Kor. J. Env. Hlth., Vol. 31, No. 6, pp 451~456(2005)

Review

Minamata Disease and the Mercury Pollution of the Globe

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(Received May 17, 2005/Accepted Nov. 3, 2005)

Abstract: Minamata disease made its first appearance in the world at Minamata City, Kumamoto Prefecture, in May 1956. In 1962 methyl mercury poisoning through the placenta was found for the first time in the world. This was called congenital Minamata disease. In all cases the clinical symptoms were consistent with those of cerebral palsy. The time and place of outbreak were the same as those for Minamata disease. Their mothers had eaten fish and shellfish during pregnancy. The principal symptoms of congenital Minamata disease are mentalretardation (100%); primitive reflexes (100%); disturbance of coordination (100%); dysarthria (100%); limb deformation (100%); growth disorders (100%); nutritional disorders (100%); chorea-athetose (95%); and hypersalivation (95%). However, today, when the world is polluted by mercury in various places and at various levels, the data we need is not represented by those severe cases, but rather by the chronic milder type. Even in Minamata, the issue of Minamata disease has not been resolved. And likewise, on a global scale the problem of Minamata disease is not yet over.

Keywords : Minamata disease, mercury, poisoning, congenital, Chisso

Minamata disease made its first appearance in the world at Minamata City, Kumamoto Prefecture, in May 1956.[1] The second outbreak occurred in Niigata City, Niigata Prefecture in 1965.[2] Both cases were attributed to methyl mercury poisoning resulting from mercury used as a catalyst in the production of acetaldehyde. Factory effluent from Chisso Corporation containing methyl mercury accumulated in fish and shellfish, poisoning people who consumed them.[3] This type of methyl mercury poisoning conveyed through the food chain as a result of environmental pollution had never been seen before. Organic mercury poisoning prior to Minamata disease occurred as a result of direct poisoning through handling on the job or through accidental contact.

The Shiranui Sea, the location of the first outbreak of Minamata disease, is an inland sea of approximately 1,200 square kilometers, once rich in fish and shellfish. At the time of the pollution incident, the coastal population of some 200,000, were engaged in fisheries and related industries. Fish and shellfish constituted the main meal for local residents year around. Some people consumed more than 500 grams of fish per day.[3,4]

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In the case of the second outbreak of Minamata disease in Niigata, residents likewise consumed large quantities of fresh water fish from the Agano River. In this case the source of pollution was a chemical factory called Showa Denko, sixty kilometers upstream from the river mouth.[2]

According to Dr. Tokuomi, patients with Minamata disease displayed the following typical symptoms: sensory disturbance (100%), constriction of the visual field (100%); impairment of coordination (93.5%); dysarthria (88.2%); impairment of hearing (85.3%); and tremors (75.8%). Moreover, the fact that 82.4% of patients also had disturbance of gait, indicated the serious nature of their symptoms.[1] These patients all exhibit typical symptoms of methyl mercury poisoning, but there were in fact many different combinations of symptoms, and there were patients in various stages of mercury poisoning, from very light to very severe cases. In fact, there were far more patients with non-typical symptoms than there were of those who fit a standard profile.[4,5]

Especially characteristic of Minamata disease are glove and stocking type and perioral sensory disturbances. These symptoms are extremely rare in other diseases.[4] Moreover, the pattern of such sensory disturbances is similar to that of peripheral nervous disorders. However, according to later studies, it became clear that these symptoms belonged to a category of central nervous system disorders. This

is because patients were found to have sensory agnosia as well as increased the thresholds of two-points discrimination.[6,7] In the case of chronic type or in mild cases, it may be difficult to identify coordination disorders. In such cases, electroophthalmography (EOG) and optokinetic nystagmus pattern (OKP) can be used for obtaining referential data.[4] In the case of Minamata disease the possibilities of the occurrence of mild and non-typical symptoms are far greater than those of typical serious symptoms.

At present, the levels of mercury in the hair (average, 2.6 ppm) of residents and of fish and shellfish (average, 0.4 ppm) along the Shiranui Sea coast are dropping. However, we still see a great variety of symptoms among the residents. These symptoms have probably remained from the time the pollution was at its worst.[8] The glove and stocking type sensory disturbance is particularly high. The patients shows the pattern of clinical symptoms and sensory disturbances of those suspected of suffering from Minamata disease.[4] The hair mercury level of residents around the Shiranui Sea has come down, but various symptoms remain.[8]

In 1962 methyl mercury poisoning through the placenta was found for the first time in the world. This was called congenital Minamata disease.[9,10] The author has identified sixty-four congenital cases.[4] Of these patients, thirteen have already died. In all cases the clinical symptoms were consistent with those of cerebral palsy. The time and place of outbreak were the same as those for Minamata disease. In the areas of greatest outbreak, 9% of children born were handicapped. Everyone in their family had contracted Minamata disease. Their mothers had eaten fish and shellfish during pregnancy. The mothers had light symptoms of sensory disorders, hearing impairment, and disturbance of coordination.

