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OBSERVATION

A CASE REPORT OF ACUTE HUMAN MOLYBDENUM TOXICITY FROM A DIETARY MOLYBDENUM SUPPLEMENT – A NEW MEMBER OF THE »*LUCOR METALLICUM*« FAMILY

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The paper gives a brief review of human molybdenum metabolism and toxicity and presents the first known case of acute clinical poisoning with molybdenum from the dietary molybdenum (Mo) supplement in a male patient in late thirties. In over 18 days, the patient had consumed a cumulative dose of 13.5 mg Mo (300-800 µg Mo/day). Followed the development of acute psychosis with visual and auditory hallucinations, a series of *petit mal* seizures, and one life threatening *grand mal* attack. The symptoms remitted several hours after the start of chelation therapy with calcium ethylene diamine tetraacetic acid (CaEDTA). A battery of neuropsychological tests and Spectral Emission Computer Tomography demonstrated evident frontal cortical damage of the brain. One year after the Mo poisoning, the patient was diagnosed toxic encephalopathy with executive deficiencies, learning disability, major depression, and post-traumatic stress disorder. The paper strongly advocates issuance of and strict adherence to written warnings on the instruction labels not to mix potentially harmful neurotoxic substances, such as molybdenum, with other nutriceuticals and to instructions stating maximal single and cumulative doses. Molybdenum is a new and unwelcome member of the »metal madness« family.

Key words:
acute psychosis, brain SPECT images, CaEDTA chelation therapy, dietary supplement, neuropsycholo-gical tests, neurotoxic substance

A cry for help was blinking on my computer screen. A young woman from California, USA, besought the whole world via Internet for anybody who knew anything about molybdenum toxicity. This was a challenge a red blooded international trace element expert in nutrition, toxicology, and radiotoxicology in man and animals simply could not resist. This paper is the result of that response to a challenge.

What do we know about acute molybdenum toxicity in man? Not much, by the look at the existing literature (1). Molybdenum (Mo) is a transitional metal that forms oxides and is a component of a pterin coenzyme essential for the activity of xantine oxidase, sulphite oxidase, and aldehyde oxidase. In other words, it is an essential trace element (2). Genetically conditioned sulphite oxidase deficiency was described in 1967 in a mentally retarded child with convulsions, opisthotonus, and lens dislocation. The disorder was due to the child's inability to form a molybdenum coenzyme despite adequate dietary molybdenum content (3, 4).

Sulphite toxicity due to molybdenum deficiency was noted in a patient on long-term total parenteral nutrition who developed tachycardia, tachypnea, headache, nausea, vomiting, and a coma. A metabolic study showed high levels of sulphite and xantine, and low levels of sulphate and uric acid in his blood and urine. Intravenous administration of ammonium molybdate 300 μg/day led to a dramatic recovery. Both genetically conditioned and nutritional deficiencies of molybdenum are rare. The intake of molybdenum varies from 100 to 500 μg/day. Principal sources are meat substitutes, whole-grain cereals, and legumes. The Food and Nutrition Board of the NAS/NRC states that a safe adequate intake of molybdenum is 75 to 350 μg/day for adults and 25 to 75 μg/day for children aged 1–6 years (4). Molybdenum has been detected in only about 25% of blood samples of the human urban population (5).

Little information is available on molybdenum metabolism in humans; data for intestinal absorption ranging from 50–70% during long term balance studies were reported by *Tipton and co-workers* (6) and *Robinson and co-workers* (7). In a recent work, *Turnlund and co-workers* (8) and *Cantone and co-workers* (9) report intestinal absorption values around 90% and 85%, respectively. In humans molybdenum is contained principally in the liver, kidneys, fat, and blood. Of the approximate body total of 9 mg of molybdenum, most is concentrated in the liver, kidneys, adrenals, and omentum (10). More than 50% of molybdenum in the liver is in a nonprotein cofactor bound to the mitochondrial outer membrane and can be transferred to an apoenzyme transforming it into an active enzyme molecule (11). More than half of the excreted molybdenum via urine. Molybdenum is considered to be rapidly excreted, mainly as molybdate. An excess may also be excreted through the bile, particularly the hexavalent forms. Experimental studies showed that injected radio-molybdenum increased the liver and kidney levels, and that the endocrine glands and the brain had an exceptionally high molybdenum content (12).

