Effects of passive smoking on outcome in pregnancy

Goel P, Radotra A, Singh I, Aggarwal A, Dua D

Department of Obstetrics and Gynaecology, Government Medical College and Hospital, Chandigarh - 160029, India.

Correspondence:
Poonam Goel, MD
E-mail: pgoel@glide.net.in

ABSTRACT

Background: Adverse health effects of exposure to environmental tobacco smoke (ETS) among non-smokers have been studied occasionally in developing countries.

Aims: To study the effects of exposure to ETS on outcome in pregnancy

Settings and Design: A cross-sectional study at a secondary level teaching hospital

Material and Methods: Consecutive 576 non-smoking women delivering a singleton live baby were studied. A pre-designed structured questionnaire was used to record the details of exposure to ETS at home. The maternal and foetal variables were compared among those who were exposed to ETS vis-à-vis not exposed. Unpaired Student t-test was used for the comparison of continuous variables and Fisher's Exact test was used for categorical variables. Multiple logistic regression analysis was performed after including all variables found to have significant differences on univariate analysis.

Results: Of the 576 women studied 141 (24%) were exposed to ETS. In the mothers exposed to ETS, there was a significantly higher incidence of pre-term birth (24.1% vs. 16.1%; $P = 0.027$) and small-for-gestation babies (31.9% vs.17.2%; $P < 0.001$) as compared to unexposed mothers. The mean birth weight of the babies born to the mothers exposed to ETS was 138 g less than that of babies in the unexposed group (2632 $\pm$577 g vs. 2770 $\pm$562 g respectively, $P = 0.014$). The multiple logistic regression analyses showed that ETS exposure during pregnancy was significantly associated with a higher risk of small-for-gestation babies (OR 2.10; 95% CI: 1.27- 3.48).

Conclusion: Exposure to ETS during pregnancy is associated with higher risk of having a small-for-gestation baby.

KEY WORDS: Passive smoking, environmental tobacco smoke, pregnancy, outcome, small-for-gestation

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Exposure to environmental tobacco smoke (ETS) has been variously described as passive smoking, 'second-hand smoke' or involuntary smoking and is recognised as a major health hazard.[1,2] The International Consultation on Environmental Tobacco Smoke (ETS) and Child Health concluded that maternal smoking during pregnancy is a major cause of reduced birth weight, decreased lung function and sudden infant death syndrome (SIDS).[3] Exposure to ETS among non-smoking pregnant women can cause a decrease in birth weight. Most of the evidence for these conclusions was drawn from studies conducted in the Western and developed countries. A study from Nagpur, India found maternal 'tobacco exposure' to be significantly associated with low birth weight in babies.[4]

ETS is a combination of side-stream smoke that is emitted from the burning end of a cigarette and the mainstream smoke exhaled by the smoker. Side-stream smoke constitutes about 85% of the smoke present in the room and contains many potentially toxic gases in higher concentrations than in the mainstream smoke.[5] ETS has been classified as a class A (known human) carcinogen along with asbestos, arsenic, benzene and radon gas.[6] Active smoking is a well-established cause of intra uterine growth retardation (IUGR) among humans.[7-9] The underlying mechanisms for these effects are yet to be elucidated. It is possible that the carbon monoxide in the smoke could be combining with haemoglobin, in turn, leading to foetal hypoxia. In addition, nicotine in the smoke could be causing vasoconstriction further aggravating foetal hypoxia. It has also been proposed that nicotinic effects may be due to the consequences of inappropriate stimulation of nicotine cholinergic receptors and its neuroteratogenicity.[10]

The association of ETS with adverse foetal and reproductive outcomes has been the subject of two recent publications.[11,12] Smoking is on the rise in developing countries.[13] Environmental conditions like overcrowding may make the health effects of ETS more pronounced. The adverse health effects of passive smoking have been studied in developing and underdeveloped countries only occasionally. Most of the studies from India are on the pulmonary effects of passive smoking.[14] In this study we examined the effects of exposure to ETS on the maternal and foetal outcome in pregnancy.

