4 (0.89 – 2.6)
TV (ml)	10.85 ± 5.65 (4.91 – 31.2)	1.02 ± 0.99 (0.28 – 5.88)

TSH: thyroid stimulating hormone, fT_3: free triiodothyronine, fT_4: free tetraiodothyronine, TV: thyroid volume.

Figure 2

Figure 1: Regression analysis showing the correlation between maternal and neonatal urinary iodine.



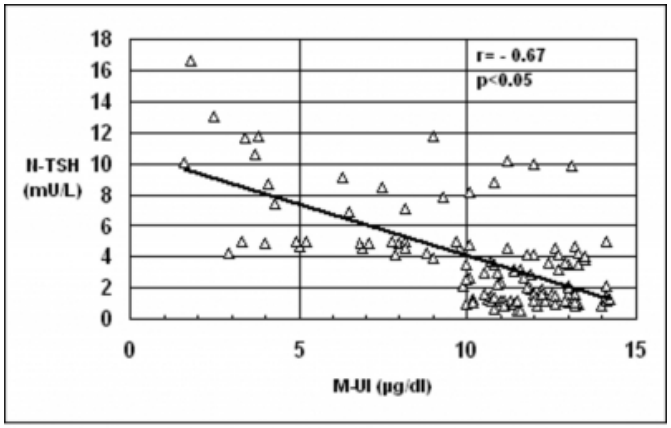
N-UI: neonatal urinary iodine, M-UI: maternal urinary iodine.

The overall prevalence of hypothyroidism among mothers with iodine deficiency was 15.15% (5/33) with 12.12% (4/33) being compensated. Also, positive correlations were detected between maternal and neonatal values of each of TSH (r = 0.5, p<0.05) and fT_3 (r = 0.8, p<0.05) but a non-significant correlation was detected between maternal and neonatal fT_4 (p>0.05). The percentage of newborns having a blood TSH level of >5 mU/L was 8.85% (10/113). Also,

there was a negative correlation (r= - 0.67, p<0.05) between maternal urinary iodine and neonatal TSH values (figure2). The percentage coverage of the use of iodized salt by pregnant mothers was 46.02 %. Among mothers who consumed iodized salt, 84.62% had normal urinary iodine excretion and 15.38% had iodine deficiency. On the other hand, among mothers who consumed non-iodized salt, 40.99% had iodine deficiency while 59.01% were non-deficient inspite of consumption of non-iodized salt. Also, there was an increase in severity of iodine deficiency with lack of salt iodization (p<0.05, table 2).

Figure 3

Figure 2: Regression analysis showing the correlation between maternal urinary iodine and neonatal TSH.



N-TSH: neonatal thyroid stimulating hormone, M-UI: maternal urinary iodine.

Figure 4

Table 2 : Percentage coverage of the use of iodized salt by mothers and its relation to severity of iodine deficiency.

Salt type	Severity of iodine deficiency (n=33)			Normal	X^2	P
	Severe (n=2)	Moderate (n=10)	Mild (n=21)			
Iodized (n=52)	0/52 (0%)	3/52 (5.77%)	5/52 (9.61%)	44/52 (84.62%)	21.73	<0.05
Non-iodized (n=61)	2/61 (3.29%)	7/61 (11.48%)	16/61 (26.23%)	36/61 (59.01%)		

p<0.05: significant.

DISCUSSION

In our study, the mean maternal urinary iodine on the 3rd day after delivery was 10.29 ± 3.11 (1.6–14.2 ug/dl) with 29.2% of mothers being iodine deficient. Mild degree was the most frequent (18.5%) followed by moderate (8.84%) and lastly severe degree (1.77%). Based on the criteria of WHO set in 1994[17] for adequate urine iodine concentrations in populations (mean value >10ug/dl and <20% of population with concentrations <5ug/dl), our sample is considered to have mild iodine deficiency.

IDD in Cairo has not changed much in the past few decades. In 1968, Abdou etal[18] reported a prevalence rate of 19.5%. In 1980, Said etal [19] found a rate of 13.3 %. In 1992, the Cairo Nutritional Institute and WHO[20] reported a rate of 5.2%. In 1995, El-Sayed etal[21] found a rate of 13.5%. In Alexandria, Hamed (1997)[22] found a rate of 19.4%.

The problem is more serious in upper Egypt and Delta region. In 1996, UNICEF and HIPH[23] found an IDD prevalence of 19.4% among primary school children in upper Egypt. After implementation of Universal Salt Iodization Program, UNICEF and HIPH[24] studied the IDD prevalence in New Valley (a desert oasis) in 1999 and the rate was 57.5% which was much lower than the rate reported by UNICEF and HIPH in 1993[25] (before implementation of salt iodization program). Lastly, Mansour etal in 2001[2] reported a rate of 31.9% and 60.1% in 2 Delta Governorates. Thus, the prevalence of IDD is still high despite the implementation of Universal Salt Iodization Program in Egypt.

