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Original article

Ultrasound surveillance of fetal growth and wellbeing in maternal occupational tobacco handling – A cohort study

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Abstract

Introduction: Tobacco exposure during pregnancy has a negative influence on fetal growth. Maternal smoking causes placental vasoconstriction and fetal growth restriction (FGR). In India, the tobacco industry employs women to hand roll cigarettes called *bidis*. A *bidi* roller handles 120 g of tobacco and therefore 3 g of nicotine per day.

Aim: To evaluate the effect of occupational tobacco handling by pregnant women on fetal growth and wellbeing.

Methods: In this cohort study, 177 pregnant *bidi* rollers (exposed cohort) and 354 pregnant non-rollers (unexposed cohort) were followed up from 18-22 weeks of pregnancy until delivery with fetal sonogram and Doppler. Fetal surveillance data (including fetal biometry, Doppler indices, amniotic fluid index, biophysical profile and placentation) were recorded and compared. Maternal nicotine absorption was quantified by serum cotinine. Intergroup differences and relative risk (RR) were determined by Chi-square test (or Fisher exact when the count was ≤ 5), and adjusted odds ratio (OR) by binary logistic regression. Intrauterine growth trend was plotted.

Results: Fetal surveillance results were abnormal in 37.9% of the tobacco-exposed group, with a RR of 1.6 ($p = 0.001$) and adjusted OR

of 1.8 (p = 0.005) in comparison with the unexposed group. Nicotine absorption was evident in 28.4% of the *bidi* rollers that had abnormal fetal surveillance results. The mean estimated fetal weight and head circumference demonstrated a sustained deceleration, starting at 28-30 weeks of gestation, in the exposed group. Doppler data suggested fetal adaptation to maintain cerebral circulation. We found a higher frequency of oligohydramnios (statistically significant), placental abruption (not statistically significant), and placenta previa (not statistically significant) in the tobacco-exposed group.

Conclusions: Occupational tobacco handling in *bidi* rollers resulted in early-onset symmetric FGR and compromised wellbeing. Pregnant women with any form of tobacco exposure require vigilant fetal surveillance.

Keywords

Bidi rolling, occupational tobacco handling, Doppler, fetal growth restriction, nicotine, oligohydramnios.

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Introduction

Monitoring of fetal growth and fetal wellbeing are key components of antenatal care and are important for obstetric decision-making. Fetal growth is influenced by maternal, fetal, and placental factors. The American College of Obstetricians and Gynecologists defines fetal growth restriction (FGR) as an estimated fetal weight (EFW) < 10^{th} centile for the gestational week (GW) determined by fetal biometry [1]. Serial sonograms and Doppler indices are necessary to differentiate between constitutional and pathological smallness of a fetus [2]. Doppler velocimetry of fetal umbilical artery (UA) and middle cerebral artery (MCA) identify uteroplacental insufficiency. Amniotic fluid index (AFI) and fetal biophysical profile (BPP) are other adjunct ultrasound determinants of fetal wellbeing [3].

About 5-10% of all pregnancies are complicated by FGR. Among maternal factors that warrant fetal surveillance, tobacco exposure is one. Maternal smoking causes placental vasoconstriction and FGR [4, 5]. About 20-30% of all FGR is early-onset before 32 weeks of gestation [2, 6]. The early-onset FGR differs in etiology, the severity of placental dysfunction, fetal deterioration, and outcome in comparison to late-onset and carries a poor prognosis. There is an absolute decrease in fetal size, resulting in symmetric FGR. Early pathologic FGR with compromised fetal wellbeing is demonstrated in maternal smoking and environmental tobacco smoke (ETS) exposure [7-9]. Adverse fetal effects are also reported with smokeless tobacco exposure, such as snuff use in Sweden [10] and tobacco chewing in India [11].

