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Short Communication

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Association of Concomitant Drinking and Smoking during Pregnancy with Placental Abruption, Fetal Growth Restriction and Sudden Infant Death Syndrome (SIDS)

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Placental abruption is the partial or complete separation of the placenta before the birth of the baby. This severe complication is seen in 0.4%-1% of pregnancies and is responsible for 10% of all preterm births and 10%-20% of all perinatal deaths [1]. For the USA and Japan the prevalence rate seems to vary around 0.9%,[2,3] but a rate of 4.4% has been reported in India [4]. Placental abruption and placenta praevia are attributed causes of stillbirth in 7.5 to 42% of 142 studies in low to middle-income countries included in a systematic review [5]. In high-income countries, placental abruption and growth restriction carry the highest population-attributable risks of pregnancy disorders for stillbirths (15% and 23% respectively) [6].

Fetal growth restriction (FGR) develops when the fetus does not reach its intrauterine growth potential, mainly because of placental insufficiency [7]. It affects 5%-10% of pregnancies and is one of the most common causes of perinatal mortality. Doppler velocimetry plays a leading role in the diagnosis of fetal growth restriction, as abnormal values, reflected by a high pulsatility index, indicate impedance to blood flow in the uterine or umbilical arteries with subsequent poor perfusion [8,9].

The association between cigarette smoking, placental abruption and pregnancy associated syndromes, such as fetal growth restriction, is well known [10]. Pregnant women who drink alcohol had a 33% greater likelihood of placental abruption, and also increased risks of placenta praevia, FGR and preterm or stillbirths [11]. In addition, there also seems to be a compound effect of cigarette smoking and alcohol consumption on low birth weight [12].

One of the first prospective studies on the effects of drinking and smoking in pregnancy was the Safe Passage Study (SPS), which was designed to investigate the effects of alcohol exposure during pregnancy on stillbirths and SIDS [13]. The SPS found that the increase in the relative risk for SIDS was 11.79 (98.3% CI: 2.59-53.7, p<0.001) in infants whose mothers reported both prenatal drinking and smoking beyond the first trimester, 3.95 (98.3% CI: 0.44-35.83, p=0.14), for drinking only beyond the first trimester and 4.86 (95% CI: 0.97-24.27, p=0.02) for smoking only beyond the first trimester as compared to those unexposed or who reported quitting early in pregnancy. Dual exposure therefore carried about a three times higher risk than single exposure [14].

The SPS reported 145 stillbirths among the outcomes of 11 892 pregnancies. Compared with a non-smoking and non-drinking reference group, after adjustment for different variables that could have an effect on stillbirths, the relative risk for late stillbirth (at 28 weeks' gestation or later) was 1.60 (98.3% confidence interval 0.64-3.98; p=0.2221) for smoking only and 2.22 (98.3% CI 0.78-6.18; p=0.0632) for drinking only and increased to 2.78 (98.3% CI 1.12-6.67) for dual exposure [15].

In an analysis of the Cape Town data from the SPS, the prevalence rate of placental abruption in 5806 women was 0.87% [16]. The rate of placental abruption in smokers was 1.15%, significantly higher (p<0.005) than the rate of 0.52% in nonsmokers. The use of alcohol did not seem to affect the rate. However, when concomitant smoking and drinking was examined, the prevalence rate of placental abruption was 0.11% in women who did not drink or smoke in contrast to 1.25% in those who drank and smoked during pregnancy (p<0.005).

Doppler flow velocity waveforms of the uterine and umbilical arteries were done in around 1319 women of this cohort. When compared to nondrinkers/nonsmokers, the Doppler pulsatility index of the uterine artery at 20-24 weeks gestation in heavy drinkers/smokers was significantly higher (<0.05). Drinking or smoking on its own did not increase the pulsatility index. The fact that an increase in the pulsatility index was limited to heavy concomitant drinkers and smokers, illustrated the synergistic effects of alcohol and nicotine on increasing resistance in the uterine artery [15].

Examination of the effects of smoking and drinking on the umbilical artery at 20-24 weeks gestation showed almost similar effects, but the increase in the pulsatility index was seen in women who drank and smoked moderately or heavily. The fact that no increase in the pulsatility index was seen in the abstainers or women who either drank or smoked, in contrast to those who drank and smoked, illustrates the synergistic effects of the two substances on the pulsatility index of the umbilical artery [16].

Z-scores of weight for gestational age, where a low score indicated FGR, were significantly lower in women who concomitantly drank and smoked, when compared to abstainers or women who either drank or smoked. This finding again illustrated the synergistic effects of the two substances [16].

Very few retrospective studies addressed the combined effects of alcohol and tobacco use on FGR. In a large retrospective cohort, the effect of health compromising behaviors on term-gestation birthweight was examined in 78,397 live births. After adjustment for covariates,

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smoking alone (OR 2.3 (CI: 2.0-2.5)) and smoking and alcohol (OR 4.4 (CI:3.4-5.7)) remained the major risk factors for term low birth weight. The addition of alcohol to smoking almost doubled the risk [17].

Although SPS has demonstrated that concomitant drinking and smoking during pregnancy was associated with sudden infant death syndrome, placental abruption and FGR, the aggravating effects of poor socioeconomic conditions on the outcome of pregnancy should always kept in mind [18].

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