The principal symptoms of congenital Minamata disease are mental retardation (100%); primitive reflexes (100%); disturbance of coordination (100%); dysarthria (100%); limb deformation (100%); growth disorders (100%); nutritional disorders (100%); chorea-athetose (95%); and hypersalivation (95%).[9,10]

We do not know the levels of mercury at birth of congenital patients, because the disease was not identified until five to eight years after these children were born. It is an old custom in Japan to preserve the umbilical cord of the newborn. Taking note of this, the author and others collected and measured the mercury concentration in these cords. The mercury content was very high. Moreover, there was a direct correlation between our estimate of the release of methyl mercury in Chisso's effluent and the concentration of methyl mercury in the umbilical cords. The level of mercury in congenital cases was over 1.0 ppm. However, in the case of infantile Minamata disease, the mercury levels in the cords were also high. This was because the environment continued to be polluted.[11,12]

On the basis of both patient pathology and experimentation it became clear that congenital Minamata disease belonged to the category of viviparous methyl mercury poisoning. This was the first time in the history of mankind that a poison had passed through the placenta.[1,13] Moreover all of these congenital Minamata disease patients had severe symptoms; none had light or non-typical symptoms. In 1971, the author and others noted that school children in the polluted areas showed mild symptoms of dysarthria, dyskinesia, and mental retardation, and we thought these were due to the influence of methyl mercury.[9] This was proven by the high mercury values of the preserved umbilical cords of children in the polluted area who had mental retardation not accompanied by dyskinesia.[12] According to recent research, even in the cases of mothers with hair mercury values safely under the 50 ppm standard, there can be influences on the fetus. This influence does not take the form of severe symptoms as in congenital Minamata disease cases, but results in mild mental impairment and nervous system disorders (IPCS).

Until 2004 there were 2,200 patients officially certified with Minamata disease. There were 800 certified patients in Niigata. However, there are some 12,000 more suspected of suffering from Minamata disease who are not recognized. Some of these are chronic cases, and their symptoms are too mild to meet certification standards.[8]

Because it was in 1968 that Chisso Chemical Plant in Minamata ceased production of acetaldehyde, there is a good possibility that methyl mercury was discharged into Minamata Bay in factory effluent until that date. Because high concentrations of methyl

mercury were found in the mud at the bottom of Minamata Bay even after production ceased, the contamination of fish and shellfish continued. Removal of sludge and reclamation did not begin until 15 years after the company ceased production of acetaldehyde.[14] If production had stopped earlier, or if the sludge had been cleaned up sooner, many people would not have become ill, or those who did would have had lighter symptoms.

In the 1970s there was a mercury poisoning incident in Canada. The source of pollution was a plant producing caustic soda. The inorganic mercury used as a catalyst by this factory became methylated in the natural environment, accumulating in fish. The polluted site was on a reservation belonged to First Nation peoples of Canada. Lake fish constituted their staple diet.[15,16] Cats on the reservation came down with Minamata disease.[17] In experiments done with cats eating fish from the lake, the disease was induced within 90 days.[18]

The hair mercury level of these First Nation peoples exceeded 50 ppm. Those with the highest concentrations had over 100 ppm. Moreover, mercury levels were higher in hair grown in the summer. Mercury levels in hair grown out in the winter were lower. This is because they ate more fish in the summer than in the winter. It was clear that they had been poisoned through consumption of fish.[16] In 1975, I found these people to have mild neurological symptoms.[15] Although the Canadian government recognized the existence of Minamata disease, the government position was that Minamata disease had not occurred on the reservation because there were no serious typical cases. The author returned to examine these people in 2002 and 2004. Although we must also consider problems of aging, all of the symptoms of Minamata disease had worsened. Although the Canadian government did not admit that there was an outbreak of Minamata disease, in 1985, a Mercury Disability Board was established. 140 people were certified as having the disease, and they received compensation. There are some complications resulting from illness, but based on actual clinical symptoms, these appear to be cases of Minamata disease. 70% of patients examined showed glove and stocking type sensory disorder. The author and others believed that these patients had Minamata disease. However, patients like this with light symptoms were not recognized as having Minamata disease and did not receive compensation. On the whole, their hair mercury levels were low, but some levels were as high as 25 ppm, and we may presume that the pollution remained in some localized areas and special individual.[19]

Early on Japan stopped using inorganic mercury as a catalyst in the production of caustic soda, but many developing countries have yet to switch to other means of production. Factories in Canada were severely criticized for continuing to use mercury as a catalyst. Similar incidents have occurred in Asia, and in Central and South America.

In Jirin City, China, a chemical factory dumped methylmercury directly into a nearby river. This was an acetaldehyde plant, just like Chisso Corporation in Minamata. However, in this case, nearly all of the fish died, and fortunately people did not contract Minamata disease. However, fish continued to live downstream in Jirin and Heilongjiang Provinces, and some fishermen who ate the river fish had hair mercury levels of over 50 ppm and displayed mild symptoms of Minamata disease. Chinese scientists said that although the symptoms were mild, they recognized them as Minamata disease. There were also cases of chronic Minamata disease, in which the symptoms persisted even after people stopped eating fish.

