

Short Communication

Prenatal exposure to Pesticides on a plane and Cerebellum Atrophy: A case report

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One daughter in a family without antecedent with two children was diagnosed for a cerebellum atrophy causing neural dysfunction and motor and developmental problems. Various genetic, metabolic, and microbial origins of the disease were explored without results. The mother was exposed during pregnancy to an intense and nauseous pesticide spray by the crew to monitor mosquitoes in a plane before departure from South America, at the time of cerebellum development. A better prevention for pregnant mothers is possible in air planes during compulsory insecticide sprays to avoid mosquitoes.

Key words: Neurodevelopment, brain, DDT, pyrethroids.

INTRODUCTION

This case concerns an observation of the medical file of the eldest of two daughters of a family. She was born at term in June 2004, with a congenital ataxia with normal karyotype, axial and peripheral hypotonia and progressively detected psychomotor delay. A cerebellum syndrome dysfunction was diagnosed at a clinical level as static and kinetic with preserved reflexes. When 10.5 years old she was still unable to walk and speak normally and had a size minus 3 standard deviations, weighing 44 kg, and strabismus. The one year younger sister is normal; there is no familial antecedent. The pregnancy was not accompanied by infection, alcohol, and tobacco or drug consumption.

Diagnosis

The first diagnosis was a partial agenesis of the cerebellar vermis. The magnetic resonance imaging confirmed in March 2007 a cerebellum atrophy concerning essentially the vermis and to some extent cerebellum hemispheres with no other neural problem. The deeper structures appear to be present. During neuroimaging after cerebral scintigraphy in 2013, there was a clear cerebellum hypoperfusion, related to the known atrophy, with a moderated one at the level of the brainstem, with no degeneration since the previous control in 2010.

Genetic and microbial origins explored

The genetic and linked possible metabolic deficiencies known with this type of diagnosis were further explored. There was no congenital disorder of glycosylation, no Smith-Lemli-Opitz syndrome identified, and no aprataxin gene mutation. Similarly, no traces of microorganisms were present in the cerebrospinal fluid where the amino acids were at normal levels. All is documented in the medical file of the patient opened with informed consent after request from the family.

Environmental hypothesis

After medical explorations of known genetic and microbial origins of the symptoms, the environmental hypothesis was explored. After deep questioning of the parents, the only exposure to toxicants identified during the pregnancy was a clinically disturbing insecticide spray to monitor mosquitoes. This happened compulsory on a plane departing from Mexico to Europe, during a travel tour; it is usual for the company. This was exactly at the end of the third month of fetal age.

The cerebellum and especially vermis is in development at this period between week 13 and 14 at the end of the first trimester in utero (Chang et al., 2012). The World Health Organization during hot weathers recommends treatments in planes with persistent insecticides including DDT and pyrethroids (Weisel, 2012).

These insecticides are identified as classical endocrine and nervous disruptors and neurotoxics especially during development (Eskenazi et al., 2009, Koureas et al., 2012). It is also known that neurosteroids play an important role in the development of the cerebellum. In particular, estradiol and progesterone appear capable of inducing increases in dendritic spine density during development, and there is evidence that both are synthesized in the cerebellum during critical developmental periods and this can be disturbed by chemicals (McCarthy 2008). The Purkinje cells and the granule cells in this area are the most important targets in cerebellum for toxic substances (Sawant et al., 2013). There is no effective physiological barrier, placenta, or blood-brain, which could stop the pesticide entrance in neural tissues. The toxicity of adjuvants helping penetration may well increase the risk and produce common effects for several pesticides (Mesnage et al., 2014). Other pesticides such as warfarin have been involved in partial brain atrophy after fetal exposure during pregnancy (Simonazzi et al., 2008). It is known since decades that chemicals such as anticoagulants have similar effects at similar periods (Stevenson et al., 1980). However this is documented mostly after oral route exposure. Cerebellar ataxia even happened after brain development in an adult after poisoning with a rodenticide, sodium monofluoroacetate (Trabes et al., 1983). It is also reported that in a pregnant mother with a 4-week-old fetus, a mixture of insecticides sprayed in a field was involved in a cerebral and cerebellar atrophy among many other anomalies (Romero et al., 1989). However, the present report is the first of this pathological fetal development after exposure of a pregnant mother to compulsory air spray on a plane.

CONCLUSION

Even if other genetic unknown origins are always possible, a peculiar sum of circumstances occurred that may explain an environmental origin of a neurodevelopmental defect. This could be due to toxic pesticides sprayed on planes without taking into account the specific period of sensitivity of brain development during the pregnancy of mothers, nor the presence of pregnant mothers themselves, nor children or sensitive persons on board.

In cases of long term or delayed toxicological effects of mixtures from environmental origin in humans, it is almost impossible to wait for a scientific causal explanation without doubt. Epidemiological or toxicological studies are not really feasible today on this subject. The addition of reports from the field may initiate prevention at this level, and a better benefit/risk assessment of conventional practices for plane companies using pesticides.

Conflicts of interests: none.

Family consent: parental informed consent obtained.

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