Mercury and Acrodynia

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Acrodynia is principally a syndrome of chronic mercury poisoning. The almost forgotten disease, mostly affecting infants and young children, is probably the best studied effect of human mercury poisoning.

Acrodynia is characterized by a) profound changes in temperament, b) skin alterations, c) neurologic symptoms, d) tachycardia, and e) stomatitis (Gorlin et al, 1976).

Acrodynia was first recognized in France in 1828 (Hanson 1987), and the term was introduced in 1830 by Chardon (Chardon, 1830). The term is derived from Greek, denotes painful extremities (Gorlin et al, 1976).

The condition was recognized in Australia in 1890 (Akabane 1983). Selter reported in 1903 eight cases of the syndrome among children between one and three years of age (Selter, 1903). Selter used the term "trophodermatoneurose", and exhibiting the characteristic picture of the syndrome. The neurologic aspects of acrodynia were emphasized by Swift in Australia in 1914 (Swift, 1914), and by Feer in Switzerland in 1923 (Feer, 1923). The condition was established as a clinical entity in the British and American literature by Byfield and Bilderback in 1920 (Bilderback, 1920).

The syndrome is also known as Pink Disease, Swift Disease, Feer Disease, Selter Disease, Erythroderma, Erythroderma polyneuritis, Dermatopolyneuritis, Trophodermatoneurose, Erythema arthricum epidemicum, Vegetativ neurose, and Vegetativ encphalit. These terms describe different aspects of the syndrome.

The etiology of acrodynia was rather vague, and it was various theories at different times (Obura, 1965). The following theories were most popular: avitaminosis, chronic infections, combination of dietary deficiency and infection, suprarenal insufficiency, derangement of sympathetic nervous system, and mercury poisoning.

According to Hanson and Pleva (Hanson & Pleva, 1991) was the mercury etiology first suggested in 1846, and again in 1922. Warkany and Hubbard from USA suggested in 1948 that the etiology of the syndrome might be chronic mercury poisoning, particularly that administrated to infants in the form of teething powders (Warkany & Hubbard, 1953).

In a review article from 1953 Warkany and Hubbard write (Warkany & Hubbard, 1953): "... calomel has been the most frequent source of mercury in the cases of acrodynia ... As pointed out by Fanconi and associates acrodynia often follows worm treatment with calomel-containing anthelmintics ... In other cases calomel was given for constipation, diarrhea, cough, or for unspecific reasons. In England, in the southern United States, and in East Africa, calomel was usually ingested in the form of teething powders."

Warkany and Hubbard demonstrated in 1948 mercury in 25 out of 28 cases of acrodynia (Warkany & Hubbard, 1953). Fanconi in Switzerland demonstrated in 1949 mercury in urine in 19 out of 20 cases (Obura, 1965). In the review article from 1953 presented Warkany and Hubbard a series of 28 new cases of acrodynia (Warkany & Hubbard, 1953). In all these cases exposure to mercury was demonstrated by history or by urinalysis. Warkany and Hubbard concluded that mercury exposure only can be rulled out if the history and repeated urinary determinations are negative (Warkany & Hubbard, 1950, 1953).

Acrodynia has been described as due to unusual sensitivity or idiosyncrasy to mercury (Warkany & Hubbard, 1953). Clinical manifestations may include several of the following symptoms which, in the well established cases, are so distinctive that there is practically no differential diagnosis (Gorlin et al, 1976; Akabane, 1983): pink hands and feet, scarlet tip of nose and cheeks, extreme irritability and restlessness alternating with periods of apathy, insomnia, anorexia, pain in extremities, profuse perspiration, generalized skin rashes, photophobia, desquamation, itching, salivation, loss of teeth,

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hypotonia which permits the child to assume many different and bizarre positions. There may be albuminuria but no blood or CSF changes, and no characteristic urinary findings except an abnormally raised level of mercury (Obura, 1965).

In England, Australia, and in the southern United States acrodynia usually occurred in infants and young children up to two years of age. The greatest age frequently occurred in the group nine months old. Acrodynia was occasionally reported as locally epidemic, and among members of the same family (Hanson 1987). The mortality rate was reported to be about five percent.

The mercury-containing teething powders were withdrawn from the market in Australia in 1953 (Hanson, 1987), followed by USA. However, the role of mercury as the primary source of acrodynia was not universally accepted even as late as 1956 (Dathan, 1965), especially in England.

The incidence and mortality rate of acrodynia have fallen dramatically since mercury-containing teething powders were withdrawn from the market. The official death-record from acrodynia in England between 1939 and 1948 is 585 (Hanson, 1987). The death rate from acrodynia shows a similar decline, from 57 in 1950 to seven in 1955, and to none in 1961 and 1962 (Dathan, 1965).

Have we something to learn from the history of acrodynia? Yes, I think so. Toxicological effects of dental amalgam has never been totally studied. Dental amalgam has however been used in dentistry in the Western World for more than 150 years (Bjørklund, 1989). Is amalgam therefore completely safe to use? This is one of the most common arguments for continued use of dental amalgam. This argument is not valid. The etiology of acrodynia was unknown for more than 100 years, before the role of mercury as the primary source of this disease was pointed.

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