Long-Term Effects of Mercury on Subclinical Kidney Function

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Abstract: The kidney is considered to be one of the main target organs in the case of occupational exposure to elemental mercury (Hg0). The long-term effects of mercury toxicity on the kidney are not well-studied. Some authors associate proteinuria with a present increased absorption of mercury[1, 2, 3]. However, another group of researchers propose that proteinuria may also be associated with a long-term exposure to Hg0[4, 5, 6]. A group of authors suggest that a slight subclinical glomerular dysfunction may be present in some exposed workers[1, 3, 4], while others detect only slight renal tubular effects[2, 5, 7, 8]. Another group of researchers again suggests that exposure to Hg0 may cause both a glomerular and tubular dysfunction[6].

High mercury (Hg) retention and accumulation have been found in the kidneys of exminers from the Idrija Mercury Mine even several years after exposure, which could be associated with possible adverse effects on the kidney function.

The objective of this study was to investigate if a long-term past occupational exposure to Hg0 damages the kidney function.

The population studied comprised 53 mercury miners from the Idrija Mercury Mine and 53 unexposed workers as the control group. The study group of miners included 33 active miners and 20 retired miners. The miners were examined in the post-exposure period and were selected according to the following criteria: at least one-year exposure to Hg0, no history of occupational exposure to lead, cadmium, or other nephrotoxic substances, no history of renal disease, diabetes mellitus or multisystemic diseases, no evidence of haematuria, pyuria, glycosuria, infections or neoplasia, and no consumption of analgesics or antibiotics for two weeks before the examination.

The controls had no history of occupational exposure to mercury, lead or cadmium and came from the places with no sources of mercury pollution in the environment.

On the basis of exposure records, the following environmental indicators of past exposure were calculated for each miner: 1) years of exposure, 2) cycles of exposure, 3) Annual Time-Weighted Exposure (ATWE) and 4) Integrated Exposure Intensity (IEI). The concentrations of mercury in urine determined during the biological monitoring of each exposed miner were used to calculate: 1) the average urine mercury concentration of the entire period of exposure and 2) the sum of the peak urine mercury concentrations.

The glomerular kidney function was determined by a quantitative analysis of albumin and immunoglobulin G (IgG) in urine. The tubular kidney function was assessed by a quantitative analysis of a1-microglobulin in urine and by the enzymatic activity of N-acetyl-b-D-glucosaminidase (NAG) and its isoenzymes. The protein concentrations in urine were expressed per gram of creatinine.

To compare the differences in the indicators of the kidney function between the exposed and unexposed groups the t-test was used.

All subjects in the study were men. The mean age of the miners was 47 and that of the controls was 44. No significant differences in cigarette and alcohol consumption were found between the miners and the controls.

The miners were examined in the post-exposure period. The miners were not exposed to Hg0 in the previous 8 to 341 months prior to the present observation. The mean exposure time in miners was 15 years. The total number of cycles of exposure varied from 13 to 119. During the cycles of exposure ATWE varied from 0.13 to 0.47 mg/m^3 and IEI ranged from 105 to 10907mg/m^3-hours.

The average urine mercury concentrations of the past exposure in miners ranged from 26 to 158 mg/L and the sum of the peak urine mercury concentrations of exposed miners varied from 794 to 11365 mg/L.

Albumin, IgG and a1-microglobulin in urine were significantly elevated in the exposed miners ($t=2.17$, $p=0.03$; $t=2.81$, $p=0.00$; $t=2.07$, $p=0.04$) compared to the unexposed controls, which might point to a slight glomerular and tubular dysfunction. No significant differences were found in urine NAG and isoenzyme B activity when comparing the exposed miners and the unexposed workers.

The indicators of glomerular function (albumin, IgG in urine) and the indicator of tubular function (a1-microglobulin in urine) were significantly elevated in the exposed miners compared to the unexposed controls, which might point to a slight glomerular and tubular dysfunction in the exposed miners

No significant differences were found in urine NAG (U-NAG) and its isoenzyme activity when comparing the exposed miners and the unexposed workers. This may indicate that NAG could not be considered as an indicator of renal function in long-term exposure to Hg0 and/or that the changes in renal tubules might be reversible after some period of time.

The results of the preliminary study suggest that a long-term occupational exposure to Hg0 could damage the kidney function, which could be considered as a long-term effect of mercury.

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