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SHORT COMMUNICATION

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Lethal manganese-cadmium intoxication. A case report

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Abstract A case of a lethal manganese-cadmium (Mn-Cd) intoxication is reported. The postmortem examination revealed a noticeable reddish-violet discolouration of the serous cutes of all body cavities, but there was no indication of any corrosive burns of the mucous membranes of the gastrointestinal tract. An Mn concentration of 899 μg/l blood and a Cd concentration of 238 μg/l blood were found in the deceased woman. These concentrations are higher than normal levels by a factor of about 100. A subacute or chronic manganese-cadmium absorption must be assumed.

Keywords Manganese · Cadmium · Lethal intoxication

Introduction

Manganese intoxications are described in literature almost exclusively as having been caused by acute poisoning with potassium permanganate ($KMnO_4$) or by acute and chronic poisoning with manganese dust (occupational diseases of persons employed in manganese ore mining).

Acute $KMnO_4$ intoxication with crystals or concentrated solutions of 5% and above has been found to cause corrosive burns to the oral cavity and gastrointestinal tract. Lethal intoxication after acute $KMnO_4$ absorption is reported for a few cases in the literature only (Grusz-Harday 1967; Justus and Gastmeier 1967; Middleton et al. 1990; Young et al. 1996; Ong et al. 1997).

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Two types of intoxication are caused in persons exposed to manganese dust (in industry): a chronic brain damage, i.e. ''manganese encephalitis'', and a rather acute ''manganese pneumonia''. Systemic intoxications with other manganese compounds are extremely rare due to the poor resorption rate (3–8% from the gastrointestinal tract) and can be expected to occur when taken parenterally only (Seeger 1990). These few cases of a parenteral resorption were mainly connected with hepatitis and pancreatitis (Lustig et al. 1982; Korc 1986).

Cadmium intoxication produces the following symptoms:

- The resorption of soluble cadmium compounds (Cd^{2+}) from the gastrointestinal tract is low as is the case with manganese ($< 10\%$). Cadmium compounds taken acutely through the mouth (threshold of effect approximately 15 mg absolute for adults) will cause severe gastrointestinal disorders such as vomiting and diarrhoea which are not life-threatening in general. The oral ingestion of 30–40 mg or more of soluble cadmium salts can lead to death (Estler 1992).
- Cadmium vapours and cadmium oxide, in particular, will more effectively be taken up through the lungs (resorption up to 30% of the quantity inhaled). After a latent period of about 24 h a pulmonary oedema and a pneumonia can develop.
- Chronic cadmium ingestion through the respiratory airways may cause degeneration of the mucous membranes of the nose and throat, i.e. ''cadmium headcold'', destruction of the olfactory epithelia, chronic bronchitis, progressive pulmonary fibrosis and pulmonary emphysema (Reichl 1997).
- The oral ingestion of cadmium-contaminated foodstuffs over a long period will cause severe osteomalaciae and osteoporoses with skeletal deformations and shrinkages of the body size, and iron deficiency anaemias (''Itai-Itai disease'').
- Cadmium is a typical cumulative toxicant which concentrates especially in the kidneys and liver. Cd^{2+} is characterised by a very high protein bond and is

discharged extremely slowly; the half-life is 10–30 years. With chronic cadmium intoxication, a yellow Cd seam can be found on the dental necks.

The report below relates to an extraordinary case of a combined manganese-cadmium intoxication.

Case report

Story

A woman, aged 35, was found lying dead in bed by her husband. She had been working as a biology teacher and had access to various chemicals. In the time before her death she had suffered from occasional convulsions. The victim had last eaten beetroot. The medical postmortem examination did not furnish any information about the cause and type of death. After the death had been reported to the police, a forensic autopsy was performed.

Postmortem findings

The serous cutes of all body cavities were found to show a noticeable reddish-violet discolouration. No indication of corrosive burns or necroses could be detected in the oral cavity or in the remaining gastrointestinal tract. Otherwise, there were distinctive signs of an acute cardiovascular collapse with hyperaemia of the organs of the minor and major circulation. Traces of injury were not found. Using routine haematoxylin and eosin staining, the only histopathological findings were a fatty liver and anthracosis of lung.

Toxicological investigation

Because of the noticeable violet discolouration (like $KMnO_4$), an investigation for Mn and other elements appeared to be appropriate in order to exclude an intoxication. The concentrations of Mn and Cd that were found in body fluids and organs are shown in Table 1.

A check was made to ensure that the sample vessels and chemicals used were not contaminated with manganese and cadmium.

The investigation for the elements mercury, arsenic, lead, chromium, thallium, zinc and iron yielded unremarkable results.

The investigations for pharmacons and ethanol did not reveal any positive findings.

Discussion

In the case described here, cadmium and manganese concentrations could be detected that were greatly above standard levels and definitely toxic.

