NEUROTOXIC EFFECTS IN WORKERS OF THE CLINICAL THERMOMETER MANUFACTURE PLANT

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Abstract

Objectives: Occupational exposure to mercury can induce adverse health effects, and the central nervous system is the major target of its toxic action. This problem especially arises in plants involved in the manufacture of mercury-containing products, where an appropriate protection against mercury exposure is not ensured. The aim of this study was to assess health effects of mercury, especially neurotoxic effects and oral disorders, in workers employed in a clinical thermometer manufacture plant and to determine mercury concentrations in the workplace ambient air.

Materials and Methods: The study population comprised 143 workers, including 51 (35.7%) men and 92 (64.3%) women employed in the plant. Mean age in the whole group was 29 years (range, 18–55 years). It was divided into three groups: control, mercury absorption and mercury poisoning. A questionnaire-based interview was used to collect data on medical history, occupational exposure and employment. For clinical diagnosis, all subjects underwent physical, neurological and oral examinations. Mercury concentrations in the air were recorded by Hg monitoring instrument and mercury levels in collected urine samples were determined by cold vapor atomic absorption spectrophotometry.

Results: Neurasthenic symptoms were found in 51.75% of the subjects, emotional changes in 27.27%, tremors in 11.19%, and inflammations in 21.68%. The case percentage of neurological symptoms in the control and mercury absorption groups did not show significant difference, but it was significantly higher in the mercury poisoning group.

Conclusions: The high occupational exposure to mercury, found in the plant and evidenced by urinary Hg concentration ≥ 0.05 mg/l, can result in chronic quantitative neurotoxic effects and qualitative health changes. Therefore, constant monitoring of the work environment and checking of workers' health status should be ensured. In addition, appropriate steps should be taken to improve work conditions and promote health among the employees.

Key words: Mercury exposure, Neurotoxicity, Chronic effects, Quantitative harm, Qualitative changes, Environmental monitoring, Medical assessment, Health improvement

INTRODUCTION

Liquid mercury (Hg) is widely used in the manufacture of industrial and medical appliances, such as manometers, barometers and thermometers. Mercury is a toxic metal known to exert several detrimental effects on human health [1]. The high exposure to mercury can lead to chronic Hg poisoning, disorders of the central nervous system (CNS) and gum inflammations. The major CNS disorders include neurasthenia (e.g., headaches, insomnia, drowsiness, weakness, fatigue, muscle atrophy, twitching, intellect decline, memory loss, altered nerve reactions), emotional changes (e.g., mood swings, irritability, nervousness, anxiety, poor self-control, shyness, depression, timidity and loss of confidence), and tremors, which can even lead to vision and hearing loss and hallucinations [2]. In most instances, the work-related adverse effects of mercury exposure are due to long-term and low-level exposures. High-level exposures may occur in production plants with poor working conditions and insufficient health protection, which can result in serious adverse health effects in workers. According to the well-evidenced data, occupational exposure to mercury can induce neurasthenic symptoms [3], impairment of psychomotor functions and emotional disorders [4].
The aim of the study was to identify neurotoxic effects, especially neurasthenic symptoms and emotional changes, in a population of workers employed in a clinical thermometer manufacture plant. All subjects gave informed consent and the study design was approved by the local health bureau.

MATERIALS AND METHODS

The study population comprised 143 workers, including 51 (35.7%) men and 92 (64.3%) women employed in the clinical thermometer manufacture plant. Mean age in the whole group was 29 years (range, 18–55 years). The mean employment duration was 6 years (range, 4 months – 26 years). A questionnaire-based interview was used to collect data on medical history, occupational exposure, employment, neurasthenic symptoms (headache, dizziness, insomnia, memory loss, fatigue, weakness), and emotional changes (mood swings, irritability, nervousness, timidity, loss of confidence). For clinical diagnosis, all subjects underwent physical, neurological and oral examinations. Clinical tests were used to record tremors (hand, tongue and eyelid) and inflammations (oral and/or gum). To assess mercury absorption and poisoning, urine samples were collected. Mercury concentrations in the indoor air were recorded by Hg monitoring instrument and environmental pollution in workplaces (purifying mercury, filling mercury, temperature measurements, sealing, engraving, and packing) was evaluated. Urine mercury concentrations (UMC) were determined by cold vapor atomic absorption spectrophotometry. According to the national health criteria, the UMC was defined as ≥ 0.01 mg/L [5]. The reference UMC indicating Hg absorption and Hg poisoning was established at 0.02–0.04 mg/L and ≥ 0.05 mg/L, respectively [6]. Depending on the UMC value, the workers were divided into three groups: control (C) (UMC ≥ 0.01 mg/L), mercury absorption (MA) (UMC 0.02–0.04 mg/L) and mercury poisoning group (MP) (UMC ≥ 0.05 mg/L).

The Chi-square test was used to assess statistically significant differences (p < 0.01) between the control, mercury absorption and mercury poisoning groups. The clinical thermometer manufacture plant lacked appropriate protection against mercury exposure. Workplaces were narrow, small and poorly-ventilated. Mercury drops were scattered on the ground and tables. Some of the workers drank water in the workplace and left the plant in working clothes. Clinical thermometers were manufactured manually.