India is the world's third largest tobacco producer. The popular Indian cigarettes known as *bidis* consist of shredded sun-dried tobacco dust hand rolled in a dried leaf. More than 4 million women are engaged in *bidi* rolling, and each roller handles around 120 g of tobacco and therefore 3 g of nicotine every day [12]. In a prospective cohort study on pregnancy outcome in *bidi* rollers, we established that the adjusted risk for FGR was 2.7 times higher in the tobacco-exposed compared with the controls [13]. The adjusted mean differences in newborn birth weight, length, and head circumference (HC) were lower among the newborns born to the tobacco-exposed maternal cohort. We also established that cotinine, an active nicotine component, gets absorbed into maternal and fetal circulation in *bidi* rollers.

In this paper, we present the ultrasonographic and Doppler data related to fetal growth and wellbeing of the same cohort of women exposed to tobacco through occupational handling in comparison with the tobacco-unexposed.

Methods

In this study, we present the ultrasonographic and Doppler data of a cohort consisting of 177 pregnant women exposed to tobacco through *bidi* rolling and 354 pregnant women with no tobacco exposure of any form. The women were followed

up from 18-22 GW until delivery. Women who engaged in *bidi* rolling for at least a year prior to pregnancy and with no other tobacco exposure constituted the exposed group, and women with no tobacco exposure constituted the unexposed group. Thus, smokers, tobacco chewers, snuff users, and those with ETS exposure were excluded from both groups. Other exclusion criteria were psychoactive drug use, multiple gestations, chronic hypertension, overt diabetes mellitus, autoimmune disease, or any preexisting condition. Parity and socioeconomic strata were matched between the groups. Further details of the participant selection, exposures, and sample size calculation have been published previously [13]. The body mass index and pregnancy weight gain were not statistically significant between the groups and hence were not confounding factors [13]. Nicotine absorption was established by surrogate measurement of serum cotinine by ELISA, and a value ≥ 2 ng/mL was considered significant. Reapproval was obtained from the Institutional Ethics Committee to analyze and present the collected anonymized data.

Sonogram and Doppler data

All women in the cohort had undergone at least one growth scan in the third trimester of pregnancy. Serial sonogram data were available in two-thirds. Fetal Doppler studies were available in 48.0% (n = 85) of the exposed cohort and 40.1% (n = 142) of the unexposed cohort. Ultrasound examination was performed by Philips® HD7 XE using 2-5MHz convex probe and Doppler fetal UA and MCA with the convex transducer in colour and pulsed wave modes. Along with simultaneous real-time B-mode, spectral tracings were obtained with a sample volume of 4 mm, pulse repetition frequency of 2.5 MHz low filter settings with the angle of intonation between 0-60 degrees.

Spectral waveforms obtained over three consecutive cardiac cycles and systolic diastolic ratio (S/D), resistive index (RI), and pulsatility index (PI) were measured in automatic mode. Measurements were repeated till two similar values were obtained. For UA, a free loop was selected after tracing the cord to either end on greyscale and artery localized on colour Doppler mode. For MCA, the M1 segment distal to the bifurcation of the intracranial internal carotid artery was localized, and the tracings were obtained avoiding probe pressure on fetal cranium. AFI was calculated by the four-quadrant method.

Fetal growth and fetal wellbeing

Fetal biometry parameters included HC, abdominal circumference (AC), femur length (FL), FL-AC and HC-AC ratios. The EFW and the growth centile for GW derived by the software were documented. In a growth-restricted fetus, constitutional and pathological smallness were distinguished by the absence and presence of further growth deceleration in serial sonograms.

A compromise in fetal wellbeing was documented as corroborative evidence of pathologic smallness. A UA S/D > 3 and RI > 0.6 with absence or reversal of diastolic flow, waveform notches, UA PI > 1, MCA S/D < UA S/D and MCA PI / UA PI < 1 (cerebroplacental ratio) suggested uteroplacental insufficiency. An AFI of ≤ 5 cm suggested oligohydramnios, and the BPP with nonstress test scores less than 6/10 or 8/10 with oligohydramnios were considered abnormal.

Study outcomes

The study's primary outcome was to evaluate the effect of occupational tobacco handling on fetal growth and wellbeing. The secondary outcome was to assess the frequency of abnormal placentation and fetal congenital anomalies in the tobacco-exposed.

