DOCTOR'S DATA INC.

ORDER: 210910-2339
TEST: U210910-2339-1
PATIENT: Anna Thomasson
ID: THOMASSON-A-00018
SEX: Female
AGE: 47 DOB: 12/02/1973

CLIENT #: 38350
DOCTOR: William Spurlock MD
Renewed Vitality
9535 Forest Ln 100
Dallas, TX 75243 U.S.A.

Toxic Metals; urine

TOXIC METALS		RESULT µg/g Creat	REFERENCE INTERVAL	WITHIN REFERENCE / OUTSIDE REFERENCE
Aluminum	(Al)	8.8	< 25	
Antimony	(Sb)	3.5	< 0.18	
Arsenic	(As)	4.4	< 50	
Barium	(Ba)	2.5	< 5	
Beryllium	(Be)	<dl	< 0.01	
Bismuth	(Bi)	0.93	< 1	
Cadmium	(Cd)	0.18	< 0.9	
Cesium	(Cs)	6.9	< 10	
Gadolinium	(Gd)	0.08	< 0.8	
Lead	(Pb)	91	< 1.2	
Mercury	(Hg)	8.6	< 1.3	
Nickel	(Ni)	2.6	< 5	
Palladium	(Pd)	0.15	< 0.3	
Platinum	(Pt)	<dl	< 0.1	
Tellurium	(Te)	0.21	< 0.5	
Thallium	(Tl)	0.77	< 0.5	
Thorium	(Th)	<dl	< 0.02	
Tin	(Sn)	28	< 5	
Tungsten	(W)	<dl	< 0.4	
Uranium	(U)	<dl	< 0.03	

URINE CREATININE	RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	47.9	30 – 225	

SPECIMEN DATA

Comments:
Date Collected: 09/06/2021
Date Received: 09/10/2021
Date Reported: 09/13/2021
Methodology: ICP-MS QQQ, Creatinine by Jaffe Reaction

Provoking Agent: DMSA 500 MG 4
Collection Period: 8 hours
pH upon receipt: Acceptable

< dl: less than detection limit
Results are creatinine corrected to account for urine dilution variations. **Reference intervals are based upon NHANES (cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions.** Chelation (provocation) agents can increase urinary excretion of metals/elements.

Introduction

This analysis of urinary elements was performed by ICP-Mass Spectroscopy following acid digestion of the specimen. Urine element analysis is intended primarily for: diagnostic assessment of toxic element status, monitoring detoxification therapy, and identifying or quantifying renal wasting conditions. It is difficult and problematic to use urinary elements analysis to assess nutritional status or adequacy for essential elements. Blood, cell, and other elemental assimilation and retention parameters are better indicators of nutritional status.

- 24 Hour Collections
  "Essential and other" elements are reported as mg/24 h; mg element/urine volume (L) is equivalent to ppm. "Potentially Toxic Elements" are reported as µg/24 h; µg element/urine volume (L) is equivalent to ppb.
- Timed Samples (< 24 hour collections)
  All "Potentially Toxic Elements" are reported as µg/g creatinine; all other elements are reported as µg/mg creatinine. Normalization per creatinine reduces the potentially great margin of error which can be introduced by variation in the sample volume. It should be noted, however, that creatinine excretion can vary significantly within an individual over the course of a day.

If one intends to utilize urinary elements analysis to assess nutritional status or renal wasting of essential elements, it is recommended that unprovoked urine samples be collected for a complete 24 hour period. For provocation (challenge) tests for potentially toxic elements, shorter timed collections can be utilized, based upon the pharmacokinetics of the specific chelating agent. When using EDTA, DMPS or DMSA, urine collections up to 12 hours are sufficient to recover greater than 90% of the mobilized metals. Specifically, we recommend collection times of: 9 - 12 hours post intravenous EDTA, 6 hours post intravenous or oral DMPS and, 6 hours post oral bolus administration of DMSA. What ever collection time is selected by the physician, it is important to maintain consistency for subsequent testing for a given patient.

