Kawasaki`s Disease, Acrodynia, and Mercury

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Abstract: A superantigen or autoimmunity has been hypothesized to be the main cause of the Kawasaki`s Disease but the etiology is unknown. Medical literature, epidemiological findings, and some case reports have suggested that mercury may play a pathogenic role. Several patients with Kawasaki`s Disease have presented with elevated urine mercury levels compared to matched controls. Most symptoms and diagnostic criteria which are seen in children with acrodynia, known to be caused by mercury, are similar to those seen in Kawasaki´s Disease. Genetic depletion of glutathione S-transferase , a susceptibility marker for Kawasaki´s Disease, is known to be also a risk factor for acrodynia and may also increase susceptibility to mercury . Coinciding with the largest increase (1985-1990) of thimerosal (49.6% ethyl mercury) in vaccines, routinely given to infants in the U.S. by 6 months of age (from 75µg to 187.5µg), the rates of Kawasaki´s Disease increased ten times, and, later (1985-1997), by 20 times. Since 1990 88 cases of patients developing Kawasaki´s Disease some days after vaccination have been reported to the Centers of Disease Control (CDC) including 19% manifesting symptoms the same day. The presented pathogenetic model may lead to new preventive- and therapeutic strategies for Kawasaki´s disease.

Keywords: Kawasaki's disease, mercury, acrodynia, thimerosal, ethyl mercury, methyl mercury, vaccine, dental amalgam.

INTRODUCTION

Kawaski's Disease (KD), first described in Japan (1967), is an acute febrile multiorgan vasculitis, which predominantly (75 – 80%) affects children younger than 5 years. The disease has an increasing frequency and, in developed countries, has surpassed rheumatic fever as the leading cause of acquired heart disease in children. Early intravenous immunoglobulins in combination with acetyl-salicylic acid have significantly reduced the prevalence of coronary artery abnormalities.

There is no test for diagnosing KD; thus the diagnosis is based on clinical signs and symptoms. Despite of this, of all cases atypical ones amount to 10-45%. Interestingly, another childhood disease, acrodynia (AD) shares most of its diagnostic criteria with KD.

By now, the cause of KD is unknown. Antigens from infections as well as superantigens and genetic polymorphisms have been implicated in the etiological hypotheses. In this review of the literature and analysis of the U.S. Vaccine Adverse Effects Reporting System (VAERS), we hypothesize that prenatal and postnatal exposure to mercury (and synergistic toxins) may be a pathogenic factor in KD.

The VAERS database is an epidemiological database that has been maintained by the Centers for Disease Control (CDC) since 1990 as a surveillance tool to evaluate vaccine safety. An examination of the VAERS database (online public access version: http://vaers.hhs.gov/scripts/data.cfm) with reports entered through January, 31, 2008 was undertaken. The keywords: "kawasaki's disease" and "kawasaki's syndrome." were used. An additional search for "mucocutaneous lymph node syndrome" did not yield any results. The strength of the VAERS database stems from its large reporting base. Its potential weakness is that all vaccine-associated adverse events experienced are not reported.

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ACRODYNIA, KAWASAKI`S DISEASE AND MERCURY

AD was considered a mysterious, systemic disorder, mainly affecting children under the age of five. At its epidemic height (1880-1950), it affected about one in 500 children in industrialized nations [1].

The onset of AD is characterized by high fever lasting more than 5 days; a varying rash such as erythematous plaques, or appearing as measles or scarlet fever; swollen lymph nodes, particularly in the neck; bright red, swollen hands and feet; red, irritated eyes without discharge; bright red, irritated mouth, lips, and throat [2,3]. Neurological, cutaneous, and cardiovascular symptoms are most commonly seen. However, the disease is highly variable; cutaneous symptoms may be mild or lacking while neurological symptoms always seem to be present. It was explained as an infection or nutritional deficiency and it occurred mostly in the teething period [4].

In 1953, as a result of work by Warkany and Hubbard, mercury – coming from teething powders, baby powders, and diapers treated with calomel (85% mercurous chloride) [2] - was accepted as the cause of AD [2]. After a federal ban of these mercury-containing products in 1954, AD disappeared [1]. It should be noted that, *in vitro*, mercurous chloride is one of the least toxic forms of mercury, about 100 times less toxic than are mercury vapor or ethyl mercury contained in vaccines [5]. In addition, it was reported that applications of vaccines (containing ethyl mercury in thimerosal) preceded the onset of AD in several cases [2,3].

KD shares its diagnostic criteria with those of the onset of AD; the two diseases are similar in their clinical appearance. More than 150 symptoms and about 50 laboratory findings which are seen in KD were also described in cases with mercury poisoning (MP), too. (See Table **1**) [2,3,6-57]. KD affects males twice as often as females. This may be explained by *in vitro* studies on human cells which have shown that testosterone synergistically increases the toxicity of mercury, while estrogen protects against mercury toxicity [1].

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