Statistical analysis

Statistical analysis was done using SPSS® (Statistical Package for Social Sciences; IBM® Corporation, New York, USA) version 24.0 and EpiInfo™ (Centre for Disease Control, USA) version 6. The inter-group differences of various fetal outcome variables calculated by Chi-square test (or Fisher exact when the count was ≤ 5) was expressed as relative risk (RR) with 95% confidence interval (CI). For statistically significant outcomes, we used binary logistic regression to determine the adjusted odds ratio (OR) for tobacco handling. Scatter plots were constructed for graphical projection of fetal growth with GW on the x-axis and the mean EFW and HC on the y-axis. Interpolation lines and linear fit with one standard deviation (SD) were generated.

Results

Tab. 1 gives the sonographic findings in the tobacco-exposed and tobacco-unexposed cohorts of pregnant women.

Four (2.3%) pregnancy losses were noted in the tobacco-exposed group, while there was none in the unexposed group.

Overall, fetal surveillance studies were abnormal in 37.9% of the tobacco-exposed group and 23.7% of the tobacco-unexposed group, with statistically significant RR of 1.6 (p = 0.001). An abnormal fetal Doppler was observed in a higher proportion of the tobacco-exposed group (11.8%) than unexposed group (4.2%), with RR of 2.8 (p = 0.032)

Pathologic FGR was seen in 13.9% and 5.4% of the tobacco-exposed and unexposed groups, respectively, with RR of 2.6 (p < 0.001). In the tobacco-exposed group, abnormal Doppler findings included higher occurrence of absent diastolic or reversal of flow in the UA and altered UA PI. This suggested compromised placental perfusion and adaptive changes in the fetus to maintain cerebral circulation. Oligohydramnios was seen mainly in the tobacco-exposed group (2.9%) than the unexposed group (< 1%). About 7% of the fetuses in the tobacco-exposed group showed abnormal BPP compared to 2.2% in the unexposed group, with RR of 3.1 (p = 0.008), suggesting fetal distress. We also found a higher frequency (not statistically significant) of placental abruption and placenta previa in the tobacco-exposed group. Urinary system anomaly, the only major fetal anomaly in either group, was comparable.

The fetal well-being was compromised significantly (p = 0.005) in the tobacco-exposed group, even after accounting for maternal age, parity, and pregnancy-related illnesses, with adjusted OR of 1.8 (**Tab. 2**). Similarly, tobacco exposure by itself accounted for significant (p = 0.002) pathologic FGR, with adjusted OR of 2.9. Oligohydramnios, abnormal fetal Doppler, and abnormal BPP, which were seen in higher proportion in the tobacco-exposed group, showed no statistical significance when adjusted for maternal age, parity, and pregnancy-related illnesses.

Nicotine absorption was evident in 28.4% (n = 19) of *bidi* rollers with abnormal sonogram and Doppler. The maximum maternal serum cotinine observed was 90 ng/mL. Seven of the 24 women with FGR had elevated serum cotinine.

The scatter plots of mean EFW (**Fig. 1A** and **Fig. 1B**) showed a deceleration in the intrauterine growth, starting at 28-30 weeks of gestation, in the

Table 1. Fetal ultrasonogram and Doppler in the tobacco-exposed and unexposed groups.

Parameter		Exposed (n_1 [a])	Unexposed (n_2 [a])	RR (95% CI)	Significance [b]
Overall abnormal fetal surveillance results (n_1 = 177; n_2 = 354)		67 (37.9%)	84 (23.7%)	1.6 (1.2, 2.1)	**0.001**
Fetal growth	**Fetal biometry < 10th centile**	54 (31.2%)	73 (17.6%)	1.5 (1.1, 2.0)	**0.007**
	Constitutionally small	30 (17.3%)	54 (15.2%)	1.1 (0.8, 1.7)	0.539
	Pathologic FGR	24 (13.9%)	19 (5.4%)	2.6 (1.5, 4.6)	**< 0.001**
Fetal wellbeing	**Abnormal fetal Doppler (n_1 = 85; n_2 = 142)**	10 (11.8%)	6 (4.2%)	2.8 (1.0, 7.4)	**0.032**
	UA diastolic flow absence or reversal	8 (9.4%)	6 (4.2%)	-	-
	UA waveform notches	5 (5.9%)	6 (4.2%)	-	-
	UA abnormal PI	8 (9.4%)	6 (4.2%)	-	-
	Abnormal cerebroplacental ratio	4 (4.7%)	4 (2.8%)	-	-
	Oligohydramnios	5 (2.9%)	2 (0.6%)	5.1 (1.0, 25.9)	**0.042**
	Abnormal BPP	12 (7.0%)	8 (2.2%)	3.1 (1.3, 7.4)	**0.008**
Others	**Absent cardiac activity (n_1 = 177; n_2 = 354)**	4 (2.3%)	0 (0%)	Undefined	**0.012**
	Placental abruption	1 (0.6%)	0 (0%)	Undefined	0.328
	Placenta previa	2 (1.1%)	0 (0%)	Undefined	0.107
	Fetal anomaly	2 (1.1%)	1 (0.3%)	4.1 (0.4, 44.8)	0.252

