

IMPACT OF ACTIVE AND PASSIVE SMOKING DURING PREGNANCY ON PLACENTAL COEFFICIENT- A COMPARATIVE STUDY

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ABSTRACT

BACKGROUND

Toxic effect of tobacco on developing foetus, newborn and placenta has been a subject of enormous interest for researchers in India and abroad. Smoking during pregnancy is estimated to account for 20% - 30% low birth weight.

Aims and Objectives- The present study intends to depict gross changes in placenta of smokers and passive smokers along with effect of active and passive smoking on placental coefficient. Also, we have tried to analyse impact of smoking on mode of delivery and pregnancy outcome.

MATERIALS AND METHODS

A hospital-based descriptive, comparative study was performed from 2008 to 2010 after seeking proper ethical approval from Institutional Review Board Committee. Pregnant females were divided in three Groups A, B and C of normal pregnant females, passive and active smokers according to the questionnaire respectively. Gross features of placenta and placental coefficient were calculated. ANOVA was used in SPSS software version 17 to compare the parametrical data and chi square for non-parametrical data.

RESULTS

In Group A, B and C foetal distress was 6.6%, 26.7% and 30% respectively. While 10%, 36.7% and 96.7% had LSCS in Group A, B and C respectively. Placental calcification was extensive in 3.3% and 6.7% of cases of Group B and C respectively. Meconium staining was observed in 3.3%, 13.3% and 20% of placenta of Group A, B and C respectively. Placental weight was 0.45 ± 0.052 gms in Group A, 0.506 ± 0.065 in B and 0.528 ± 0.063 in Group C. Birth weight was 2.8 ± 0.26 kgs in Group A, 2.18 ± 0.21 kgs in Group B and 2.1 ± 0.3 kgs in Group C. Placental coefficient was < 0.2 in Group A, while it was > 0.2 in Group B and C.

CONCLUSION

Smoking as well as passive smoking greatly affects placental coefficient as foetal hypoxia caused by smoke leads to compensatory hypertrophy of placenta and reduced foetal weight.

KEYWORDS

Placenta, Passive Smoking, Active Smoking, Placental Coefficient.

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BACKGROUND

Tobacco in any form is injurious to all systems of the body. Its toxic effect on developing foetus, newborn and placenta has been a subject of enormous interest for researchers in India and abroad. Tobacco and its smoke contain more than 4000 chemicals, of which around 50 are known carcinogens and 250 are highly toxic and poisonous.¹ The World Health Organisation has defined passively inhaled smoke as the smoke that individuals breathe when they are located in the same airspace as smokers.² Smoking during pregnancy is estimated to account for 20% - 30% low birth weight.¹ Smoking during pregnancy leads to retention of chemicals like tar and

nicotine. Whilst the chemicals like cadmium, arsenic, polonium, nicotine, polycyclic aromatic hydrocarbons are exhaled, which forms the main component of second-hand smoke which pose a serious health hazard for pregnant passive smokers.^{2,3,4}

Certain mechanisms have been proposed by which smoking leads to reduced birth weight; they are foetal hypoxia, vasoconstriction in placenta and direct toxic effect.^{2,5} Placental coefficient can be defined as ratio of placental weight and birth weight.²

The present study intends to depict gross changes in placenta of smokers and passive smokers along with effect of active and passive smoking on placental coefficient. Also we have tried to analyse impact of smoking on mode of delivery and pregnancy outcome.

MATERIALS AND METHODS

Study Design

Descriptive comparative study in Dept. of Obstetrics and Gynaecology of Zanana and Mahila Chikitsalaya of SMS MC, Jaipur was undertaken after seeking approval from Institutional Ethical Committee from year 2008 to 2010. The

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target population was the pregnant females at term coming with labour pain. Of those we screened, 90 pregnant females randomly who fulfilled our inclusion and exclusion criterion.

