The Effect of Secondhand Smoke Exposure on Level of Maternal Thyroid Hormones

Sepideh Tadayon[1], Ziba Raisi Dehkordi[2], Lobat Jafarzadeh[3*]

[1]Resident of Obstetrics & Gynecology, School of Medicine, Shahrekord University of Medical Sciences, Shahrekord, Iran,
[2]MSc of Midiwifery, Shahrekord University of Medical Sciences, Shahrekord, Iran,
[3]Associate Professor, Department of Obstetrics and Gynecology, Shahrekord University of Medical Science, Shahrekord, Iran.

ABSTRACT

Background: Exposure to secondhand smoke in pregnancy has severe maternal and fetal effects. No study has been yet conducted to study the effects of secondhand smoke exposure on maternal thyroid hormones. Therefore, this study investigated the effect of exposure to secondhand smoke on the level of maternal thyroid hormones by women's self-report and urinary cotinine level at delivery. Methods: This retrospective, analytical, cohort study was conducted on 108 pregnant women who were randomly assigned, with equal probability, to two groups of 54, exposed and unexposed. The pregnant women exposed to secondhand smoke were non-smoking individuals with exposure to the cigarette smoke of an individual smoking every day (at home or workplace). In this study, the data were collected by the interview and questionnaires on lifestyle and exposure to secondhand smoke. Height, weight, serum levels of TSH, T4, and T3, and urinary cotinine levels in mothers were also measured. At the onset of pregnancy, body mass index (BMI) was measured by routine protocols and interviews. Results: T3 and T4 levels significantly decreased in the women with exposure to secondhand smoke by both their own self-reports and urinary cotinine levels. Maternal TSH level did not change significantly. A significant correlation between the women's self-report on exposure to secondhand smoke and their urinary cotinine levels was observed. Conclusions: Exposure to secondhand smoke led to increased maternal serum levels of T3 and T4. Therefore, it has been recommended to explain the risks and harms of smoking during the pregnancy to women, and prevent them from smoking and being exposed to secondhand smoke.

Key Words: *Exposure To Secondhand Smoke, Thyroid Hormones, Pregnancy*.

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INTRODUCTION

Smoking specially tobacco smoking is a threat to human's health and affects human life from birth to adulthood, during pregnancy, menopause, and aging, and also declines quality of life [1]. The prevalence of smoking has declined in developed countries while it is growing in developing countries [2]. According to the World Health Organization, the most prevalent smoking in Iran is tobacco smoking [3, 4]. Secondhand exposure to tobacco smoke threatens the health of half of the women and children worldwide [5]. Different rates of exposure to secondhand smoke in pregnant women have been reported in epidemiologic studies across the globe, including 13.5% in the United Kingdom [6], 35.9% in Brazil [7], and 69.1% in China [8]. In a study of Iran, exposure to secondhand smoke in pregnancy was reported 56.2% [9]. Exposure to secondhand smoke is measured by various methods including questionnaire-assisted self-report [10]. The studies on the effect of exposure to tobacco smoke in pregnancy have been mainly based on women's self-report, which has probably undermined the reliability of data [11]. Recently, use of cigarette smoke biomarkers such as cotinine has improved the reliability and accuracy