Material and Methods

The study was conducted at a large secondary care teaching hospital

in North Western India. The study group comprised 576 consecutive non-smoking women delivering a singleton live baby. Following the delivery, each woman was interviewed according to a pre-designed structured questionnaire to enquire if they had been exposed to ETS at home. The questionnaire recorded details of the average daily exposure, besides enquiring into their knowledge and perceptions about passive smoking. Obstetric and medical details of the mothers were noted from hospital records, as were the details of the baby.

Based on the history of exposure to ETS the study population was divided into two groups. The maternal and foetal variables were compared in the subjects exposed to ETS with those who were not exposed. The exposure to ETS was quantified by calculating a cumulative "exposure index", which was a product of the number of smokers in the house to whom the case was exposed, average number of bidis/cigarettes smoked per day by them in the presence of the case and the period of gestation in weeks. For the purpose of studying dose response, the exposed group was divided into four categories according to the quartiles of the exposure index.

Preterm birth was defined as delivery at 37 weeks of gestation or less.

A small-for-gestation baby was defined as birth weight less than 10th percentile of weight for that gestational age.

Low birth weight (LBW) was defined as birth weight less than 2.50 kg.

Pregnancy-induced hypertension (PIH) in the antenatal period was defined as two records of systolic blood pressure of 140 mmHg or above and/ or diastolic blood pressure of 90 mmHg or above at least 6 hours apart after 20 weeks of gestation.

Anaemia was defined as haemoglobin concentration less than 11 gm/dl in the first and third trimester or haemoglobin concentration less than 10.5 gm/dl in the second trimester.

SPSS (Ver 10.0) was employed for data analysis. Unpaired Student t-test was used for the comparison of continuous variables and Fisher's Exact test was used for the comparison of categorical variables. Multiple logistic regression analysis was performed after including all co-variables found to have significant differences on univariate analysis for categorical outcome variables. For continuous outcome variables multiple linear regression modelling was done on similar lines.

Results

Of the 576 women studied 435 (75.6%) did not have any exposure to tobacco smoke (Group I) and 141 (24.4%) reported exposure to ETS from the husband and other family members (Group II). The mean age of the pregnant women, in both the groups, was comparable. Table 1 shows the detailed comparison of all the maternal variables studied in the two groups. The women exposed to ETS had higher parity and a greater number of live births as compared to women not exposed to ETS. The women exposed to ETS were less educated as compared to those not exposed to ETS (11.3% vs. 4.8% illiterates and 16.3% vs. 45% graduates or above, respectively) and fewer were employed. Among the antenatal problems, there was a significantly higher incidence of anaemia among the group exposed to ETS *(P=0.003)*. The median exposure index in the study group was 115.8 (range 31 to 2880, inter-quartile range 133.5).

There was no significant difference in the mean period of gestation, however the incidence of pre-term birth was significantly higher among the women exposed to ETS (Table 2). The onset of labour (spontaneous or induced) and the mode of delivery (vaginal or caesarean section) were similar in the two groups. More babies born to mothers exposed to ETS were small-for-gestation and their mean birth weight was 138 g less than that of babies in the unexposed group of mothers (2632

Table 1: Antenatal profile of mothers exposed to environmental tobacco smoke vis-à-vis not exposed mothers

Characteristics	ETS not exposed (n=435)	ETS exposed (n=141)	*P value*
Age: years Mean (SD)	25.34 (3.62)	25.90 (4.26)	0.131
Pregnancies: Mean ± SD			
Gravida	1.75 ± 0.25	2.08 ± 1.47	0.003
Abortions	0.99 ± 0.97	0.33 ± 0.97	0.240
Live	0.56 ± 0.40	1.17 ± 0.70	0.004
Education:			<0.001
Illiterate	21 (4.8)	16 (11.3)	
School Only	218 (50.1)	102 (72.3)	
University Graduate	196 (45.1)	23 (16.3)	
Occupation:			0.007
Employed	58 (13.3)	8 (5.7)	
Not employed	377 (86.7)	133 (94.3)	
Residence			
Rural	157 (36.1)	60 (42.6)	
Urban	278 (63.9)	81 (57.4)	0.101
Antenatal Problems			
Hypertension	88 (20.2)	23 (16.3)	0.221
Anaemia	128 (29.4)	60 (42.6)	0.003
Abruptio-placentae	2 (0.5)	0	0.570
Placenta Praevia	4 (0.9)	0	0.324
Exposure Index*Median (Range)	–	115.8 (31-2880)	