Data are presented as n (%).
[a] n_1 = 173, n_2 = 354, unless otherwise stated; [b] Fisher exact test for value < 5.
BPP: biophysical profile; CI: confidence interval; FGR: fetal growth restriction; PI: pulsatility index; RR: relative risk; UA: umbilical artery.

Table 2. Adjusted odds ratio (OR) for adverse fetal outcome in the tobacco-exposed cohort.

Parameter	Adjusted OR[a]	95% CI	Significance
Overall abnormal fetal surveillance results	1.8	1.2, 2.7	**0.005**
Pathologic FGR	2.9	1.5, 5.6	**0.002**
Abnormal fetal Doppler	1.7	0.5, 5.8	0.379
Oligohydramnios	5.2	0.9, 30.0	0.062
Abnormal BPP	2.2	0.7, 6.5	0.146

[a] Adjusted for maternal age, parity, anemia, hypertension, diabetes mellitus, and tobacco handling.
BPP: biophysical profile; CI: confidence interval; FGR: fetal growth restriction; OR: odds ratio.

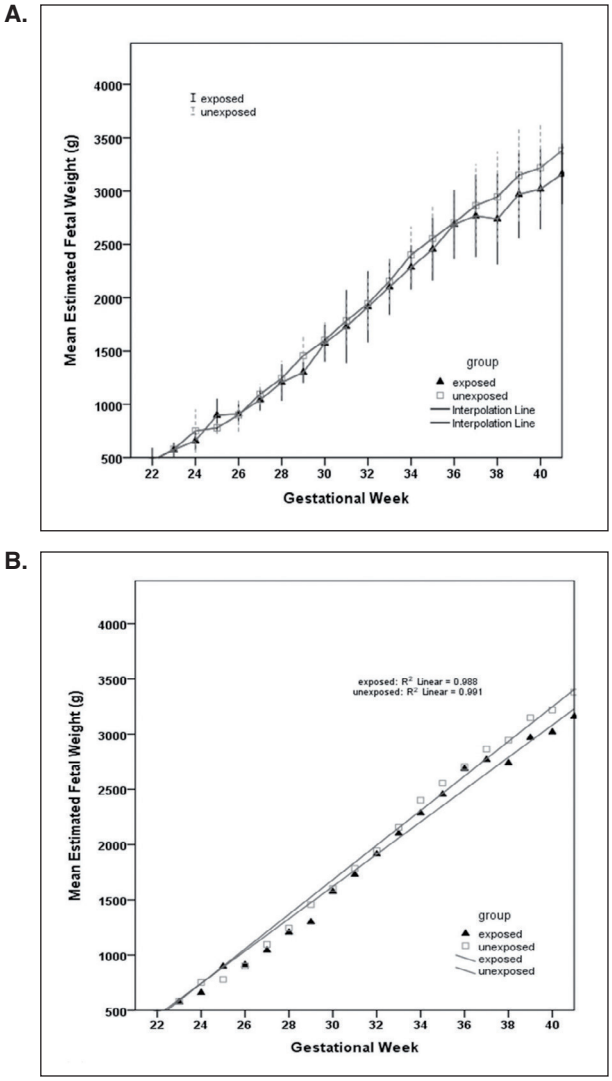

Figure 1. Graphical presentation of the mean estimated fetal weight (EFW) for the gestational week (GW) among the tobacco-exposed and tobacco-unexposed groups. **A.** Interpolation lines. **B.** Linear fit.

tobacco-exposed cohort. This deceleration in fetal growth was sustained throughout the later weeks of gestation, with widening of the gap in EFW towards maturity.

