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Fetal Alcohol Syndrome (FAS)

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Abstract

Fetal alcohol syndrome (FAS) is a complex, multifactorial, and intriguing disorder. The consequences that can ensue from alcohol consumption, besides amount, dose, and stage of alcohol exposure, only 5% of alcohol drinking women have a newborn with FAS. Although the incidence of alcohol abuse is increasing in the western world, the nutritional and physical status of this woman may have significant impact on the FAS occurrence.

Introduction

Fetal alcohol syndrome (FAS) is caused by a pregnant mother drinking alcohol and exposing her fetus to the substance. FAS is a birth defect that primarily affects the brain. People with FAS are born with the disorder and will not outgrow its affects, another more recent term for FAS is fetal alcohol spectrum disorders (FASD). FASD is describing the range of effects that can occur in an individual whose mother drank alcohol during pregnancy, FAS is the most identifiable and most serious disorder under the FASD. Alcohol is a teratogen a toxic substance that can inhibit healthy development of the fetus. When alcohol is consumed during pregnancy it can easily cross from the mother to the baby via the placenta, exposing the developing baby to blood alcohol levels similar to those in the mother. FAS is serious global problem due to increased prevalence of alcohol abuse among young women during pregnancy because, the most common and leading preventable cause of mental retardation. Prenatal alcohol exposure (PAE), induces deleterious effect on physical, neurological, and behavioral development of the child, and important to prevent FAS at an early stage to avoid its deleterious consequences in adult life. Risk factors include race, low socioeconomic status, binge drinking during pregnancy, emotional stress, and use of tobacco and other drugs along with alcohol. the fetus with these syndrome have characteristic feature for example physical appearance cleft Lip, droopy eyelids, short, upturned nose ,thin upper lip, hearing disorders the severity of the hearing disorder will vary from child to child ,Eye disorders, other features like Low birth weight, small head of age (microcephaly), large or malformed ears, musculoskeletal abnormalities, and Immune disorders⁽¹⁾. Alcohol is metabolized within hepatocytes by 1 of the 3 following pathways: Alcohol dehydrogenase pathway (ADH): The first

pathway, known as ADH, occurs in the cytosol of the hepatocyte. ADH metabolizes ethanol to acetaldehyde, which is subsequently converted into acetic acid in mitochondria. In the ADH pathway, ethanol competes with vitamin A, or retinol, for metabolism because both substrates are metabolized by the same pathway. Ultimately, ethanol is oxidized, which leads to the production of acetaldehyde and large amounts of NADH. Microsomal ethanol oxidizing system (MEOS): The second pathway, MEOS, occurs in the endoplasmic reticulum. The MEOS pathway activates cytochrome P450 activity, specifically cytochrome P450 2E1 (CYP2E1), which metabolizes and activates substrates to produce toxic by-products (Das and Vasudevan, 2007). Chronic alcohol exposure causes increased cellular production of CYP2E1, therefore leading to an increased tolerance to ethanol. This pathway inhibits scavenger enzymes, such as glutathione, and causes the production of reactive oxygen species, such as superoxide (O_2^-) and hydrogen peroxide (H_2O_2), which results in lipid peroxidation. Therefore, the MEOS pathway has beneficial effects in individuals who consume alcohol acutely, yet also produces harmful effects because chronic alcohol exposure leads to increased enzyme activity, ethanol tolerance, and cellular oxidative stress. Catalase: The third pathway is through the enzyme catalase, which is located in the peroxisomes. In this mechanism, ethanol and H_2O_2 are converted to acetaldehyde and water. Oxidation of ethanol by catalase is more common in individuals who consume large amounts of alcohol. These individuals tend to accumulate higher amounts of FAs in the liver, causing hepatic steatosis, which may progress to hepatitis and cirrhosis. With the alcohol-induced fat accumulation, there is increased peroxisomal oxidation of FAs, and this may provide an explanation for the increased catalase activity⁽²⁾.

Aims of the Study

FAS is the most common and leading preventable cause of mental retardation. The present study provides information to identify in newborns of mothers who consume alcohol the presence of FAS, other congenital defects, and/or neurodevelopment disorders related to the maternal ingestion of alcohol.

Materials and Methods

In a public maternity in the city of São Paulo, 1,964 puerperal women were interviewed and 654 had consumed alcohol at some point during gestation. The

newborns were clinically and laboratorially examined in order to identify the occurrence of fetal alcohol syndrome, congenital defects or neurodevelopment disorders related to alcohol.

Results

Three children were found with fetal alcohol syndrome, 6 with congenital defects related to alcohol, and 67 with developmental disorders related to alcohol. The congenital malformations found in these children were thin or absent corpus callosum, brain⁽³⁾.

Discussion

Alcohol consumption by pregnant women is without a doubt a serious worldwide Public Health concern, since it damages the fetus not only physically, but also in terms of behavior. Despite the known adversity of prenatal exposure to alcohol, the children that suffer the most are often not identified since the diagnosis of a newborn with FASD is difficult in most cases. Clinical findings of FASD may go unnoticed, since they result from the combination of various factors that act at different critical periods of fetal development. Thus, calling attention to the different aspects of FASD will contribute towards its early identification. The specific pathophysiology of alcohol in fetus is still unknown, but there is probably no one single mechanism that can explain all the damaging effects on the unborn child. There are no markers capable of determining the specific action of alcohol on the fetus, nor the precise influence of the dose on the mechanism of developing of the syndrome. It is a known fact that alcohol passes through the fetal barrier between the blood and the brain, and its effects on cerebral development are extremely complex. In certain groups of cerebral cells it can lead to death. It is a well-known fact that exposure to alcohol at any time during pregnancy may cause effects on the CNS, which will be more damaging if they occur in the first five weeks of gestation. One evident result is the decrease in cerebral growth, manifested by microcephalus and microencephaly, but occasionally, prenatal exposure to alcohol can cause more specific brain lesions. Habitually, however, brain damage is generalized and non-specific, with an increase in the appearance of functional abnormalities throughout the child's development,

which may present structural alteration of the CNS with FAS, with no detectable functional deficit⁽⁴⁾.

Conclusion

Fetal alcohol syndrome (FAS) results from maternal alcohol use during pregnancy and carries lifelong consequences. Newborns of mothers who consumed alcohol may have congenital malformations of various organs and systems, and early diagnosis is fundamental for a probable and occasional more effective resolution and progress.

References

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