The fumes of molybdenum, used as an additive to some welding rods or to steel, produce a unique clinical picture. Symptoms of chronic molybdenum exposure are decreased appetite, listlessness, weakness, fatigue, anorexia, headache, arthralgia, myalgia, chest pain, nonproductive cough, and diarrhoea. Molybdenum increases production of xantine oxidase and is a cofactor required for transferases to bind iron. Therefore, patients with high molybdenum levels develop gouty attacks and a hypochromic microcytic anaemia. The patients may develop an X-ray pattern that looks like a pneumoconiosis. Chronic molybdenism causes testicular atrophy (13).

Copper prevents accumulation of molybdenum in the liver and may antagonise the absorption of molybdenum from food (14). The antagonism of copper depends on the dietary sulphate (15). It has been suggested that copper and sulphate may displace molybdate in the body (16, 17). Instead of deficiency, diethyldithiocarbamate (DEDTC), a chelating agent used for treatment of copper poisoning in sheep, was found to provoke an excess of copper (Cu) liberated from the lipophilic complex Cu-

DEDTC in the brain (18). The finding was confirmed by *Haywood and co-workers* (12) who also demonstrated molybdenum accumulation in the brain and endocrine glands of DEDTC-treated sheep. The toxic effect of molybdenum lies in the inhibition of the activity of the sulphide oxidase system and in the formation of thiomolybdate from molybdate and endogenous sulphides. Sulphate ions activate the enzymic system oxidising sulphide and thiomolybdate, restraining thus the formation of thiomolybdate and accelerating the oxidation of thiomolybdate into the less toxic molybdate (19).

THE CASE

Acute molybdenum toxicity in a human subject in self-imposed molybdenum exposure

A male health practitioner in his late thirties (the patient), apparently in full health, wanted to be even the healthier. His colleague suggested that he should try a molybdenum supplement (Molybdenum Chelated, 100 μg molybdenum per tbl., Nutriwest Co., Douglas, WY) to cure his »allergy« to perfume of his patients. That far he had had a remarkably empty medical record and was outstanding in his educational curriculum. According to his wife's testimony, he was »*easy going, doesn't get angry often, and it takes a lot to rattle him*«. The patient consumed a HCl supplement for »indigestion«, and other non-noxious nutriceuticals (Table 1) before he started to take molybdenum on 1 July 1997. The manufacturer's recommendation read: »*One tablet per day, or as directed*«. Instead, the patient followed the recommendation from his physician »*Take as needed*« and consumed an average of 7 to 8 tablets of molybdenum per day (700–800 μg). He did not start with 700 μg; he started with about 300 μg a day, and then gradually increased the intake over a period of 18 days. On day 7 he showed the first signs of anxiety and agitation. On day 14 he became mildly psychotic and experienced visual and auditory hallucinations. He also exhibited excessive craving for salt; so much so that he woke his spouse up at 2:00 a.m. to get him salt from a grocery store. On day 18 he could »*smell the molybdenum all around him*« and he abandoned molybdenum supplements. The smell was like the one of salt water, distinctive but mild. By the time the patient stopped taking molybdenum, he had already consumed a total cumulative dose of 13.5 mg of molybdenum. On day 19 he had severe psychosis with strong audio and visual hallucinations, insomnia, intense craving for salt, diarrhoea, and painful and cold extremities. On day 22 his hallucinations were accompanied by *petit mal* seizures and on day 24 he tried to take his life with a knife and, »*chased by the devil*«, he ran through a plate glass window and jumped headlong off a six-metre wall. The flight ended up with multiple body contusions and difficulties in movement. He struggled with an officer when the police picked him up, as he thought that they were trying to hurt him.