The fetal HC (**Fig. 2A** and **Fig. 2B**) also showed deceleration, paralleling the observations in EFW, suggesting symmetric growth restriction.

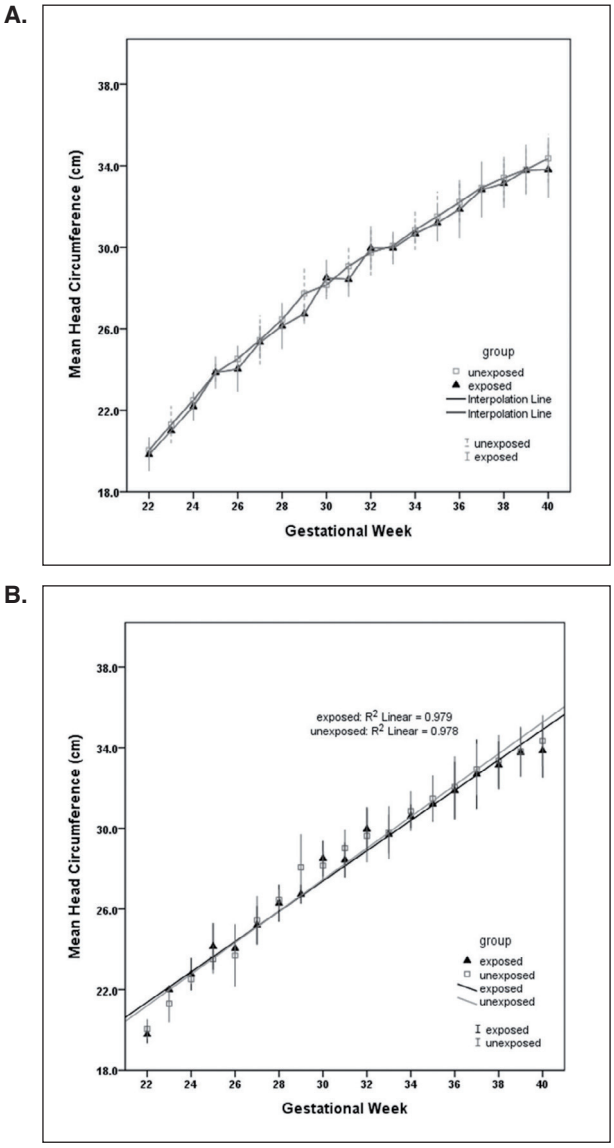

Figure 2. Graphical presentation of the mean head circumference (HC) for the gestational week (GW) among the tobacco-exposed and tobacco-unexposed groups. **A.** Interpolation lines. **B.** Linear fit.

Discussion

In the present study, we analyzed the ultrasound and Doppler data of tobacco-exposed and tobacco-unexposed pregnant women who were followed up from 18-22 weeks of gestation until delivery. We

found early and symmetric FGR with compromised fetal wellbeing. Nicotine absorption was evident in one-third of the *bidi* rollers that had abnormal fetal surveillance results.

The adjusted OR of 1.8 for abnormal fetal surveillance results in our study was comparable to that seen in other forms of maternal tobacco exposure. Dejmek et al. [5] report an adjusted OR of 1.6 (95% CI: 1.1, 2.5) for FGR in maternal smoking. Exposure to ETS was additive for FGR, with an adjusted OR of 2.1 (95% CI: 1.7, 2.7). We noted increased resistances in UA and MCA flow patterns in the tobacco-exposed group, comparable to the results obtained by Albuquerque et al. [14] and Yildiz et al. [15] among pregnant smokers. However, in the Alptekin et al. [16] study, the fetal MCA indices in the smokers were comparable with non-smokers. In our study, the adjusted OR for FGR with compromised fetal wellbeing among tobacco-exposed was 2.9 and statistically significant. In the only other ultrasound study in pregnant *bidi* rollers, Jadhavar et al. [17] found altered UA indices with absence or reversal of diastolic flow. The authors also showed significant risk for oligohydramnios in the *bidi* rollers. Sardesai et al. [18] found increased basement membrane thickening, villous fibrosis, and calcification in placental histopathology examination in pregnant *bidi* rollers.