According to an investigation conducted by Haerbin University, fishermen on the Songhua River showed mean and maximum hair mercury levels of 20 and 71.2 ppm respectively. 44% of those with hair mercury levels of more than 20 ppm, displayed sensory disorders, and 17% displayed constriction of the visual field.[21]

In the 1980s methyl mercury pollution became an issue in the Amazon River basin. The author and others conducted hair mercury level analyses in conjunction with the University of Tokyo, Yokohama National University, and Para State University, Brazil. As a result it was shown that not only laborers working in the gold mines (*garimpeiros*), but fishermen along the river had high hair mercury levels.[21] At the gold mines (*garimpo*), in order to capture placer gold, sand is mixed with mercury to make an amalgam, and after the sand is washed away, the mercury is burned with a torch. The mercury vaporizes, leaving the gold. The author determined

that the laborers suffered from inorganic mercury poisoning from breathing the vapors.

Downstream, fishermen who had nothing to do with gold mining, were found to have high levels of mercury in their hair. In 1994 these levels measured over 100 ppm. In over 90% of the cases this was methyl mercury. Because fish caught in their area had maximum mercury levels of 3.17 ppm, we believed that they became ill due to fish consumption. We worried about the influence of methyl mercury on the health of the fishermen. A special feature of the author's survey was to also check subjective and clinical symptoms. Then we would be able to correlate hair mercury levels with the symptoms. However, Minamata disease was not detected by the author and his colleagues in clinical examinations conducted in 1994.[22,23]

There are five stages before the outbreak of Minamata disease.

In the first stage the *garimpeiro* inhales mercury vapors directly and gets inorganic mercury poisoning.

In the second stage, the mercury that was released enters the natural environment and becomes methylated.

In the third stage, methylmercury accumulates in fish and shellfish. Mercury levels are biomagnified in the food chain.

In the fourth stage, methylmercury begins accumulating in the human body as contaminated fish is consumed. Mercury levels in human hair, blood, urine, umbilical cords and so on rise.

In the fifth (final) stage there is an outbreak of Minamata disease.

The Amazon River basin is now at stage 4. At this point the issue is the question, "What constitutes Minamata disease?" In other words, what are the criteria for diagnosis? In the past, in Minamata we certified as Minamata disease patients only those who had a full spectrum of serious symptoms. Those who had mild or non-typical symptoms were said not to have Minamata disease. Thus, the former concept of Minamata disease became very narrow. If we look at those people affected by methylmercury in Canada and the Amazon River basin from this perspective, we cannot say that an outbreak of Minamata disease occurred. When we re-visited the Amazon basin in 1995, our clinical examinations revealed that fishermen already exhibited mild symptoms.[24]

Because the fetus is the most sensitive of all to methyl mercury, the welfare of the fetus is our greatest concern. Clinical epidemiological investigations conducted in Canada, Iraq, New Zealand, and the Faeroes Islands revealed that even if the hair level mercury of the mother was below the so-called safe maximum of 50 ppm, there was a chance that the fetus would be affected. It has been reported that 10 ppm is the line below which the fetus will not be affected.[25,26] When the author and his colleagues conducted a study of the mercury levels in umbilical cords of congenital Minamata disease patients, we found that the mothers displayed a minimum hair mercury level of 21.5 ppm.[12,23]

Extraction of gold using mercury occurs in East Africa, especially around Lake Victoria; in the Philippines, and in Indonesia. The author has conducted surveys in these regions, but to date neither gold miners nor fishermen show signs of Minamata disease and their hair mercury levels are low. This is because the gold mining occurs on a smaller scale, and a smaller quantity of mercury is used in the process than on the Amazon River. However, if the scale of mining increases, there will be cause for concern.[27]

Although mercury levels of fish caught in Lake Victoria were very low—0.0089 through 0.063 ppm[27]—some women showed extraordinarily high hair mercury values, ranging from 470 to 720 ppm. Because this was inorganic mercury, however, we determined that contamination did not take place through the food chain. Our investigation revealed that the women were using cosmetic soaps with a mercury content of 2% to whiten their skin; this was what was affecting their health.[28] The government prohibited their use, but such substances could become a new source of mercury pollution.

Today 2,200 patients are officially certified with Minamata disease are receiving compensation from Chisso. In addition, approximately 12,000 more suffers who had requested to be recognized, reached a compromise settlement with Chisso in 1996. In October 2004 the Supreme Court ruled that the country and prefecture were responsible for Minamata disease. This gave rise to a new movement in Minamata. 2,000 more sufferers newly have de-

manded certification and compensation to goverments.

Minamata disease was discovered through the presence of patients with severe textbook symptoms. However, today, when the world is polluted by mercury in various places and at various levels, the data we need is not represented by those severe cases, but rather by the chronic milder type. Unfortunately we have not collected enough data of this nature.

Even in Minamata, the issue of Minamata disease has not been resolved. And likewise, on a global scale the problem of Minamata disease is not yet over.

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