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Effect of industrial cadmium contamination on parathyroid hormone secretion; a new look at an old problem

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Implication for health policy/practice/research/medical education

Cadmium is a toxic heavy metal, which, if inhaled or ingested, can damage various body systems and organs. Regarding its renal handling, most of its transport occurs in the proximal kidney tubules. High-level contact with this metal is associated with significant renal and bone impairment. The hazardous consequences of cadmium exposure on parathyroid gland structure and function are controversial and need further investigation.

Keywords: Cadmium, Parathyroid hormone, Heavy metal, Parathyroid gland, Kidney, Parathormone, Cadmium toxicity

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Cadmium is a toxic substance and is a heavy metal, which can affect various body systems and organs (1). Exposure to this substance is from polluted water, air and soil, which is a worldwide dilemma due to rapid industrialization (2). Other forms of exposure to cadmium include food contamination, like cereals and root vegetables, and smoking through high cadmium quantity in tobacco. This metal has harmful effect on several body systems and organs (3,4). The hazardous consequences of cadmium toxicity on parathyroid gland have been reported extensively; however, the results are contradictory (2). Previous studies showed that high-level contact with this metal is accompanied by significant renal and bone impairment (5). Regarding the renal handling of cadmium, most of its transport occurs in the proximal renal tubules; however, some data suggest that distal renal tubules may also have a role in the transport of this metal (6). Along with renal injury, chronic exposure to this metal has been related to osteotoxicity in some studies. This condition is characterized by diminished bone mineral density and enhanced risk of osteoporosis and also fractures (7). Recent studies showed that cadmium toxicity could occur in the absence of renal tubular dysfunction (4). The association of kidney damage and osteomalacia (itai-itai disease) was first described in Japan (in the late 1940s). The cadmium-induced renal toxicity and its association with bony effects of this metal were first

reported in 1950 (8). Nogawa et al showed that chronic cadmium toxicity may be associated with increased parathormone secretion (9). Following the analysis of a large sample of 9400 individuals (with age ≥ 18 years), Sang et al found that cadmium was considerably and negatively associated with serum parathormone when taken as a continuous variable. The data for study was derived from a survey of the general population in the US National Health and Nutrition Examination (2003-2004 to 2005-2006). However, the correlation among cadmium and parathormone was not meaningful in females. Sang et al showed that cadmium was negatively correlated with parathormone (2). To examine the possible harmful consequences of occupational cadmium contamination on parathyroid gland, Ibrahim et al studied urine and serum cadmium concentrations, renal function, serum parathyroid hormone along with some minerals of a group of individuals. This study showed that the contaminated workers had markedly higher cadmium concentrations in urine and serum than control subjects. The mean value of serum parathormone, magnesium and phosphorus were considerably lower in the exposed cases. They also showed a noteworthy positive correlation between cadmium value in urine and serum in the exposed patients. Ibrahim et al also found that the biological cadmium contamination indices correlated negatively with blood parathormone concentration, while, they correlated positively with serum

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calcium. They finally concluded that industrial cadmium contamination has an adverse effect on parathormone concentration (6). These studies show the significance of cadmium exposure on parathyroid gland structure and function. In view of the contradictory data on the exact effect of cadmium exposure on parathormone secretion, further investigations on this subject are necessary to define any link between cadmium toxicity of kidney and parathyroid gland.

Authors' Contribution

Conceptualization, validation: LM; Research, data curation, writing—original draft preparation, writing—reviewing and editing, visualization, supervision: MM & LM.

Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

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