*Exposure index: product of number of smokers in the house to whom the case was exposed, average number of bidis/cigarettes smoked per day by them in the presence of the subject and the period of gestation in weeks. Figures in parentheses indicate percentages

Table 2: Outcome of pregnancy in mothers exposed to environmental tobacco smoke vis-à-vis not exposed mothers

Variable	ETS not exposed (n = 435)	ETS exposed (n = 141)	*P value*
Period of gestation: weeks, mean ± SD	37.99 ± 2.29	37.60 ± 2.76	0.092
Preterm birth:	71 (16.3)	34 (24.1)	0.027
Labour:			0.085
Spontaneous	343 (78.9)	110 (78.0)	
Induced	92 (21.1)	31 (22.0)	
Delivery:			0.482
Normal Vaginal Delivery	278 (63.9)	85 (60.3)	
Caesarean section	157 (36.1)	56 (39.7)	
Birth Weight mean ± SD (gm)	2770 ± 0.561	2632 ± 0.577	0.014
Low Birth Weight	110 (25.3)	46 (32.6)	0.089
Small-for-gestation	75 (17.2)	45 (31.9)	<0.001
APGAR 1: Mean ± SD	8.73 ± 0.83	8.52 ± 1.07	0.018
APGAR 5: Mean ± SD	9.18 ± 4.38	8.97 ± 0.49	0.566
Congenital Malformations	8 (1.8)	3 (2.1)	0.735

Figures in parentheses indicate percentages

± 0577 g vs. 2770 ± 0562 g respectively, *P=0.014*). Though the incidence of LBW was more in the group exposed to ETS as compared to the unexposed group (32.6% vs. 25.3%), the differences were not significant. The mean APGAR in these babies was poorer at one minute. There were no significant differences in the number of babies born with congenital malformations (Table 2).

The univariate and multivariate logistic regression analyses for outcome in relation to exposure to ETS are presented in Table 3. The crude odds ratios were significant for pre-term birth and small-for-gestation babies. The multiple logistic regression analyses, including all the cofactors and confounders that were significant in the antenatal period, showed that exposure to ETS during pregnancy was significantly associated with a higher risk of small-for-gestation babies (OR 2.10; 95% CI: 1.27-3.48). The linear regression modelling for birth weight and APGAR at one minute did not show any significant association with exposure to ETS (adjusted $r^2 = 0.055$ and 0.011 respectively indicating a poor fit; variables entered in the model were age, education, occupation, birth order, number of live issues and anaemia).

A dose response relationship between the amount of exposure and risk of adverse outcome (small-for-gestation baby and preterm birth) was seen using logistic regression analysis. The exposure was categorized into four categories as per the quartiles. There was significant increase in the risk of small-for-gestation baby and pre-term birth with each category rise in the exposure index from no exposure to highest exposure (OR = 1.31; CI: 1.13 – 1.52 and OR =1.22; 95%CI: 1.05 – 1.43, respectively).