Corresponding author: Lobat Jafarzadeh
Address: Associate professor, Department of Obstetrics and Gynecology, Shahrekord University of Medical Science, Shahrekord, Iran.
E-mail: ✉ lobatjafarzadeh@gmail.com
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of the data of self-report assisted investigations [12]. cotinine is the most important and first metabolite derived from nicotine decomposition and could be measured in different parts of the body such as hair, saliva, meconium, urine, plasma [13], and breast milk [14]. cotinine is extensively used as a biological marker of exposure to tobacco smoke [15-17]. Few studies have been conducted to investigate the reliability of women's self-report on exposure to secondhand smoke in pregnancy by measuring urinary cotinine level at delivery [12]. Most studies have addressed the correlation of women's self-report on exposure to secondhand smoke with umbilical cotinine level [12, 18]. Exposure to secondhand smoke in pregnancy and prenatal period lead to adverse effects such as miscarriage [19], low birth weight [20, 21], placental abruption, premature rupture of fetal membranes, intrauterine growth restriction [22], and vaginal bleeding [17]. Some robust studies on direct tobacco smoke contribution to variations in thyroid function have been conducted [23, 24]. There are general data regarding the effect of exposure to secondhand smoke on thyroid [25], revealing that this type of exposure, irrespective of gender, leads to variations in thyroid function [26]. Chronic exposure to secondhand smoke leads to a decline in thyroid hormones' function [23, 26]. Thyroid hormones, i.e., thyroxine (T4) and triiodothyronines (T3), are derived from the amino acid tyrosine [27]. Disruption of the serum levels of these hormones leads to various disorders [28]. In the women with hypothyroidism, the rates of development of neuropsychiatric disorders, intrauterine growth restriction, low birth weight, fetal distress, miscarriage, fetal death, preterm delivery, early placental abruption, and preeclampsia are likely to increase [29, 30]. With regards to increase in exposure to tobacco smoke in 56.2% of Iranian women during pregnancy [31], prevalence of hypothyroidism and the associated complications among them [30], and lack of adequate data regarding the effect of tobacco smoke exposure on thyroid hormones in Iran, where the complications of exposure to second hand tobacco smoke in pregnancy have been studied only based on self-report, and because no study has yet been conducted to investigate the correlation between pregnant women's self-report and their cotinine levels, we were encouraged to investigate the effect of exposure to secondhand smoke on the levels of women's thyroid hormones according to their self-reports and urinary cotinine levels at delivery.

MATERIALS AND METHODS

This retrospective, analytical, cohort study was conducted on 108 pregnant women referred to a teaching hospital in Shahrekord, southwest Iran in 2014. The sampling was performed as convenience. The inclusion criteria were exposure to secondhand smoke, singleton pregnancy, lack of using any tobacco substance, lack of history of maternal thyroid disease, and fertility. The exclusion criteria were any medically diagnosed disease such as granulomatous disease, certain malignancies, diabetes, hypertension, thyroid disease, liver disease, kidney disease, bacterial and viral infections, or any chronic disease that affects the metabolism of thyroid hormones, taking any drugs affecting the thyroid hormones, such as corticosteroids, lithium, heparin, cimetidine, sulfonamides and phenyl to in during pregnancy, family history of hypothyroidism, and history of ovarian cysts. The women were randomly assigned, with equal probability, to two groups of 54 each, exposed and unexposed. The pregnant women exposed to tobacco smoke were nonsmoking individuals with exposure to the tobacco smoke of an individual smoking every day (at home or workplace). After the researcher introduced herself to the participants and explained the research purposes, the participants signed informed, written consent form and then, the data were collected through interview and by using the questionnaires of lifestyle and exposure to secondhand smoke. The questionnaire of lifestyle included sociodemographic information. The questionnaire of secondhand smoke exposure in pregnancy was filled out by the participants of the two groups at the beginning of the study. The validity and reliability of the questionnaire were also investigated. For this purpose, 10 experts were asked to judge the appropriateness of the items of the questionnaires to research purposes. After ensuring the content validity of the questionnaires, they were administered to 10 pregnant women twice with a 10-day interval. To calculate reliability coefficient, Kappa coefficient was derived 80%. The height and weight of the participants were recorded by routine protocols for measurement of body mass index (BMI). After completion of the questionnaires, a 5 ml serum sample was collected from the participants and transferred to the hospital laboratory for investigation of TSH, T3, and T4 levels. In addition, urinary sample of the participants was transferred to the hospital laboratory for investigation of maternal urinary cotinine and the results were expressed as ng/mL. Data analysis was done by the individuals who were blind to the status of secondhand smoke exposure in the participants. Data were analyzed by using independent *t*-test, chi-square, and Spearman correlation coefficient in SPSS 17.

RESULTS

The mean age of the participants was 29.04 years in the exposed group and 28.07 years in the unexposed group. There was no significant difference in education level, occupation, BMI, area of residence, and income status between the two groups (Table 1). Regarding spouse's education level (p=0.01) and occupation (p=0.029), there was no significant difference between the two groups. Moreover, regarding pregnancy history including gestational age, parity, number of children, and number of abortions there was no significant difference between the two groups. The TSH levels of the two groups based on the participants' self-reports were not significantly different, but there was a statistically significant difference in T3 and T4 levels between the two groups (p=0.001), so that T3 and T4 were lower in the exposed group than in the unexposed group. There was also a statistically significant difference in TSH based on urinary cotinine levels between the two groups (p=0.001), but no statistically significant difference was observed in T3 and T4 was based on urinary cotinine levels between the two groups (p=0.001), so that T3 and T4 levels were lower in the exposed group than in the unexposed group. The data indicated a significant correlation between the participants' self-report and urinary cotinine levels (sensitivity: 98%, specificity: 100%, predictive value: 100%, and negative predictive value: 98%)

