FEATAL ALCOHOL SYNDROME

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ABSTRACT

Fetal alcohol syndrome is a permanent birth defect syndrome caused by maternal consumption of alcohol during pregnancy. The prevalence is seen more in U.S, Atlanta, Alaska, and North Dakota. It causes the alcohol related birth defects. Overall, the available literature points to a prevalence rate of FAS of 0.5 to 2 babies per 1000 birth in U.S during 1980’s and 1990’s. There were 63 children less than ten years age with FAS under pediatric care in 1993. The above observation strongly predetermines the prevention efforts for fetal alcohol syndrome. Prevention include the selective, indicated preventions and universal preventions diagnosis done for without confirmed maternal exposure Partial FAS with confirmed maternal alcohol exposure. A daily dose of disulfiram can be given daily by orally and involvement in Special Education and Social Services most kids with FASD do with a combination of Stimulant + Selective Serotonin Receptive Inhibitor (SSRI).

Keywords: Flattened Philitrum, Microcephaly, CNP abnormalities and brain abnormalities.

1. INTRODUCTION

Alcohol ingested during pregnancy can have a range of deleterious consequences for the development of the fetus. The most server conditions caused by parental alcohol exposure is FAS which is characterized by a particular pattern of facial anomalies, growth retardation and developmental abnormalities in central nervous system. The term fetal alcohol syndrome was introduced in 1973 by Jones and Smith (1973), whose original diagnostic criteria have changed very little even after being reconsidered by other groups, such as the Fetal Alcohol Study Group of the Research Society on Alcoholism (Rosett 1980; Sokol and Clarren 1989). However, after the FAS diagnostic criteria were introduced, it became clear that there were people who likely had been adversely affected by prenatal alcohol exposure but who did not completely fulfill the criteria for a diagnosis of FAS. One term that had been introduced to include such cases was fetal alcohol effects (FAE) (Clarren and Smith 1978). But, unlike the term FAS, not all clinicians and researchers used the term FAE uniformly. Consequently, the IOM addressed this confusion by introducing more refined definitions, which have helped to provide consistency in the terminology used to describe the problems caused by prenatal alcohol exposure. For this reason, it is worthwhile to review the diagnostic criteria in the IOM report in some detail.

2. SIGNS AND SYMPTOMS

Evidence for characteristic patterns facial anomalies. Short palpebral fissures and abnormalities in the premaxillary zone. (eg; flat upper lip, flattened philitrum, and flat midface)

Fig.1: Baby effected with featal alcohol syndrome
Evidence for growth retardation. Brain birth of baby without and with alcohol exposure. Low birth weight for gestational age. Decelerating weight over time not due to other in defined causes. Disproportional low weight to heights. Evidence to CNS abnormalities. cranial size at birth, structural brain abnormalities. (eg: microcephaly, cerebella hypoplasia). Neurological hard(or)soft signs(as age appropriate), such as impaired fine motor skills, neurosensory, hearing loss, poor tandem gait, poor hand eye coordination.

3. PREVALENCE

<table>
<thead>
<tr>
<th>Type and Location of Study</th>
<th>Years Covered</th>
<th>Rate of FAS per 1,000 births</th>
<th>Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Passive Method</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>United States</td>
<td>1979-1992</td>
<td>0.20</td>
<td>General</td>
</tr>
<tr>
<td></td>
<td>1992</td>
<td>0.37</td>
<td>General</td>
</tr>
<tr>
<td></td>
<td>1993</td>
<td>0.67</td>
<td>General</td>
</tr>
<tr>
<td></td>
<td>1981-1986</td>
<td>0.60</td>
<td>African-American</td>
</tr>
<tr>
<td></td>
<td>1987-1991</td>
<td>0.08</td>
<td>Hispanic</td>
</tr>
<tr>
<td></td>
<td>1992</td>
<td>2.90</td>
<td>American Indian</td>
</tr>
<tr>
<td></td>
<td>1993</td>
<td>0.03</td>
<td>Asian</td>
</tr>
<tr>
<td></td>
<td>1994</td>
<td>0.09</td>
<td>White</td>
</tr>
<tr>
<td>Atlanta</td>
<td>1981-1989</td>
<td>0.10</td>
<td>General</td>
</tr>
<tr>
<td>Alaska</td>
<td>1977-1992</td>
<td>0.20 - 0.30</td>
<td>Alaska, non native</td>
</tr>
<tr>
<td></td>
<td>1993-1994</td>
<td>3.00 - 5.20</td>
<td>Alaska Native</td>
</tr>
<tr>
<td>North Dakota</td>
<td>1991-1994</td>
<td>1.10 - 2.00</td>
<td>General</td>
</tr>
</tbody>
</table>