Table 3: Logistic regression analyses to assess the odds of adverse pregnancy outcome in relation to the ETS exposure

Outcome variable	Univariate analysis		Multivariate analysis	
	Crude OR	95% CI	Adjusted OR*	95% CI
Preterm birth	1.60	1.01 – 2.54	1.15	0.69 – 1.92
Induced labour	1.05	0.65 – 1.70	0.85	0.54 – 1.35
Caesarean section	1.17	0.78 – 1.75	0.98	0.63 – 1.52
Low birth weight	1.43	0.95 – 2.16	1.03	0.65 – 1.65
Small-for-gestation baby	2.25	1.43 – 3.55	2.10	1.27 – 3.48
Congenital malformation	1.16	0.20 – 4.92	1.27	0.33 – 5.15

*Odds ratio adjusted for age, education, occupation, birth order, number of live issues and anaemia

Table 4: Knowledge and perceptions about passive smoking in 576 pregnant women

Question	ETS not exposed (n = 435)	ETS exposed (n=141)	*P value*
	N (%)	N (%)	
Do you know the difference between active and passive smoking?			
Yes	55 (12.6)	14 (9.9)	0.457
No	380 (87.4)	127 (90.1)	
Is smoking harmful during pregnancy?			
Yes	349 (80.2)	115 (81.6)	0.081
No	15 (19.8)	10 (18.4)	
Is exposure to tobacco smoke from others also harmful during pregnancy?			
Yes	269 (61.8)	91 (64.5)	0.535
No	96 (22.1)	25 (17.7)	

The knowledge and perceptions about passive smoking were similar in the two groups (Table 4). The majority of the women studied did not know the difference between active and passive smoking. More than 80% of women in both groups felt that smoking was harmful for the foetus and about two-thirds felt that exposure to tobacco smoke from others could be harmful to the developing foetus. Fifty-seven out of 141 (40.7%) women who were exposed to ETS during pregnancy reported exposure to ETS from parental smoking before marriage, as compared to 52 out of 435 (12.0%) women who reported no ETS exposure during pregnancy *(P<0.001)*.

Discussion

We have found in this study that exposure to ETS in pregnant women was significantly associated with a higher risk of small-for-gestation birth. Several epidemiological studies have shown similar effects. A meta-analysis of studies conducted before mid-1995 reported an overall RR of 1.2 (95 % CI, 1.1 to 1.3) for LBW at term or small-for-gestational age among the infants born to mothers exposed to ETS during pregnancy.[15] A recent small, case-control study found an association of IUGR with detectable nicotine level in mothers' hair samples. With non-smoking women whose hair nicotine concentrations were in the lowest quartile as the reference group, the odds ratio (OR) for small-for-gestational-age birth was increased among women with concentrations in the upper and two middle quartiles (ORs 2.1, 95% CI=0.4 - 10.1 and 3.4, 95 % CI=1.3 - 8.6 respectively).[16] The reported results were not adjusted for confounders, although the authors stated that several potential confounders had no effect. There are some studies that have utilized urinary cotinine levels as a biomarker of exposure and found a higher risk of IUGR in babies born to mothers exposed to ETS.[17,18]

One of the most difficult tasks in all studies on passive smoking has been to quantify the exposure to ETS. We have used self-reported exposure in this study for quantifying the exposure. The validity of self-reported exposure to ETS has been tested in a large multi-country multi-centred collaborative trial including Chandigarh, India, in which urinary cotinine levels were found to correlate with the history of exposure to ETS.[19] The study demonstrated that non-smoking women could provide appropriate estimates of their exposure which correlated well with their biochemically measured exposure levels. A recently published study has stated that self-reporting could be underestimating the low levels of exposure.[20]

We have also demonstrated a dose response relationship with a higher risk of pre-term birth and small-for-gestation babies with increasing cumulative exposure. The data in the literature on the dose response relationship, particularly in studies relying on self-reported exposure is confusing. While a few studies showed no indication of a greater effect at higher exposure levels,[21,22] some others suggested a greater effect.[23,24]