Lucor molybdeni – acute molybdenum toxicity induces seizures and psychosis

The emergency room physicians first thought he was drugged with phencyclidine (PCP), then believed that he had an episode of acute maniac-depressive psychosis. The medical staff of the hospital thought that his spouse was in a state of denial

Table 1 *List of nutriceuticals* the patient had already been consuming by the time he started molybdenum supplementation*

Trade Name	Ingredients
Betafood	150 mg beet root powder 100 mg vacuum dried beet leaf and hoof juice
Protefood	Bone meal, defatted wheat germ, vacuum dried bovine adrenal, choline bitartarate, carrot powder, ribonucleic acid, vacuum dried veal bone meal, d-methionin, lysine monohydrochloride, glutamic acid, peanut bran, ascorbic acid
Min-Tran	Calcium lactate, kelp powder, magnesium citrate, alfalfa powder, calcium stearate
Inositol	405 mg Inositol in honey and calcium stearate
Zipan	180 mg Betaine Hydrochloride, 65 mg Pancreatin (3x), 35 mg Bovine Pancreas Cytosol Extract, 25 mg Pepsin (1:10,000), 10 mg Ammonium Chloride
Allorganic trace minerals- B_{12}	lactate, kelp powder, potassium paraaminobenzoate, honey, zinc-iron-copper liver chelate, vacuum dried alfalfa, magnesium citrate, vacuum dried buckwheat juice and seed, vacuum dried pea, vine juice, bone meal, bovine orchic glandular extract, copper liver chelate, defatted wheat germ, oat flour, calcium lactate, calcium stearate, carrot powder, vacuum dried veal bone meal, protamine iodine, peanut bran, cyancobalamin

*Standard Process Inc., Palmyra, WI, USA

about the patient's psychotic condition. There were no signs of skull fracture or haemorrhage on nuclear magnetic resonance image. Yet, the patient was mentally incapable of answering the most basic questions and some of his answers proved to be false on later examination. He was placed on heavy neuroleptics but continued to have fits, seizures, and hallucinations. The medical personnel in the local hospital did not realise that acute molybdenum toxicity could have been at the root of the acute psychic disorder. Having in mind the patient's dietary exposure to molybdenum, his wife insisted, unsuccessfully, that he should be treated for molybdenum poisoning. On 25 July and 16 August the patient's blood molybdenum was 7.7 ng/ml and 1.7 ng/ml, respectively (normal 0.9–1.8 ng/ml; Mayo Med. Lab., Rochester, MI). After the blood molybdenum analysis arrived, the patient was diagnosed toxic encephalopathy and was given multiple medication to control his bizarre behaviour and *petit mal* seizures. On 16 August he had a severe psychosis with auditory and visual hallucinations, *petit mal* seizures, and later on, a *grand mal* seizure for 20 minutes when already in a hospital. He went into a short coma after large medication for his *grand mal* seizure. The condition continued the following day and the day after that the patient was reduced to a wheelchair and could barely see.

Chelation, behavioural changes, and CNS sequelae

Only after the patient was chelated with CaEDTA on 22 August 1997 did his fits, seizures, and psychosis stop within two hours. Few days after his psychotic signs, fits and seizures came under control, he developed an attitude of »*he knows everything about everything*«. He wanted to grow long hair to look like Jesus; he felt it was all right to have affairs with many women; he felt the chelation would do him harm; and he became fixated on things. The patient was a kind and loving man before he took molybdenum supplements and now he was a complete stranger to his spouse. The patient underwent chelation therapy for a total of 10 cures. After the fourth chelation treatment this odd behaviour stopped and did not repeat. The first long-lasting sequela to molybdenum poisoning was an immediate, severe depression, totally non-responsive to Zoloft® and nortriptilyne. The patient's sex drive was nil and six month later it was found that his blood testosterone was low and his depression appeared somewhat responsive to testosterone patch treatment. A year later he had persistent nightmares, severe fatigue, non-existent sex drive and other sexual difficulties (could have erection but could not feel ejaculation), and depression which needed treatment. Even much later the patient was experiencing depression. Occasionally, his spouse would have to help him pick clothes, as the decision was too difficult for him. And, occasionally, he would do just fine.