DISCUSSION

The findings of the present study indicated a statistically significant difference in maternal T3 and T4 levels between the exposed and unexposed groups, while maternal TSH level was not significantly different between the two groups. The findings based the women's self-report and urinary cotinine cut point were similar. In the study of Woodward et al. [8], the maternal urinary cotinine was reported 309-315 ng/mL in the smoking women. In a study of tobacco smoke effect on thyroid function in male rats, the level of T3 and T4 hormones increased significantly in the rats exposed to tobacco smoke when compared to the control group. In addition, the serum levels of the two hormones increased significantly in the rats exposed to hookah smoke when compared to the control group, and the serum TSH level decreased *in*significantly in the rats exposed to tobacco smoke and increased *in*significantly in the rats exposed to hookah smoke [32]. In a study on mice, the exposure to tobacco smoke did not cause any decrease in T3 and T4 [33]. The study of Shield et al. in the United Kingdom, indicated that the level of T3 and T4 increased significantly in smoking women when compared to nonsmoking women, while TSH level decreased significantly in smoking women [34]. In the study of Georgos et al. in Greece, urinary and serum cotinine levels became positive after an hour of exposure to tobacco smoke and T3 and T4 hormones increased significantly when compared to before exposure to tobacco smoke, but other thyroid parameters did not change after the intervention [35]. In the study of Mc Donald et al. on tobacco smoke effect on maternal and fetal thyroid hormones at delivery, TSH level was observed to decrease significantly in the exposed group when compared to the unexposed group while other thyroid hormones in the two groups were not different [36]. Physiopathologically, the mechanism of such effect is mainly unknown, but sympathetic activation induced by nicotine in tobacco or hookah smoke may the cause of increased secretion of thyroid hormones. However, some studies have indicated that nicotine injection does not affect the level of thyroid hormones [37]. On the other hand, the effects of nicotine in tobacco or hookah smoke can be exerted through the central nervous system. In this regard, some studies have shown that tobacco contains monoamine oxidase inhibitors; monoamine oxidase leads to decomposition of certain neurotransmitters such as monoaminergic, dopamine, and norepinephrine. On the other hand, studies have shown that dopamine affects the hypothalamus and therefore pituitary, and then influences thyroid function [36]. As a result, it can be argued that the nicotine in tobacco or hookah smoke may cause an increase in dopamine and norepinephrine by producing inhibitory effect on monoamine oxidase, resulting in increased thyroid function and serum levels of T3 and T4. With regards to lack of a change in serum TSH level in the present study, the variations in serum level of these hormones and the study duration might not have sufficed to cause a significant change in serum TSH level [35].

Regarding the accuracy of self-report on exposure to tobacco smoke in pregnancy, there was a correlation between the participants' self-report and urinary cotinine level(sensitivity: 98%, specificity: 100%, predictive value: 100%, and negative predictive value: 98%), representing the reliability of the results from the women's reports on the rate of exposure to secondhand smoke. Similarly, Chio et al. investigated exposure to tobacco smoke in pregnant women using questionnaire and its correlation with umbilical cotinine level, and concluded that the pregnant women's self-report on exposure to tobacco smoke was highly correlated with umbilical cotinine level [37]. Jederiosky et al. reported a high correlation between umbilical cotinine level and the pregnant women's self-report on tobacco smoke exposure [38]. In another study, 98% sensitivity, 100% specificity, 100% predictive value, and 98% negative predictive value were reported for the pregnant women's self-report on exposure to tobacco

smoke and their urinary cotinine levels [5]. The present study's findings on the effect of tobacco smoke on maternal and fetal thyroid hormones at delivery, could serve as a basis for future studies and pave the way to develop interventions to enhance prohibition of smoking in the families with pregnant women and infants and thus promote family health.

CONCLUSION

In light of the present study's findings and the effect of the exposure to tobacco smoke on maternal T3 and T4 hormones and the reliability of self-report results, special attention should be paid to the vulnerability of pregnant women to environmental tobacco smoke and the importance of the prohibition of smoking at home. It is also recommended that the necessary guidelines concerning the prohibition of smoking at home and in car be incorporated into national health planning, and the harms caused by exposure to tobacco smoke be highlighted in smoking control programs. The present study that investigated the effect of tobacco smoke on maternal and fetal thyroid hormones at delivery for the first time, could lead to further investigations and provide the background to develop interventions to improve prohibition of smoking in the families with pregnant women and infants and hence promote family health. Enrollment of a small sample size as compared with similar studies, cross-sectional design, and lack of a prospective, cohort design were some of the current study's limitations.

