Severe Symptoms after IV Administration of Alpha Lipoic Acid for "Heavy Metal Detoxification"

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Objective

Alpha lipoic acid (ALA; thioctic acid) is an organosulfur compound derived from octanoic acid. It can be found in mitochondria of almost all eukaryotes and is essential for cellular respiration.

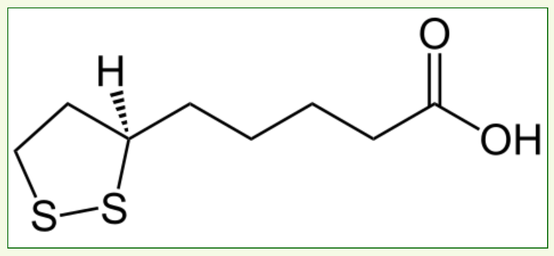

Figure 1 - alpha lipoic acid

ALA is used traditionally as an antioxidant and for treatment of diabetes, neuropathy, and AIDS. It was also investigated in several studies as a treatment of Amanita poisoning, but was not effective (1). To date, it is marketed as dietary supplement and pharmaceutical drug, usually containing 600 milligrams per single dose.

Many natural health professionals and alternative practitioners offer therapies for "heavy metal detoxification" which often contain chelators in non-therapeutic doses as well as other antioxidants, such as ALA. Although ALA may have some benefits as an antioxidant, its use for "heavy metal detoxification" is highly questionable.

We report on five cases of severe symptoms after IV administration of ALA in a mixed infusion for "heavy metal detoxification".

Case series

Within 3 days in August 2017, four cases with very similar symptoms were reported to the Poisons Information Centre Erfurt. A fifth case became known to us only about a month later, but happened within the same week.

First signs ranged from flu-like symptoms (qualm, chills, muscle pain) and gastrointestinal disturbances, to haematomas and petechiae. Subsequently, elevation of liver enzymes and hepatitis or acute liver failure, as well as coagulopathy or DIC were observed.

It soon became apparent that all patients had been under treatment for "heavy metal detoxification" at the same practitioner of holistic medicine. They were 35 to 54 years old, and four were female, one male. Within hours to four days previous to their admission to different hospitals, they had allegedly received an identical mixed infusion of 250 mg DMPS (2,3-Dimercapto-1-propanesulfonic acid), 1900 mg EDTA calcium disodium, 100 mg procaine, 8400 mg sodium bicarbonate, and 600 mg alpha lipoic acid. Initially, the dosage was unknown, but in the fifth case that was reported later, the dosage was specified as stated above.

All patients largely recovered within two or three weeks, respectively, but in at least one case long-term sequelae could not be ruled out completely.

Case report

A 53 year-old female obtained said infusion on a Thursday afternoon between 4.30 and 6.00 pm. An hour after completion, nausea, vomiting, fever and myalgia occurred. Upon IV administration of an analgesic, local haematomas appeared immediately. She was admitted to hospital, where laboratory findings showed leukocytosis, thrombocytopenia, and elevated liver enzymes. Further symptoms were nosebleed, chills and fever, clotting disturbances, and massive elevation of ferritin. ALA serum level was 10.28 mg/L at 6.5 hours, and 1.305 mg/L at 12.5 hours after the end of infusion, respectively (*Table 1*). The patient was in intensive care and received clotting factors, as well as blood transfusion. Within 13 days, her general health condition improved and there seemed to be no lasting liver injury or bone marrow damage. However, three months later the patient still had elevated liver enzymes and showed signs of primary biliary cholangitis, which may as well have been caused by previous administration of ALA.

Kinetic evaluations

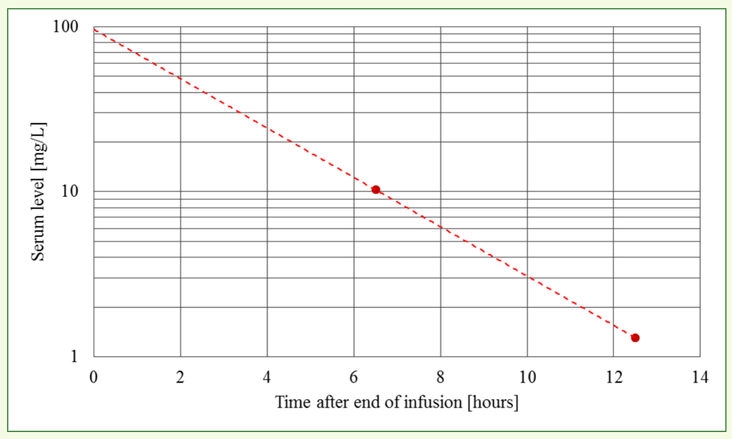

Figure 2 - Serum levels of ALA after end of infusion with trendline

Using alpha lipoic acid serum levels from the case report, we could extrapolate the "initial" serum level, assuming first-order kinetics of ALA elimination after the end of infusion (*Figure 2*).

Body weight	67 kg
Volume of distribution (2)	2.2 L/kg
Half life (2)	10 minutes to 2 hours
Calculated half life (with data from case report)	2 hours
Serum level maximum after administration of 600 mg (2)	3.5 to 10 mg/L
Serum level 6.5 hours after end of infusion	10.28 mg/L
Serum level 12.5 hours after end of infusion	1.305 mg/L
Estimated serum level (at end of infusion)	97 mg/L
Estimated dose (at end of infusion)	14297.8 mg = **14.3 g**

Table 1 - Kinetic values and serum levels of ALA from case report

Similar results were obtained with first-order kinetic calculation (*Table 1*). Considering the fact, that this dose is estimated at the end of infusion, the total dose might have been even higher (approx. 18 g).

Discussion

Therapeutic doses of all stated components of the mixture do not sufficiently explain the symptoms. Additionally, four of the five patients had previously received that same mixture without showing any of the current effects. Therefore, we assume that the mixture had been incorrectly produced and an at least tenfold overdosing of ALA has caused the symptoms in these cases.

Although there are only few reports on ALA overdose, one published case report describes severe symptoms, including cardiac failure, seizures, renal failure, thrombocytopenia, coagulopathy, multi organ failure, and death (3). There is also evidence for liver toxicity following high doses in animals (4).

Conclusion

The symptoms we observed in our cases were quite similar to the symptoms described in the literature (3), backing our theory of ALA overdose. Also, although valuable serum levels for ALA were obtained only in one of our cases, a rough estimation of the actually administered dose was possible. This estimated dose (at the end of infusion) was considerable higher than the stated dose, confirming our first assumption of a significant overdosing.

References

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