

MEDICAL INFORMATION:

Itai-itai: Cadmium Exposure

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In 1946, there was an outbreak of cadmium poisoning in Jintzu, Japan. The source was contaminated water from a zinc mine. The outbreak was characterized by bone pain and bone fractures. Due to the pain, especially when walking, the disease became to be known in Japan as itai-itai disease, which means ouch-ouch. In this article, I will focus on medical surveillance for cadmium exposure. I will not discuss the diagnosis and treatment of acute or chronic cadmium poisoning, except for a few tidbits of information that you might find interesting. The primary sources of cadmium are the combustion of fossil fuels and the smelting operations of zinc, copper, and lead ores. Occupational exposure comes most commonly by way of soldering, smelting, brazing, electroplating, and the manufacture and disposal of nickel-cadmium batteries. (Recycle your batteries or at least dispose of them properly!) Cadmium exposure can also occur in your home. Because cadmium stearate is used as a stabilizer in plastics, burning plastic in your wood stove can result in accidental exposure.

Inhalation is the primary route of exposure. However, ingestion may also lead to toxicity. Cadmium exposure, in the general population, comes primarily from smoking cigarettes and eating certain foods. Cadmium is readily absorbed into plants: grains, cereals, and leafy vegetables, and especially tobacco leaf.

If we encounter any of the listed symptoms of cadmium poisoning, then we have failed in our job to protect

the worker. We need to develop a medical surveillance program to identify and rectify exposure hazards before toxicity occurs. The medical surveillance program for workers with potential exposure to cadmium has three goals:

- 1) to detect excessive exposure to cadmium before any biologic effects occur;
- 2) to detect any early biologic effects of cadmium;
- 3) to detect any disease or disorder that would place the worker at increased risk of material impairment of health if exposed to cadmium.

Detection of Excessive Exposure

Monitoring for excessive exposure to cadmium before the development of biologic effects involves assessment of urine and blood cadmium levels. It is exceedingly unusual to find nonoccupationally exposed adults with urine cadmium levels greater than 2 µg Cd/g creatinine. Any level above 3 µg Cd/g creatinine could be considered excessive exposure. OSHA's cadmium standard (29 CFR 1910.1027) specifies that if the urine cadmium is at or below 3 µg Cd/g creatinine and the blood cadmium is at or below 5 µg/L and the Beta 2 microglobulin is at or below 300 µg/g creatinine, then a repeat evaluation needs to be completed within one year. The cadmium standard requires shorter intervals for retesting as these three measured levels increase. (The cadmium standard calls for medical removal when the urine cadmium is greater than 15 µg/g creatinine, or if the blood cadmium is greater than 15 µg/L, or if the Beta 2 microglobulin is greater than 1500 µg/g creatinine.)

There is good evidence that a significant risk of renal injury exists for urine cadmium levels above 10 µg/g creatinine. In my opinion, a worker should not be allowed to reach a urine cadmium level ≥ 5 µg/g creatinine without a thorough evaluation of his/her worksite environment, work practices (smoking, eating, hygiene), and personal protective equipment. Elevated blood cadmium levels generally reflect acute exposure, while urine cadmium levels reflect chronic exposure. It is thought that a blood level of cadmium less than 5 µg/L is a no-effect level. I recommend that whenever the blood level reaches 5 µg/L, a thorough workplace evaluation, as discussed above, be undertaken.

The effects of exposure to cadmium may be either acute or chronic:

Acute effects

Fever, chills, and shortness of breath (metal fume fever)
Chemical pneumonitis
Renal failure
Gastrointestinal disturbances

Chronic effects

Proteinuria
Fanconi's syndrome
Osteomalacia
Emphysema
Anemia
Anosmia
Lung cancer
Prostate cancer

IMPORTANT

The collection of Beta 2 microglobulin needs to be a second voided specimen because the Beta 2 microglobulin is affected by remaining in the bladder for any length of time.

Follow the correct collecting procedure:

- When the worker arrives for testing, have him or her void and keep that specimen for urinalysis and determination of creatinine level in the urine.
 - Just before the worker leaves, obtain a second urine specimen. Submit this second sample for Beta 2 microglobulin determination. Keep it on ice if possible.
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Detection of Early Biologic Effects

The earliest renal effects of excessive cadmium exposure may be detected through screening with standard urinalysis (dipstick analysis may be used) and assessment of Beta 2 microglobulin levels. A Beta 2 microglobulin level in excess of 300 µg/g creatinine may indicate renal dysfunction resulting from cadmium exposure. Additional evaluation would be indicated, and it would be prudent, although not required by the standard, to remove the individual from exposure at greater than the action level (2.5 µg/m³ of cadmium in the air over an eight hour period) would require.

Early pulmonary effects are best assessed by periodic spirometry and chest radiography. The medical evaluation should be attentive to any pulmonary symptoms. Spirometry should be completed on an annual basis, and it is recommended that a chest x-ray be obtained every two years for workers with potential cadmium exposure. A decrease in pulmonary function greater than 5%, as measured by spirometry, or any chest x-ray abnormality, requires further evaluation and may justify removing the employee from cadmium exposure.

We know that cadmium has effects on the prostate gland. We do not, as yet, have all the information we need to definitively state that elevation of prostate-specific antigen is an early indicator of excessive cadmium exposure. However, it would not be unjustified to monitor PSA.

Excessive cadmium may affect bones. However, determination of bone density or assessment of calcium and phosphorus levels would not be appropriate tools for early detection of biologic effects.

The two main sites of cadmium deposition are the kidneys and the liver. One would not anticipate that abnormalities of liver function would be early indicators of excessive cadmium exposure.

Likewise, there are no cardiovascular findings that would indicate early biologic effects of cadmium exposure. Although cadmium binds significantly to red blood cells, there are no early effects on blood or blood formation that would serve as an indicator of excessive exposure. Anemia resulting from cadmium exposure would be a later finding.

Increased Risk Posed by Cadmium

Individuals with a history of significant pulmonary disease may be at increased risk from exposure to cadmium. This consideration would include individuals with asthma, because of cadmium's very irritative effect on the pulmonary system. Clearly, individuals with chronic obstructive lung disease, emphysema, pulmonary fibrosis, or other significantly compromising pulmonary diseases are considered at increased risk. Significant renal disease would also exclude an individual from work in an environment where there was potential exposure to cadmium. Individuals with low calcium, low protein, low iron reserves, or anemia are at increased risk because, with these conditions, cadmium is absorbed more efficiently. Such individuals stand at greater risk of developing toxicity. As an aside, it may be that the increased incidence of itai-itai in women is explained by the higher absorption rate of cadmium in females, caused by a higher incidence of iron deficiency. "Ouch-ouch!" I hear you saying. Enough information for one sitting!

For Further Information

Visit OSHA to learn more about the applicable standards and definitions: http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=STANDARDS&p_id=10035. ♣