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Perspective

Alcohol and It's Side Effects during Pregnancy

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ABOUT THE STUDY

The most prevalent definition of Preeclampsia (PE), a subtype of Hypertensive Disorders of Pregnancy (HDP), is new onset hypertension after 20 weeks of gestation with new-onset proteinuria or end-organ damage. 14% of maternal fatalities globally are caused by PE and other hypertensive conditions. Preterm birth and perinatal death are both mostly caused by PE. Pregnancies with PE at 34 weeks had a seven-fold increased stillbirth risk than those without PE. Within the first year of delivery in the United States, the cost burden of PE was $2.18 billion. The cause of PE is yet unknown, though. PE was linked to some maternal risk factors, including obesity, diabetes mellitus, and renal illness, which were also linked to women's risk for cardiovascular disease. Even at lower levels of use, drinking alcohol was linked to a higher risk of cardiovascular disease development [1].

Furthermore, a strong causal link between alcohol use and hypertension in the non-pregnant population was established by a few intervention studies and Mendelian randomization experiments. Heavy to moderate drinking is more likely to develop hypertension. On the subject of the link between prenatal alcohol consumption and PE or HDP, there isn't a clear consensus. Most professional guidelines advise abstinence during pregnancy since alcohol intake during pregnancy can result in congenital and neurodevelopmental problems (such fetal alcohol spectrum disorders). Even so, up to 10% of pregnant women in Canada and 15% of pregnant women in the US consume alcohol. Alcohol consumption during pregnancy is also linked to the development of disorders including PE and HDP in the mother, in addition to its effects on the foetus [2].

The included studies showed significant heterogeneity. Only a small number of studies have been done on the relationship between drinking alcohol while pregnant and the risk of PE or HDP, according to our review. The majority of included studies were of poor quality, and many of them did not account for potential confounders. Despite the fact that we used a meta-analysis to calculate the overall risk, the substantial level of heterogeneity implies that these findings should be regarded with caution. The primary cause of PE has been identified as suboptimal placental function. Alcohol can cause placental trophoblast cells to go into apoptosis, according to *in vitro* research. Animal studies revealed that ethanol exposure during pregnancy impaired placental trophoblastic cell invasion and differentiation as well as placental implantation depth [3].

Exposure to alcohol during pregnancy was also shown to raise blood pressure in pregnant rats. Because observational studies are prone to bias and because no analysis of the dose-response relationship or adjustment for potential confounding were carried out in the majority of the included epidemiologic research [4]. The main causes of the existing discrepancies in conclusions about the connection between alcohol and PE or HDP may be unmeasured confounders and limits in the proper evaluation of drinking dose, frequency, method, time, and patterns. Therefore, in future research, a dose-response analysis could be useful to determine whether pregnant women also exhibit the J-shaped connection reported in non-pregnant women, which has a protective impact at low doses and a deleterious effect at high doses. Mendelian randomization, family-based designs, and natural experiments are new analytical techniques that can enhance causal inference and get beyond confounders like socioeconomic issues [5].

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