RESULTS

The results of the monitoring, survey and examinations showed that workplaces in the plant were polluted with mercury. The study indicated mean mercury concentrations in the workplace ambient air of 0.027 mg/m$^3$ (range, 0.011–0.057 mg/m$^3$), and mean urine mercury concentration of 0.03 mg/l (range, 0.01–0.05 mg/l). All the monitored indoor air samples reached or exceeded the national

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<th>Table 1. Results of monitoring, survey and examinations of the workers employed in the clinical thermometer manufacture plant</th>
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safety criteria for mercury concentration (≤ 0.01 mg/m³) [7], which led to increased urine mercury concentrations and the higher incidence of neurotoxic effects (Table 1). The outcomes of physical examinations demonstrated that 51.75% of the workers showed neurasthenic symptoms, 27.27% emotional changes, 11.19% tremors, and 21.68% of the subjects displayed oral and/or gum inflammations. The higher exposure to mercury first induced neurasthenia and serious neurological symptoms then emotional changes, oral and/or gum inflammation, and finally tremors. However, the percentage of cases with neurological symptoms in the mercury absorption group was not significantly higher than in the control group (MP vs. C, p > 0.01); yet it was significantly higher in the mercury poisoning group than that in either group (MP vs. C, p < 0.01; MP vs. MA, p < 0.01). There was significant relationship between severity of neurotoxic effects and the level of mercury exposure.

DISCUSSION
The study carried out in the clinical thermometer manufacture plant showed that the higher occupational exposure to mercury could lead to chronic toxic effects, especially if an appropriate protection of workers' health is not ensured. Numerous laboratory data demonstrated that mercury compounds could inhibit neuronal glutamate transport [8] and induce microtubule alterations, oxidative damage, impairment of calcium homeostasis, and intensify glutamatergic neurotransmission [9]. Methylmercury can inhibit L-glutamine-D-fructose-6-phosphate aminotransferase (GFAT) and ubiquitin transferase (Ubc3) in brain cells [10], reduce the rate of synthesis of ATP and poly(A)-segments of mRNAs in cells [11], and decrease protein synthesis in the retina and optic nerve [12]. Mercury exposure is associated with a wide range of central and peripheral nervous system dysfunctions [13], fine tremors in the fingers, eyelids and lips, which are early signs of mercury toxicity [14]. It has recently been suggested that Hg can impair the function of astrocytes with subsequent neuronal lesion [15] and lead to chronic neurotoxic effects [16]. Occupational exposure to mercury vapors may lead to neurological alterations [17], and chronic exposure may result in slowing of conduction velocity in long nerves [18]. Some data indicated that despite the fact that mercury-exposed plant workers showed more symptoms than unexposed controls the association of the exposure with neurological functions was poor [19]. There were no differences between study groups with respect to motor nerve conduction velocity or tremor frequency spectra of physiological tremors and the correlation between the results of neurological tests was not significant [20].

Some of the study outcomes support the findings concerning the alterations of neuroendocrine secretion and motor coordination at very low occupational exposure levels of inorganic mercury, below the current biological exposure index [21]. They also indicate that abnormal tremors and other neurological effects may even occur in lower urinary mercury concentrations [22]. Nevertheless, other studies have disclosed that exposure to high concentrations of mercury may produce fatalities and devastating neurological damage among adult survivors [23]. The comparison studies have revealed several qualitative and quantitative similarities in neuropathological effects of mercury in humans and animals at high levels of exposure, neuropathological effects at lower levels of exposure were observed only in animals, and specific neurobehavioral end-points affected across species were similar at high levels of exposure [24]. Mercury can affect the performance of subjects exposed to its higher levels [25]. Numerous occupational exposure studies have indicated that workers with urinary mercury concentrations > 0.05 mg/L exhibited neurotoxic effects, such as decreased performance on verbal concept formation and memory tests [26]. Our study indicated that despite the fact that the workers of the control and mercury absorption groups showed some incidence of neurological symptoms, the high UMC of ≥ 0.05 mg/L was able to induce qualitative harmful changes. Exposure to high mercury concentrations could cause adverse effects of greater significance than those induced by exposure to lower concentrations. The authors suggest that different sensibility to mercury, physical conditions and age, malnutrition and strenuous
work could be also regarded as factors determining the quality and severity of neurological symptoms in the study population of workers.

CONCLUSIONS

Exposure to higher concentrations of mercury can bring about quantitative and qualitative neurological changes and oral disorders, resulting mostly in neurasthenia, emotional changes, tremors and oral and/or gum inflammations. The risk of neurological symptoms among the workers due to higher concentrations of mercury in the indoor air was not trivial. The risk estimation indicated that the workers in the study area were put at an excessive health risk owing to occupational exposure to higher concentrations of mercury. It is recommended to improve production technologies, working conditions, personal protection, and workplace ventilation. There is also a need to ensure the work environment monitoring, medical surveillance and educational programs. Further studies of health effects and their assessment as well as health promotion programs and a comprehensive approach to the reduction of occupational exposure to mercury are under way.

ACKNOWLEDGEMENTS

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REFERENCES


ERRATA

The date of receiving the paper „De minimus no curat lex – virtual thresholds for cancer initiation by tobacco specific nitrosamines – prospects for harm reduction by smokeless tobacco” by R. Nilsson, published in Vol. 19, No. 1, 2006, has been mistaken. The correct one is November 1, 2005; the paper was accepted for publication on 23 February 2006.