There were three Groups- A, B and C. Each group consisted of 30 pregnant females. Pregnant females between 36 to 40 weeks after taking consent were assessed via questionnaire for each group before labour.

Inclusion Criterion

Group A consisted of normal pregnant females not taking tobacco in any form, neither was exposed to second smoke. Group B consisted of passive smokers and Group C active smokers.

Group B consisted of pregnant females who were exposed to tobacco smoke for > 4 hrs./day from husband/close relative at home or at office consuming > 20 bidis/day or > 20 cigarettes in a day.

Group C consisted of pregnant females consuming > 10 bidis/10 cigarettes/day or taking zarda > 1 packet/day and keeping in mouth for > 30 minutes/day.

Exclusion Criterion

Women suffering from pre-eclampsia, diabetes mellitus, anaemia or any infective ailments were excluded. Women taking other forms of drug abuse with post-dated pregnancy or abortions, less than 18 yrs. or more than 35 yrs., greater than 4th parity were excluded.

Maternal and neonatal data such as mother’s body weight on admission, gestational age, parity, diseases, birth weight and Apgar score were obtained from the hospital records.

Questionnaires and Consent

A questionnaire was given to the mothers before labour. Questions included basic information such as smoking habits of mother and other household or office members, occupation, per capita income and medication. Written consent was obtained from each eligible pregnant woman who was willing to allow her placenta for research purposes. An approval from the Faculty Ethical Committee was obtained before the study was conducted.

Tissue Collection

The umbilical cord was clamped at the placental insertion immediately after delivery and cord cut approximately 5 mm from the insertion point before the membranes were trimmed away and blood clots removed. Gross features like calcification and meconium staining were noted and placentae were weighed on electronic weighing machine. ANOVA was used in SPSS version 17 to compare the parametrical data and chi square for non-parametrical data. Mode of delivery and pregnancy outcome were also noticed in three groups.

RESULTS

In Group A, B and C foetal distress was 6.6%, 26.7% and 30% respectively. While 10%, 36.7% and 96.7% had LSCS in Group A, B and C respectively. Placental calcification was extensive in 3.3% and 6.7% of cases of Group B and C respectively. Meconium staining was observed in 3.3%, 13.3% and 20% of placentae of Group A, B and C respectively. Placental weight was 0.45 ± 0.052 gms in Group A, 0.506 ± 0.065 in B and 0.528 ± 0.063 in Group C. Birth weight was 2.8

± 0.26 Kgs in Group A, 2.18 ± 0.21 Kgs in Group B and 2.1 ± 0.3 Kgs in Group C. Placental coefficient was < 0.2 in Group A, while it was > 0.2 in Group B and C.

	Group A (n=30)	Group B (n=30)	Group C (n=30)
FD	2 (6.70%)	8 (26.67%)	9 (30%)
LB	28 (93.30%)	22 (73.33%)	21 (70%)

Table 1. Outcome of Pregnancy

FD- Foetal distress, LB- Live Birth without foetal distress, NS- Not Significant, S- Significant.

Chi square value= 0.057, df= 2, P value= 0.03 (S).

	FTVD	LSCS	Assisted Vaginal Breech
Group A n=30	27 (90%)	3 (10.00%)	0 (0%)
Group B n=30	18 (60%)	8 (36.67%)	4 (1.30%)
Group C n=30	16 (53.30%)	10 (46.70%)	4 (1.30%)

Table 2. Mode of Delivery

FTVD- Full Term Vaginal Delivery, LSCS- Lower Segment Caesarean Section.

Chi square value= 0.025777, df= 4, P value= 0.0001 (Significant).

Placental Calcification				
	Nil	Patchy	Diffuse	Extensive
Group A n=30	28 (93.30%)	1 (3.30%)	1 (3.30%)	0.00%
Group B n=30	17 (56.70%)	2 (6.70%)	10 (33.30%)	1 (3.30%)
Group C n=30	14 (46.70%)	3 (10.00%)	11 (36.70%)	2 (6.70%)

Table 3. Placental Calcification

Chi square value= 16.798, df= 6, P value= 0.01 (Significant).