The mean birth weight of babies born to mothers exposed to ETS, in this study, was lower than that of babies born to unexposed mothers, though the differences were insignificant after multiple linear regression analysis. Studies correlating the urinary cotinine levels and birth weight have shown a deficit varying from 48 to 104 g in the babies born to mothers with higher urinary cotinine levels as compared to mothers with lower cotinine levels.[17,18] Similarly, the birth weights have been found to be lower in studies that have compared self-reported ETS exposure from all sources (home and work). After adjustment for potential confounders, most of the studies showed small to moderate decrements in mean birth weight (10 to 90 g) associated with ETS exposure.[25,26] Previous studies that have used the same methodology as ours have demonstrated that the differences in birth weight amongst infants born to ETS exposed and ETS non-exposed mothers vary from a decrement of 5 g to a decrement of more than 200 g.[27,28] There is limited data available from India. In a study from Vellore, India, it was shown that "Passive smoking was associated with a decrease in birth weight of 63 g (95% CI 12-114 g) even after adjusting for other variables known to affect birth weight".[29]

There are obvious difficulties in comparing the results of different studies because of their many differences, including the location and nationality of study populations, the sample size and selection, the extent to which confounders were controlled, and the analytical methods used. Smoking habits in India are peculiar. In India, bidi is the most popular product used for smoking. Bidis are made of crude sun-dried tobacco wrapped in a dried Tendu (Dyospyros melanoxylon) leaf. Another smoking product used in different parts of India is the chillum or hookah, which is like a pipe made of clay. Tobacco is burnt along with molasses and coal and smoked either directly at the other end or through a long pipe with smoke passing through a water container. The amount of nicotine and tobacco alkaloids present in the mainstream (MS) or sidestream (SS) smoke from such products is likely to be different than that known for standard cigarettes due to differences in tobacco processing, burning rate/temperature and design of the smoking product. In a study from Mumbai, bidi, an Indian cigarette and a brand of American cigarette were analysed by gas chromatography-flame ionization detection for the levels of nicotine and minor tobacco alkaloids in the MS and SS smoke.[30] The analysis demonstrated a higher content of nicotine and minor tobacco alkaloids in tobacco from bidi (37.7 mg/g) as compared to the Indian or American cigarettes (14–16 mg/g) studied. This study also demonstrated higher delivery of nicotine and alkaloids by the bidi as evidenced by higher concentration of nicotine in the MS smoke (MS/SS) for bidis as compared to cigarettes. Thus, there might be genuine qualitative and quantitative differences in the ETS in India as compared to the West.

Also, in the present study, it was generally seen that the women reporting exposure to ETS were less educated, had larger families (higher parity and more number of previous live issues) and were unemployed. This would point to a poorer socio-economic background of these mothers. This in itself could lead to lower birth weight secondary to malnutrition (there was a significantly higher incidence of anaemia). Overcrowding and poorly ventilated homes, particularly in the lower socio-economic strata of the society are also likely to enhance the ef-

fects of the ETS exposure. The differences in the incidence of small-for-gestation birth were significant even after adjusting for all possible confounders.

In our study, very few women actually knew the differences between active and passive smoking, though most women believed that smoking was harmful to the developing foetus. Moreover, the couples in the exposed group did not take any preventive steps to avoid exposure to ETS. These differences could be related to the socio-economic and educational factors that influence smoking habits. A study carried out among schoolteachers in India has shown that educational qualification had an effect on tobacco habits as fewer (20.0%) graduate teachers used tobacco in comparison to non-graduate teachers (55.7%). Though most of them (92.4%) were aware of the harmful effects of tobacco use, only 29.6% stated that it was a cause for concern. Awareness about the harmful effects of their smoking to others was seen only in 33.7% smokers.[31] In a study reported from Australia, it was shown that men whose partners were pregnant, were largely unaware that their own smoking could pose a specific risk to the foetus and were reluctant to quit smoking.[32] Interestingly, the observation in this study that a greater number of women exposed to ETS from their husbands also had a parent who smoked, has also been noted earlier in a large multi-country multi-centred study.[33]

In summary, exposure to environmental tobacco smoke during pregnancy is associated with more than two-fold higher risk of small-for-gestation baby even after adjusting for all possible confounders and there appears to be a dose response relationship between the quantum of smoke inhaled and the magnitude of weight reduction in the baby. The awareness about the harmful effects of passive smoking is poor. Evaluation of ETS exposure and steps to avoid it during pregnancy should be an important part of antenatal care.

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