CADMIUM INTOXICATION

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Abstract: *The author presents a few particularities on the cadmium intoxication in children: cadmium pollution sources, toxicokinetics and toxicodinamics of cadmium intoxication, the physiopathology, the clinical aspects and the treatment of cadmium intoxication.*
Keywords: *child, cadmium intoxication.*
Rezumat: *Autorul prezintă câteva aspecte legate de intoxicaţia cu cadmiu la copil: sursele de cadmiu cu potenţial de poluare, toxicocinetica si toxicodinamica cadmiului, fiziopatologia intoxicaţiei, tabloul clinic şi tratamentul intoxicaţiei cu cadmiu.*
Cuvinte cheie: *copil, intoxicaţia cu cadmiu.*

Cadmium is a metal which enters into the composition of a large number of industrial compounds (cadmium acetate, flourborate, carbonate, nitrate, oxide, stearate, sulphate and sulphide). The environment cadmium contamination caused in Japan a disease, called the „Itai-Itai" disease, which manifested by severe arthralgia and osteomalacia in people having reduced quantity of calcium and vitamin D.

The cadmium sources (3) came mainly from the human activities:

- Contaminated food (rice), contaminated soil or contaminated cigarettes;
- Oil products combustion (oil distillery);
- The fabrication process of nickel-cadmium – based batteries;
- Pigments used in the fabrication of plastic, ceramic or glass materials;
- The fabrication of certain welding metals and of alloys (3).

Toxicokinetics and toxicodinamics. Cadmium (metal or under the form of salts) has a reduced volatility and may be found in the air under the shape of fine particles. The absorption takes place mainly at the level of pulmonary alveoli, 25% of the inhaled cadmium being absorbed. The absorption from cigarettes seems to be larger, taking into account the more reduced sizes of the cadmium particles which may be found in cadmium, favouring their "penetration" up to the alveolus level. Only 5% of the ingested cadmium is absorbed, its absorption being favoured by the iron or calcium deficit or by fat-rich diets. Cadmium is not transdermally absorbed.

Once absorbed, cadmium is distributed in the entire body, with prevalence at the liver and kidney level. Cadmium may pass into the mother milk during the nursery period of time and may traverse the placenta (1,2), exposing the foetus and the baby to cadmium risk intoxication. The plasmatic cadmium is related to albumin and to a protein called metallothionein. The largest part of cadmium is released by faeces, but it can also be release (in small quantity) by urine. It is still unknown if, within the organism, cadmium is metabolized by alkylation, oxidation or reduction. Once deposited in the kidneys, the cadmium halving time is of 6-38 years and at the liver level, of 4-19 years.

It was proved that after one single exposure to a non lethal large quantity of cadmium, the pulmonary function may be affected for years. The medical literature described the case of a worker who exposed himself to cadmium for one hour when welding, by using cadmium-containing electrodes, his pulmonary function being affected for more than 4 years.

Death may occur either after chronic exposures to cadmium or in more reduced quantities. Of the mediatized cases, we mention the case of a woman working in a melting factory in Japan, Takako Nakamura, who committed suicide in 1969 as a result of the increased abdominal pain. Although the cadmium toxicity is validated taking into account a concentration of 1 ppm (part per million), the women's autopsy showed cadmium concentrations of about 4.540 ppm in kidneys and of 22.400 ppm in liver (3).

Physiopathology. The kidney represents the main target organ for the chronic exposure. The proximal tubular disorder characterised by proteinuria, aminoaciduria and glycosuria is gradually installed and the renal insufficiency is rare. The acute exposure leads to the pulmonary edema and in case of survival, the pulmonary fibrosis is installing in time. Regarding the possible secondary events, we mention the pulmonary emphysema and the chronic bronchitis (3).

Clinical manifestations. Renal effects. The tubular proteinuria and the reduction of the glomerular filtration were repeatedly proved in the populations chronically exposed to cadmium in Japan, Belgium and China.