Evident frontal cortical damage was confirmed by an extensive battery of neuropsychological tests and by the Spectral Emission Computer Tomographic Images (SPECT) (20). Milder impairment was registered in the descending degree in the parietal lobe, temporal lobe, and even basal ganglia. Prior to the poisoning, the patient excelled in schooling performance; he completed a seven-year college, never had to retake a class, and passed the State and National Boards exams straight away with high scores. Unfortunately, the current degree of his brain involvement is such that it is doubtful whether and when the patient would be able to resume his previously successful carrier. A year after the episode of acute molybdenum intoxication, the patient was diagnosed the following: toxic encephalopathy with executive system deficiencies, learning disability, major depression, and post-traumatic stress disorder. I would like to stress that the results of Motivation/Personality analysis exclude the possibility of the patient's possible malingering (»*These results demonstrate that the patient has no serious pre-existing psychopathology and answered the questions in a manner that would relate good effort*«).* He honestly gave his best, which is a fact of primary importance in any case possibly involving compensation.

DISCUSSION

Heavy metal damage to the brain is well documented for mercury, manganese, lead, and cadmium. Even the term *Lucor manganicum* (manganese madness) has been coined. This is the first case of acute human molybdenum poisoning which dem-

* Full medical reports can be obtained from the author of this paper upon request

onstrated that molybdenum is a new and unwelcome member of the *Lucor metallicum* family. Otherwise, molybdenum toxicity does not distinguish itself from other heavy metals (21) except, perhaps, in the specific impairment of bone collagen which resembles lathyrism (22).

The increased acidity promotes the absorption of molybdenum from the stomach into the bloodstream (23). Indeed, the fractional absorption of molybdenum from the gastrointestinal tract was almost 100% when administered in diluted HCl and 50% in infant formula (24). Blood so tainted with the heavy metal nourishes the brain (23). Assuming that molybdenum tissue distribution and excretion follows the first order kinetics and knowing that the biological half-life ($t/_2$) of molybdenum in plasma is about eight hours (1), then molybdenum in the patient's plasma at the time when it was first determined would represent the value of molybdenum after 18 biological half-lives. And if one extrapolated the actual molybdenum plasma level as it was at the moment of the patient's admission to the hospital, his estimate would say that the patient would have about 1 mg molybdenum/ml plasma. It is an unreally high figure because the patient's total consumption of molybdenum over the 18 days was 13.5 mg and the plasma volume measures in litres. Evidently, our knowledge about the relationship between the dose of molybdenum and its biological half-life in plasma is lacking and it may well be that the actual molybdenum $t/_2$ in plasma is longer when molybdenum dose is high. Certainly, the patient was consuming molybdenum at a rate faster than the shortest $t/_2$ of eight hours and progressively accumulated molybdenum in the blood. Indeed, the admini-stration of molybdenum to experimental animals rapidly increased plasma molybdenum (25). However, biological half-life of molybdenum in the body is much longer than that in the plasma. Ten days after intravenous administration of carrier free ^{99}Mo to human volunteers, about 25% was excreted via the urine and between 1% and 6% via faeces, indicating that 70% was retained and redistributed elsewhere in the body (22). Unlike the carrier free molybdenum isotope, stable isotope molybdenum in human plasma can be detected only if the levels of circulating molybdenum are 2.0 ng/ml or above, that is, above the normal range reported by the Mayo Clinics, Rochester, MI (24).

When molybdenum or some other heavy metal is deposited in the grey matter of the brain, it will inhibit the nerve transport, increase the breakdown of various neurotransmitters, and stimulate the production of harmful proteins (22). The consequences of molybdenum deposits can include seizures, decreased learning ability, impaired motor coordination, memory loss, and even psychotic reactions. That sequence of events might explain the persistence of psychiatric symptoms in the patient and the later development of brain sequelae at the time when his blood molybdenum was already normal as revealed by SPECT. The brain is rich in fat tissue and molybdenum has shown preference for depositing in the fat tissue (10). Molybdenum in plasma appears to reflect the »hide-and-seek« situation where molybdenum is rapidly disappearing from plasma and accumulating in the fat tissue of the brain, or elsewhere. Therefore, molybdenum plasma concentrations would not be a good indicator of molybdenum intoxication, not even shortly after exposure to high doses of that metal. It reminds me of a bitter dispute between, now both late, *Beritić* (26) and *Kehoe* (27), about whether it was possible to have clinical signs of lead colics with normal blood lead levels. Evidently, the actual dynamics of lead kinetics and the retention in plasma and in the body (28) is much slower than that of molybdenum, but apparently, it

gives more credit to the *Beritić's* view on the appearance of lead colics when the blood lead levels are already normal.

