LETTER TO THE EDITOR

Cerebral Palsy and Alcohol Consumption during Pregnancy: Is There a Connection?

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(Rceived 15 June 2010; accepted 27 August 2010)

Fetal alcohol syndrome (FAS) is a clinically identifiable diagnosis, consisting of pre- and/or postnatal growth retardation (below the third percentile), characteristic facial features, including a thin upper lip, indistinct philtrum and short palpebral fissures (two standard deviations below normal for age), and neurobehavioral abnormalities (Abel, 1998; Jones et al., 1973; Plant, 1987; Sokol et al., 2003). Although FAS is the leading known cause of mental retardation in the USA (Abel and Sokol, 1986), brain injury involving milder forms is the leading known cause of mental retardation in the USA (Abel, 1998; Aronson et al., 1993; Meyer et al., 1990). The strongest evidence supporting an association between FAS and CP comes from a Swedish prospective study in which one-third of all pregnancies in Goteborg (population 450,000) were followed at antenatal clinics between 1977 and 1978. Four of 48 (8.3%) children with FAS were diagnosed with CP (one hemiplegia and three ataxia) compared with only one of the 7600 children born in the general Swedish population during the same period (Olegard et al., 1979). Less rigorous studies lacking control data noted two cases of CP among 20 children born to mothers with a history of chronic alcoholism in Australia (Lipson et al., 1983) and two cases among 40 FAS children in Scotland (Beattie et al., 1983). CP had also been noted prior to 1992 in several individual case studies of FAS (Bierich et al., 1976; Chan et al., 1991; Palmer et al., 1974). More recently, a 4% prevalence of CP was reported for children with FAS in North Dakota (although no details were given as to ascertainment; Burd et al., 2003). In the most recent retrospective study, from South Africa, 4 of 242 children with CP had FAS, a prevalence of 5.7% (Van Toorn et al., 2007). While these latter studies generally lack experimental vigor and some are sparse in detail (e.g. Burd et al., 2003), they are nevertheless consistent in pegging the prevalence of CP among children with FAS at between 2 and 10%.

A major problem in detecting FASD in children with CP is that neither CP or FASD are usually identified before 1 year of age (MacLennan, 1999). This means that unless a child also has the facial features characteristic of FAS, or a history of maternal alcohol consumption is known, the association will likely go unnoticed. Indirect evidence, that CP may be a comitant of prenatal alcohol exposure, is the presence of the many common secondary symptoms noted in Table 1.

While there are many possible mechanisms in the etiologies of both CP and FAS/FASD a mechanism both appear to have in common is prenatal oxidative stress (Abel and Hannigan, 1995; Goodlett and Horn, 2001; Korzeniewski et al., 2008; Lin, 2003). Although Korzeniewski et al. (2008) contend that inflammatory phenomena are the more probable causes of CP (Yoon et al., 2000), this does not preclude the involvement of oxidative stress since tissue hypoxia results in oxygen free radicals which in turn lead to inflammatory responses to deal with the injury (Stamler et al., 1997; Vink et al., 2005). Conceivably, both CP and FAS/FASD are conditions related to a more general fetal inflammatory response syndrome (Gomez et al., 1998).
exposure and its associated malformations as causal would be suggestive but much harder to prove since the neuroimaging correlates are similar, but a maternal history of drinking during pregnancy should be considered in cases of CP.

Acknowledgments — I thank Dr. Robert J. Sokol for critical comments and suggestions.

Conflict of interest statement. None declared.

REFERENCES


Table 1. Secondary symptoms common to CP and FASD

<table>
<thead>
<tr>
<th>Impaired development milestones</th>
<th>References</th>
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<tr>
<td>Obstructive sleep apnea</td>
<td>Abel, 1990; Dolk et al., 2006; Nagel, 2003; Abelev et al., 2001; Harris et al., 1993; Lin, 2003; Majewski, 1966; Nelson, 2008</td>
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<tr>
<td>Serous otitis media</td>
<td>Finucane, 1980; Harris et al., 1993; Landesman-Dwyer et al., 1978; Lin, 2003; Pierog et al., 1977; Staudey and Fried, 1983; Streissguth et al., 1980</td>
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<td>Delayed rolling over, sitting up, crawling, walking</td>
<td>Finucane, 1980; Harris et al., 1993; Landesman-Dwyer et al., 1978; Lin, 2003; Pierog et al., 1977; Staudey and Fried, 1983; Streissguth et al., 1980</td>
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<td>Speech/language disorders</td>
<td>Church and Abel, 1998</td>
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<td>Neuroanatomical disorders</td>
<td>Archibald et al., 2001; Clarren, 1986; Korzeniewski et al., 2008; Lin, 2003; Mattson and Riley, 1996</td>
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CONCLUSIONS AND IMPLICATIONS

Perhaps one of the reasons an association between CP and prenatal alcohol exposure has largely gone unnoticed is that clinicians/researchers have been overly attentive to the facial features currently required for the FAS diagnosis. However, as previously noted, prenatal alcohol exposure produces a spectrum of clinical conditions that include an increased risk for CNS deficits which can occur in the absence of FAS (Koren et al., 2003; Mattson et al., 1997). Another reason, the association between CP and in utero exposure has not received more scrutiny is that like FASD, CP is an umbrella term for a spectrum of motor dysfunctions. The previously cited evidence indicates that CP may occur in as many as 2–10% of cases of FASD, but has not been widely recognized as such.

Recognition of the occurrence of CP in conjunction with heavy drinking during pregnancy has important implications for medical malpractice. The most common reason, obstetricians are sued for medical malpractice, is the claim that they caused a child to be born with CP (ACOG, 2003). Evidence that such damage occurred prenatally from alcohol is strongly preemptive of such claims. Any case involving CP should, therefore, consider an evaluation of FAS/FASD. In the absence of FAS/FASD, evidence of prenatal alcohol...