The problem of IDD is also prevalent in other Eastern Mediterranean countries. In 2004, WHO/UNICEF/ICCIDD[26] studied the prevalence of IDD in the Eastern Mediterranean area where the highest rate was detected in Syria (70%) and the lowest in Tunisia (0.58%).

In our study, the mean neonatal urinary iodine was 6.13 ± 1.72 (1.2–9.7 ug/dl) which correlated positively with maternal urinary iodine values. This goes with the result obtained by Kurtoglu etal, 2004[27] who suggested that maternal iodine nutrition influences urinary iodine levels of their newborns. Moreover, maternal iodine deficiency affects fetal brain development through hypothyroidism [28, 29,30] .

In our sample, the mean maternal thyroid volume was 10.85 ± 5.65 ml (4.91 – 31.2 ml) and ultrasonographic evidence of goiter was detected in 10.6% of mothers. The finding of an increase in thyroid volume during the 3 trimesters of pregnancy and after delivery is consistent with previous reports from areas of low dietary iodine intake (~50 ug/day) in which the thyroid gland enlarges as an adaptation to the threat of iodine deficiency[31]. Moreover, a positive

correlation (P<0.05) was detected between maternal and neonatal thyroid volumes which was also confirmed by another study[18] .

The overall prevalence of hypothyroidism among mothers with iodine deficiency was 15.15% with 12.12% being compensated. In normal pregnant women, the thyroid gland maintains euthyroidism with only minor fluctuations in the serum T_4 (transient decrease) and TSH (transient rise). However, in women with limited thyroid reserve, due to iodine deficiency, hypothyroidism can develop[32,33].

Also, a positive correlation (P<0.05) was detected between maternal and neonatal values of TSH and fT_3 which proves the fact that changes in thyroid functions as a result of maternal iodine deficiency directly influence fetal thyroid functions causing impaired fetal brain development indirectly by producing hypothyroidism in mother and fetus[34]. On the other hand, a non significant correlation (P>0.05) was detected between maternal and neonatal fT_4. 0ur finding was confirmed by some authors[28] and was opposite to others [27] .

In our series, the mean neonatal TSH was 3.87 ± 3.19 mU/L with 8.85% of newborns having a blood TSH level of >5mU/L. According to the criteria set by WHO/UNICEF/ICCIDD in 1994[35] to asses the severity of iodine deficiency based on frequency of elevated TSH concentrations in newborn screening programs, our sample is said to have mild iodine deficiency. Also, WHO/UNICEF/ICCIDD, 1994 [35] suggested that thyroid function in the newborn reflects fetal thyroid function. So, if iodine deficiency impairs fetal thyroid function, this should result in higher TSH concentrations in the newborn. Moreover, a negative correlation was detected between maternal urinary iodine and neonatal TSH confirming that maternal iodine deficiency is responsible for maternal and fetal thyroid dysfunction[33]. On the other hand, a non-significant correlation was detected by other authors [4,36] who suggested that raised TSH levels might be attributed to factors other than iodine deficiency such as transient congenital hypothyroidism.

Concerning the consumption of iodized salt, 46.02% of mothers in our sample consumed iodized salt and there was an increase in the severity of iodine deficiency with lack of salt iodization (P<0.05). The universal salt iodization program started in Egypt in 1996[37]. WHO, United Nations International Children's Emergency Fund and ICCIDD[38] emphasized the importance of periodic monitoring and adjustment of salt iodide concentrations especially in developing countries since salt iodization is arguably the most effective way to correct iodine deficiency in the long run[32].

Moreover, in our sample, among mothers who consumed iodized salt, 84.62% had normal urinary iodine levels while 15.38% had iodine deficiency inspite of consumption of iodized salt. This means that although adequate iodine intake is the main permissive factor in occurance of iodine deficiency and goiter, yet other factors as bacterial contamination, pollution, malnutrition and concomitant iron deficiency anemia might play a role[25].. Such factors are frequently encountered in rural areas and areas with poor socioeconomic conditions which is the situation in Abbassia district where our study was conducted as most of the population were of low social class[11]. Also, in Egypt, increased consumption of bread among lower social classes can be considered an important contributing source of dietary iodine deficiency[25]. Also, WHO in the year 2000[39] reported that in many developing countries including Egypt, despite improvement of salt production and marketing technology, the quality of salt is still poor, or the salt is incorrectly iodized or spoilt due to excessive exposure to moisture, light, heat and contaminants[25]. On the other hand, 59.01% of mothers in our sample were non-deficient inspite of intake of non-iodized salt which raises the role of genetic susceptibility in occurrence of iodine deficiency as suggested by Ghalioungui in 1965 [40] and Lisenkova etal, 1991[41] .

In conclusion, iodine deficiency remains a public health problem in Egypt. The campaign of salt iodization in Egypt must be reinforced and periodically monitored to achieve proper elimination of IDD with its frightening consequences. Successful iodine supplementation must target reproductive-age and pregnant women.

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