Regarding the renal effects, mention must be made of the tubular nephropathies manifesting by proteinuria, aminoaciduria, glycosuria, phosphaturia and important losses of calcium. The chronic sequels are characterised by the glomerular filtration reduction and the renal lithiasis

The effects of the respiratory system. The cadmium particles are intensely irritating on the respiratory tract, yet the symptoms occurrence may be late. The respiratory symptomathology after the inhalation is characterised by fever, headaches, dyspnea, thoracic pain of pleural cause, rhinitis, conjunctival congestion, dysphagia and cough, occurring 4-12 hours after the exposure.

The noncardiogenic pulmonary edema may progress and may lead to death; the respiratory insufficiency is developed 3-10 days after the exposure. There has not been reported the dose of cadmium which triggers the respiratory effects. The chronic exposure induces the pulmonary emphysema.

Other effects. The ingestion of increased quantities of cadmium induces the irritation of the gastro-intestinal epithelium with gastro-enterocolitis clinically validated by nausea, vomiting, abdominal pain, salivation; the hepatic affection was also described.

The cardio-vascular system seems not to be affected by the cadmium exposure.

The bone system is also affected, characterised by bone pains, osteomalacia and osteoporosis, evolving even to sudden fractures (the Itai-Itai disease). Within the region of the Jinsu River basin of Japan, the osteomalacia and the Itai-Itai disease were frequently associated with the malnutrition and a severe calcium deficit (4). It seems that the effects on the bone system are secondary to the alteration of the vitamin D, calcium and phosphorous metabolism. The cadmium exposure was also associated with the urinary lithiasis and the yellow colouring of teeth.

The neurological effects consisted in peripheral neuropathy (with the decrease of the nervous conducting speed), the alteration of the equilibrium and disabilities in the concentration process (6).

Diagnosis. The seric level of cadmium in the exposed workers is between 10-100 mg/l. The levels over 0,7 μg/dl indicate a serious exposure. The evaluation of the seric level of cadmium may be used for the recent exposure evaluation. The tubular disorder determined by cadmium is irreversible and may be evaluated by the α_1 dosing of the urinary macroglobulin. If the urinary excretion of cadmium is under the value of 2 mg/day, the risk of the renal affection remains reduced.

The most accurate procedure of assessing the cadmium intoxication remains the spectroscopy by the atomic absorption and the spectroscopy of atomic emission.

Treatment. The initial care of a case exposed to cadmium supposes the monitoring of the respiratory sufferance, the assisted ventilation, if it is necessary and the oxygen administration.

In case of pulmonary edema, the case will be treated with diuretics, mechanical ventilation and corticosteroids. The monitoring of the ingesta-excreta balance is required, having in view the possible alteration of the renal function (5). In case of ingesting large quantities of cadmium, taking into account the fact that the solutions may be caustic, it is not recommended the gastric lavage; the dilution with water or milk of the ingested toxic element could be benefic, although this effect has not been proved until present; the use of the activated carbon did not proved its efficiency.

In case of ocular or skin exposure to the basic cadmium compounds, it is recommended to remove the clothes, to wash the teguments and the eyes.

In case of the exposure of the respiratory apparatus, the respiratory symptomathology occurs in a few hours and it is recommended to remove the victim from the polluted environment, also the sustaining therapy of the respiratory apparatus.

The evolutive particularities in cadmium intoxication. Certain recent-made studies **(**1,3**)** consider that cadmium stimulates the RNA synthesis and the cell proliferation. It seems that this metal stimulates the expression of certain genes classes (including the proto-oncongene) and interferes with the repair processes of RNA. Within this context, the „Agency for Research on Cancer working group" defines cadmium as "a probable human carcinogen", although the research made until present (England, Japan, Belgium) have not proved the increase of the cancer incidence in the populations exposed to large concentrations of cadmium.

Antidotes. Although a series of substances has been tested, their efficiency in case of overdose of cadmium remains reduced.

After the intoxication by ingestion, the succimer may stimulate the cadmium excretion. The dimecarpol increases the renal toxicity and it is not recommended. Certain studies consider that the N-acetylcysteine may reduce nephrotoxicity in the population exposed to cadmium acutely or chronically (at present, there are studies in progress regarding this observation).

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