Meconium staining		
	Present	Absent
Group A (n=30)	1 (3.30%)	29 (96.70%)
Group B (n=30)	4 (13.30%)	26 (86.70%)
Group C (n=30)	6 (20.00%)	24 (80.00%)

Table 4. Meconium Staining

Chi square value= 3.96, df= 2, P value= 0.14 (NS)

	Group A n=30	Group B n=30	Group C n= 30	F value	P value
Placental Wt. (Gms)	0.48 ± .052	.506 ± .065	0.528 ± 0.063	64.56	0.01 (S)
Birth weight (Kgms)	2.8 ± 0.26	2.18 ± 0.21	2.1 ± 0.3	4.87	0.000(S)
Placental coefficient	0.17 ± 0.018	0.24 ± .046	0.255 ± 0.035	47.65	0.000(S)

Table 5. Placental Coefficient, Placental Weight and Birth Weight

S= Significant, Gms= grams, Kgms= Kilograms

DISCUSSION

In Group A, B and C foetal distress was 6.6%, 26.7% and 30% respectively. Herriot et al and Heron in 1962 also found foetal distress to be more common in smoking mothers.^{6,7} Placental calcification was extensive in 3.3% and 6.7% of cases of Group B and C respectively. Roberta E Christianson et al also noticed high prevalence of calcification in placentae amongst smokers.⁸ Tindall and Scott in 1965 graded calcification as mild, moderate and severe.⁹ Meconium staining was observed in 3.3%, 13.3% and 20% of placentae of Group A, B and C respectively. Placental weight was 0.45 ± 0.052 gms in Group A, 0.506 ± 0.065 in B and 0.528 ± 0.063 in Group C. Birth weight was 2.8 ± 0.26 Kgs in Group A, 2.18 ± 0.21 Kgs in Group B and 2.1 ± 0.3 Kgs in Group C. In 2001, Rath et al found increased incidence of low birth weight babies in passive smokers which was consistent with our study too.¹⁰

Placental coefficient was < 0.2 in Group A, while it was > 0.2 in Group B and C. A slightly lower placental weight among smokers was observed by O'lane,¹¹ Targett, Gunese, McBride and Beischer,¹² Kullander and Kallen,¹³ Wilson¹⁴ and Jarvinen and Osterlund,¹⁵ the amount of difference being 4, 8, 9, 11 and 13 g respectively. However, the difference is significant in only one of the studies.⁶ Mulcahy, Murphy and Martin¹⁶ observed a heavier placental weight among smokers. Spira et al also found a higher placental coefficient in heavy smokers.¹⁷ In 2004, Vogt linked increased incidence of low birth weight with maternal smoking.¹⁸

The ratio between placental weight and newborn weight (Placental coefficient) was higher in the passive and active smoking group. Similar observation was reported by Rocha et al in 1998 and Van der Salm et al, although this was observed in active smoking women. There may well be a state of chronic hypoxia in women who smoke.^{19,20} It is known that a high level of carboxyhaemoglobin is found in smokers and that it persists longer than was previously thought after each cigarette, resulting in chronic hypercarboxyhaemoglobinaemia, the influence of which on the foetus, and particularly on its birth weight has been proved.^{21,22}

Major limitation of the study was urine cotinine estimation was not evaluated in passive and active smokers, which are more accurate indicator of the exposure to tobacco smoke. Another limitation was the sample size was kept 30 for each group, i.e. 90 for convenience.

CONCLUSION

Passive smoking is equally harmful as is active smoking during pregnancy. There may well be a state of chronic hypoxia in women who smoke. A high level of carboxyhaemoglobin is found in active smokers and passive smokers and that it persists longer than was previously thought after each tobacco intake resulting in chronic hypercarboxyhaemoglobinaemia, which greatly influences placental coefficient.

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