Table 1. Manganese (Mn) and cadmium (Cd) concentrations in body fluids and organs

Material	Mn concentration	Cd concentration
Serum (heart blood)	127 μg/l[a]	not determined
Heart blood	899 μg/l[a]	238 μg/l[b]
Stomach contents	0.1 μg/g	0.4 μg/g
Brain	0.2 μg/g	3.6 μg/g
Liver	0.8 μg/g	20.2 μg/g
Kidney	3.6 μg/g	34.3 μg/g
Small bowel contents	1,266 μg/g	<0.1 μg/g

[a]Normal range: 0.3–1.1 μg/l serum, 6.0–11.0 μg/l blood
[b]Tolerance range: <2.0 μg/l blood

According to literature (Degner et al. 2000), the normal ranges for Mn are 0.3–1.1 μg/l in serum and 6.0–11.0 μg/l in blood. The deceased women was found to have a serum Mn concentration of 127 μg/l and a blood Mn concentration of 899 μg/l (Table 1). There is no information available on toxic or comatose/lethal concentration ranges.

Cadmium concentrations above 20 μg/l are considered as toxic. There is also no information available on comatose/lethal concentration ranges for Cd.

The noticeable reddish-violet discolouration of the serous cutes of the body cavities is most probably due to the consumption of the beetroot which the deceased woman had eaten in the evening of the day before her death. Such a violet discolouration of the total peritoneum is reported to have occurred after the consumption of "beetroot juice" (Klug and Maxeiner 1981).

Manganese

Concentrations as high as those found in the case described here are not to be expected in an acute intoxication with manganese salts because of the poor resorption rate of 3–8% only. In persons who had survived a subacute $KMnO_4$ intoxication, Degner et al. (2000) detected a manganese concentration of 150 μg/l whole blood only and Grusz-Harday (1967) found less than 250 μg/l whole blood. On the grounds of the very high manganese concentration in the blood and serum and the exclusion of necroses of the mucous membranes of the gastrointestinal tract concerned (Justus and Gastmeier 1967; Lustig et al. 1982; Holzgraefe and Poser 1986), it is not an acute but more probably a subacute or chronic intoxication that can be assumed in our case.

Due to the very long half-life of Mn in the human body, it will accumulate when taken repeatedly. The repeated ingestion of smaller doses over a longer period may easily lead to intoxication (Young et al. 1996; Florence and Stauber 1988; Degner et al. 2000). Degner et al. (2000) report a subacute intoxication that was survived by a man who had erroneously been drinking a few millilitres of an 8% $KMnO_4$ solution over a period of at least 4 weeks. The patient suffered from heavy abdominal pain and, after a period free of symptoms, from a gastric ulcer, loss of hair, impaired sight and psychiatric symptoms with reduced power of concentration, poor urge, anxiety attacks and tiredness as well as symptoms of the type developed in Parkinson's disease.

The case discussed here may also be due to a subacute $KMnO_4$ intoxication in which the $KMnO_4$ concentrations are too low to cause a corrosive burn. With a certain latency provided, parkinsonian symptoms are reported to occur (Degner et al. 2000), which however were not observed in the present case. The period between the intoxication and the occurrence of death might have been too short for these parkinsonian symptoms of chronic manganese intoxication to develop.

Seeger (1990) describes two types of the (industrial) manganese dust poisoning: chronic brain damage, i.e. manganese encephalitis, and the rather more acute manganese pneumonia of persons working in ore mines and having inhaled dusts containing manganese. This industrial intoxication can be excluded in the present case because the deceased woman was, according to police reports, neither confronted with manganese dust over a long period of time nor were pathological changes detected in her lung.

Cadmium

Compared with normal levels, the Cd blood concentrations are increased by a factor of 100 and the organ concentrations by 10- to 20-fold in the case investigated in our institute. With acute oral intoxications, higher blood, liver and kidney concentrations were reported in literature than were seen the present case, whereas a higher Cd concentration in the lung was mentioned for acute inhalative intoxication with Cd levels in the liver and kidney being in the normal range (Baselt 1982; Merian 1984). For the case discussed, there is no figure available for the concentration in the lung but the histological examination of the lung did not reveal any noticeable pathological findings.

Considering the Cd concentrations determined alone, a chronic Cd intoxication had to be assumed with the highest degree of probability. But since the histological examination of the kidney did not yield any pathological changes such as fibrosis of kidney, it would be also probable here – as was the case with the manganese – that the patient had suffered from a subacute intoxication.

Cadmium and cadmium compounds are hardly accessible to the public. Cadmium is used at a rate of 60% for making alloys resistant to corrosion. The highest intoxication hazard exists from Cd (oxide) vapours in industrial metal working. Further applications of cadmium are in the manufacture of dry batteries, cathode-ray tubes and colouring pigments. In the 1920s tableware and other goods made of metal were coated with cadmium for protection against corrosion. Since that time some intoxications became known, which were caused by acids contained in foodstuffs (e.g. citric, tartaric, acetic acids) dissolving the cadmium into food kept in such containers. The cadmium-plating of water and food containers has been prohibited since these intoxications became recognised.

For the case of the lethal intoxication of a female biology teacher aged 35, the police have been so far unable to find out whether it was a suicidal or accidental intoxication; a letter of farewell was not found.

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