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Table 1. Individual characteristics of women in two groups of exposed and unexposed

Group / Characteristics	Exposed to smoke (n=54)	Unexposed to smoke (n=54)	P value

Women's age (yr) Range of variation	5.69±29.04 19-39	4.9±28.7 18-40	0.384
Women's education level Illiterate Primary Guidance Highschool Diploma Academic	(9.3)5 (8.14)8 (20.3)11 (14.8)8 (35.2)19 (5.6)3	0 (9.2)5 (20.4)11 (5.6)3 (51.8)28 (13)7	0.75
Women's occupation Employed Housewife	(1.9) 1 (98.1) 53	(11.1) 6 (88.9) 48	0.08
Spouse's education level Illiterate Primary Guidance Highschool Diploma Academic	(5.6)3 (31.4)17 (31.4)17 (5.6)3 (22.4)12 (3.8)2	(7.4)4 (31.4)17 (9.3)5 (9.3)5 (46.3)25 (5.6)3	0.01
Spouse's occupation Civil servant Labourer Jobless Others	(5.6)3 (25.9)14 (11.41)6 (57.4)31	(20.4)11 (13)7 (3.71)2 (63)34	0.029
Body mass index Less than 18.5 (low weight) 18.5-24.9 (normal) 25-29.9 (overweight) 30-39.9 (obese) Over 40 (extra obese)	(3.7)2 (66.1)33 (35.9)14 (9.3)5 0	(5.6)3 (57.4)31 (29.6)16 (7.4)4 0	0.917
House area (m^2)	20.31±64.94	31.87±72.75	0.19
Income adequacy Adequate Relatively adequate Inadequate	(24.1)13 (55.6)30 (20.3)11	(48.1)11 (40.7)22 (11.2)6	0.09

Table 2. Characteristics of pregnancy history of women in two groups of exposed and unexposed

Group / Variable*	Exposed to smoke (n=54)	Unexposed to smoke (n=54)	† P value
Gestational age (week) Range of variation	0.8±39 40-37.6	0.72±39.02 40-37.5	0.151
Parity 1 2 3 and more	(40.7)22 (22.3)12 (37)20	(37)20 (33.4)18 (29.6)16	0.419
No. of children 0 1 2 3 and more	(42.6)23 (37)20 (7.4)4 (13)7	(48.1)26 (42.6)23 (7.4)4 (1.9)1	0.18
No. of abortions 0 1 Two and more	(79.9)41 (16.7)9 (7.4)4	(74.1)40 (22.2)12 (3.7)2	0.577

Table 3. Serum level of T3, T4, and TSH of women in two groups of exposed and unexposed per self-reports

Group / Variable*	Per women's self-report: Unexposed to smoke (n=54) Mean±standard deviation	Per women's self-report: Exposed to smoke (n=54) Mean±standard deviation	Pvalue
TSH (µg/dL)	1.35±2.22	1.41±2.12	0.68
T3 (µg/dL)	0.56±1.3	0.33±1.84	0.0001
T4 (µg/dL)	3.03±9.07	1.33±10.87	0.0001

Table 4. Serum level of T3, T4, and TSH of women in two groups of exposed and unexposed per urinary cotinine level

Group / Variable*	Per urinary cotinine cutpoint (ng/mL): Unexposed to smoke (n=54) Mean±standard deviation	Exposed to smoke (n=54) Mean±standard deviation	P value
TSH (µg/dL)	1.35±2.26	1.41±2.17	0.49
T3 (µg/dL)	0.56±1.31	0.33±1.84	0.0001
T4 (µg/dL)	1.23±9.97	3.94±10.01	0.0001

Table 5. Accuracy of self-reports on exposure to cigarette smoke per urinary cotinine level

Maternal urinary cotinine cutpoint	Exposure to cigarette smoke: Per women's self-report: No	Yes	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Unexposed (≤7)	(98.1)53	(100)54	98%	100%	100%	98%
Exposed (> 7)	(1.1)1	-				
Total	(100)54	(100)54				