Jaddoe et al. [7] demonstrated progressive decrease in fetal HC (-0.56 mm/week [95% CI: -0.73, -0.40]) in smokers from 25 weeks of gestation. Hanke et al. [8] found a negative correlation between fetal biometry measurements at 20-24 weeks and maternal serum cotinine levels in active and passive tobacco exposures. The adjusted bi-parietal diameter decreased by 0.49 mm/week per unit logarithmic change in serum cotinine. We observed early and sustained deceleration of EFW and fetal HC in *bidi* rollers, but could demonstrate nicotine absorption in only one-third of the *bidi* rollers that had abnormal fetal surveillance results. A meta-analysis on fetal biometry in smokers showed significant symmetric reduction in EFW (-0.18 SD score [95% CI: -0.24, -0.11]) and HC (-0.09 SD score [95% CI: -0.16, -0.01]) by the second trimester [19]. The fetal HC deceleration was progressive (-0.18 SD score [95% CI: -0.23, -0.13]) by the third trimester. Fetal nicotine exposure causes decreased brain growth and altered brain structure through epigenetic changes and microRNA expression dysregulation [20].

Studies also show a significant association between smoking and placental abruption [21], placenta previa [22], and stillbirth [23]. The adjusted risk for stillbirth is reported at 1.4 (95% CI: 1.0, 2.0) among Swedish snuff users [10] and 2.6 (95% CI: 1.4, 4.8) in the Indian tobacco chewers [11]. Maternal smoking before or during pregnancy significantly increases birth defects like cleft lip palate and hypospadias by 1.3 times [24]. However, a United Kingdom database study showed an adjusted OR of 1.82 (95% CI: 0.85, 3.89) for urinary system anomalies (p = 0.04) in nicotine patch users [25].

Most of the studies utilized questionnaires to determine and quantify tobacco exposure. Hanke et al. [8] used serum cotinine assay to categorize exposure. In the present study, we found evidence of nicotine absorption in one-third of the *bidi* rollers with abnormal fetal surveillance results. Majority of the women stopped tobacco handling early in the third trimester [13]. Also, serum cotinine levels are influenced by pregnancy and ethnicity.

The present study was done in a tertiary care teaching hospital with the assurance of accurate clinical and fetal surveillance data in this cohort study. However, the sample size was inadequate for establishing abnormal placentation and fetal congenital anomalies. Abnormal fetal surveillance results could not be correlated with placental histopathology, as it was done only in a few.

Conclusion

Occupational tobacco exposure due to *bidi* rolling leads to early-onset symmetric FGR with compromised fetal wellbeing. The Doppler data suggest increased vascular resistance in the maternal-placental-fetal unit and fetal adaptive findings to maintain cerebral circulation. Pregnant women with tobacco exposure require vigilant fetal surveillance. Take-home messages are presented in **Tab. 3**.

Table 3. Take-home messages.

What was already known?	Tobacco exposure during pregnancy through smoking or ETS causes FGR.
What does this study add?	Occupational tobacco handling during pregnancy causes early-onset symmetric FGR and compromised fetal wellbeing.
	Pregnant women handling tobacco require vigilant fetal surveillance.

ETS: environmental tobacco smoke; FGR: fetal growth restriction.

Data availability statement

The raw data supporting the study results are available on reasonable request from the corresponding author.

Ethics statement

This study involving human participants was in accordance with the 1964 Helsinki Declaration and comparable ethical standards. The original study had the approval of the Institutional Ethics Committee; reapproval dated 27/01/2020 was obtained for the presentation of sonographic data. Anonymized data is presented.

Declaration of interest

The Authors declare that there is no conflict of interest.

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