*Clinic-Based Studies

| United States              | Various studies (avg.) | 1.90 | Western World |
|                            | Various states (avg.)  | 2.20 | United States and Canada |
|                            | Various studies (avg.) | 0.33 | United States |
| United States and other countries | Various studies (avg.) | 0.97 | Western World |
|                            | Various studies (avg.) | 1.95 | United States |
|                            | Various studies (avg.) | 2.29 | African-American |
|                            | Various studies (avg.) | 0.26 | White American |

| Active Case Ascertainment |                          | 9.00 | Plains Indian |
|                          | United States            |      |Navajo |
|                          | 1969 – 1982              | 1.40 | Pueblo  |
|                          | 1969-1982                | 2.00 | Southwestern Plains Indian |
|                          | 1969-1982                | 9.80 | Southwestern Indian |
|                          | 1969-1982 ( avg. )       | 1.80 | 1st grade students (one county) |
| Washington State         | 1995-1997               | 3.10 |                |

Approximately 1% children affect by FAS disorder. The majority children fail to receive a proper diagnosis and another study found 40 new born babies with FAS 100% left host without diagnosis.
4. DIAGNOSIS

4.1. Done for without confirmed maternal exposure
3Pattern of facial anomalies, Include palpebral fissures and abnormalities of premaxillary zone (flat upper lip, flat mid face)

4.2 Partial FAS with confirmed maternal alcohol exposure
Growth retardation, Complex pattern of behavioral (or) congestive abnormalities inconsistence with developmental level and unexplained by genetic background.

4.3 Environmental conditions
(eh: learning difficulties deficits in school performance)
- Poor impulse control
- Specific defects in mathematical skills
- Problems in memory attentions

5. Alcohol related birth defects
a. Cardiac: atrial septal defects
b. Skeletal: shortened fifth digits
c. Renal: hypo plastic kidneys
d. Auditory: conductive hearing loss

6. Alcohol related neurodevelopment disorders
Small head size.
- Impaired fine motor skills
- Poor hand eye coordination's.

7. Preventions for Parental Alcohol Use
7.1 Selective prevention
It shows the effects on target specific groups who are higher in the population in general.
For example people may reside in a community with heavy per capital use.

7.2 Indicated prevention: targets on individuals
For example persons with known drinking problem.
Give the various degrees of effort needed to address the problem of drinking in pregnancy among different populations.

8. Treatment of Fetal Alcohol Syndrome
Many doctors with recognized expertise in FASD recommend that, in general, most kids with FASD do with a combination of Stimulant + Selective Serotonin Receptive Inhibitor (SSRI). One exception is the child who has Bipolar disorder in addition to the FASD, in which case stimulants and/or SSRIs may cause an increase in behavior problems. (See note on "co-occurring conditions" below.) Many doctors also prescribe Clonidine for children who have problems with sleep, anxiety, or aggression in addition to the hyperactivity.

Stimulants that seem to be effective include Adderall, Ritalin, Concerta, or Dexadrine. SSRIs most commonly prescribed are Paxil, Prozac, Zoloft, and Celexa. Stimulants are reported to be effective 80%-90% of the time, when a correct diagnosis of FASD has been made
a. A daily dose of disulfiram can be given daily by orally
b. Risperdal (risperidone) antipsychotic (may cause weight gain)
c. Serzone (Nefazodone) antidepressant, also for seizures.

8.1 Involvement in Special Education and Social Services
12Children who receive special education geared towards their specific needs and learning styles are more likely to achieve their developmental and educational potential. Children with fetal alcohol syndrome show a wide range of behaviors and severity of symptoms. Special education allows for individualized educational programs. In addition, families of children with fetal alcohol syndrome who receive social services, such as respite care or stress and behavioral management training, have more positive outcomes than families who do not receive such services.

8.2 A Loving, Nurturing, and Stable Caretaking Environment
13Particularly sensitive to disruptions, transient lifestyles, or harmful relationships compared to children who do not have the condition. Community and family support are needed to prevent long-term effects in individuals with fetal alcohol syndrome.

9. EFFECTIVE MECHANISMS AT EARLY PREGNENCY
Effective cell death in a special population of embryonic cells which gives rise to facial anomalies and certain peripheral nerves. Effective loss of brain cell numbers in cerebellum

10 Alcohol teratogen effect on babys developing brain
During first semester alcohol interferes with the migration and organization of brain cells. During second semester i.e 10th-20th week after conception seems to cause more clinical features of fetal alcohol syndrome that at other times during pregnancy. During third semester hippocampus greatly affected leads to problems with encoding visual and auditory information.
CONCLUSION
Due to the up growing or due to work stress women along with men are liable to take alcohol this will cause the addiction during pregnancy therefore it causes the effects on fetus development so avoid drinking during pregnancy

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15. Featal alcohol syndrome and fetal alcohol effects neurobehavioral toxicology and tetralogy.