De Stasio and co-workers (29) found a much larger uptake of aluminium and molybdenum in neurone cultures than that of other elements, indicating higher neurone cell affinity for molybdenum. We too, found specific predilection for environmental radon daughters of the proteins and lipids in patients with the Alzheimer's Disease and Parkinson's Disease, respectively (30). It should be noted that with respect to molybdenum biokinetics, metabolic processes such as intestinal absorption and plasma clearance are individual characteristics which tend to remain stable for at least a few years (24). One of serious sequelae of molybdenum poisoning was the patient's lasting depression and the idea that impairment of molybdenum metabolism may be related to depression is supported by findings of higher than normal molybdenum values in the hair of the depressed. Apparently, depressed people accumulate molybdenum in the body by excreting less molybdenum via the urine (31). Acute molybdenum toxicity affects male gonads and leads to testicular atrophy (13) (correspondence Haywood – Shelton from 9 March 1999), as evidenced by the patient's low testosterone and the improvement of his depression after the testosterone patch therapy.

It was to be expected that, sooner or later, the appearance of a wide range of mineral supplements on the market – some of them with neurotoxic potential – would lead to tragic accidents like this one. Indeed, it is absolutely impossible to predict what »cocktail« a prospective consumer is able to make. Having in mind the fact that the complex dose-dependent interactions between mineral supplements and other »nutriceuticals« (32, 33) are largely unknown and poorly understood (34, 35), instruction labels will have to stress that a product is guaranteed only if not mixed with other supplements and will have to state single and cumulative maximum doses. Poison Control Centres should be aware of molybdenum as a new member of the »*Lucor metallicum*« family and physicians should be advised about the necessity of an urgent chelation treatment.

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Sažetak

KLINIČKI SLUČAJ AKUTNOG OTROVANJA MOLIBDENOM IZ PREHRAMBENOG PRIPRAVKA – PRINOVA U OBITELJI »*LUCOR METALLICUM*«

Iznesen je kratak kritički prikaz metabolizma molibdena u ljudi. Prikazan je prvi poznati slučaj akutnog otrovanja molibdenom (Mo) iz prehrambenog pripravka, preparata molibdena, u zdravstvenog radnika (pacijent). Nakon 18 dana uzimanja preparata molibdena u ukupnoj količini od 13,5 mg Mo (300–800 μg Mo na dan) u njega se razvila slika akutne psihoze sa slušnim i vizualnim halucinacijama, nizom *petit mal* i jednim za život opasnim *grand mal* napadom. Navedeni akutni klinički simptomi povukli su se u roku od nekoliko sati nakon započete terapije kelatirajućim spojem kalcij etilen diamin tetraoctenom kiselinom (CaEDTA). Baterijom neuropsiholoških testova i spektralnom emisijskom kompjutorskom homografijom (Spectral Emission Computer Tomography, SPECT) utvrđena su opsežna oštećenja frontalnog korteksa mozga. Godinu dana nakon otrovanja molibdenom u pacijenta je dijagnosticirana toksična encefalopatija sa deficitom provedbenih funkcija, nesposobnost učenja, teška depresija (djelomično poboljšana liječenjem testosteronom) i posttraumatski stresni poremećaj. Koncentracija molibdena u plazmi nije pouzdan dokaz otrovanja jer je njegov biološki poluživot u plazmi ($t/_2$) svega 8 sati. Afinitet molibdena prema masnim tkivima moguća je biokemijska podloga brze redistribucije molibdena iz krvi u mozak. Upozoreno je na potrebu striktnih uputa kako se prehrambeni pripravci s potencijalno opasnim svojstvima, poput molibdena, ne smiju miješati s drugim sličnim pripravcima te da je nužno navesti (i poštovati) njihovu maksimalnu pojedinačnu i kumulativnu dozu. Molibden je neželjena prinova u obitelji »*metalnog ludila*«.

Ključne riječi:
akutna Mo psihoza, kinetika apsorpcije, neurotoksikološka procjena, smanjena izvršna poslovna sposobnost, spektralna emisijska kompjutorska tomografija (SPECT) mozga, kelatirajuća terapija CaEDTA-om

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