If an essential element is sufficiently abnormal per urine measurement, a descriptive text is included with the report. Because renal excretion is a minor route of excretion for some elements, (Cu, Fe, Mn Zn), urinary excretion may not influence or reflect body stores. Also, renal excretion for many elements reflects homeostasis and the loss of quantities that may be at higher dietary levels than is needed temporarily. For these reasons, descriptive texts are provided for specific elements when deviations are clinically significant. For potentially toxic elements, a descriptive text is provided whenever levels are measured to be higher than expected. If no descriptive texts follow this introduction, then all essential element levels are within acceptable range and all potentially toxic elements are within expected limits.

Reference intervals and corresponding graphs shown in this report are representative of a healthy population under non-provoked conditions. Descriptive texts appear in this report on the basis of measured results and correspond to non-challenge, non-provoked conditions.

Chelation (provocation) agents can increase urinary excretion of metals/elements. Provoked reference intervals have not been established therefore non-provoked reference intervals shown are not recommended for comparison purposes with provoked test results. Provoked results can be compared with non-provoked results (not reference intervals) to assess body burden of metals and to distinguish between transient exposure and net retention of metals. Provoked results can also be compared to previous provoked results to monitor therapies implemented by the treating physician. Additionally, Ca-EDTA provoked results can be used to calculate the EDTA/Lead Excretion Ratio (LER) in patients with elevated blood levels.

CAUTION: Even the most sensitive instruments have some detection limit below which a measurement cannot be made reliably. Any value below the method detection limit is simply reported as "< dl." If an individual excretes an abnormally high volume of urine, urinary components are likely to be extremely dilute. It is possible for an individual to excrete a relatively large amount of an element per day that is so diluted by the large urine volume that the value measured is near the dl. This cannot automatically be assumed to be within the reference range.

This analysis of urinary metals was performed by ICP-Mass Spectroscopy. Urine metal analysis is traditionally used for evaluation of very recent or ongoing exposure to potentially toxic metals. The urinary excretion of certain metals is known to be increased (provoked) to a variable extent after administration of specific chelating agents. Reference values and corresponding graphs are representative of a healthy population under non-provoked conditions; reference values have not been established for provoked urine samples. Reference values are age and sex specific.

For timed, random or first morning urine collections, metals are reported as µg/ gram creatinine. Normalization per creatinine reduces the potentially great margin of error that can be introduced by variation in the sample volume (concentration). It should be noted that creatinine excretion for an individual may vary to some extent over the course of a day, and from day to day. For 24 hour (h) urine collections elements are reported as µg/24 h. Results are also reported as µg element/gram creatinine to ensure clinically useful information in the event that an inaccurate 24 h urine volume was reported to the laboratory.

Descriptive texts appear in this report if detected levels of specific elements are abnormally high by comparison to the unprovoked reference values. If no descriptive texts follow this introduction, potentially toxic metals are within reference limits.

Antimony High

This individual's urine antimony (Sb) is higher than expected, but potential associated symptoms and toxic effects may not be present. This is because antimony has two valences: Sb+3 and Sb+5. Sb+3 is inherently the more toxic but is mostly excreted in feces. Sb+5, less toxic, binds less well to body tissues and is excreted mostly in urine. The current analysis does not differentiate the two forms of Sb.

Antimony can be assimilated by inhalation of Sb salt or oxide dust, ingested with (contaminated) foods or fluids, or absorbed transdermally. Inhalation may occur in industrial areas that involve smelting or alloying is done (usually with copper, silver, lead, tin). Sb is present in tobacco at about 0.01% by weight; about 20% of this is typically inhaled by cigarette smoking (Carson et al., Toxicology and Biological Monitoring of Metals in Humans, Lewis Pub. p. 21, 1987). Antimony compounds are used for fireproofing textiles and plastics, and this element may be found in battery electrodes, ceramics and pigments. Antimony can be absorbed with the handling of gun powder or the frequent use of firearms. Recent studies indicate high levels of antimony in sheepskin bedding produced in New Zealand. Antimony contaminationof soft plastic-bottled water is time and temperature dependent.

Symptoms of mild Sb exposure/retention may be insidious and multiple including: fatigue, muscle weakness, myopathy, and metallic taste. Chlorides and oxides of both valences of Sb can be mutagenic and may affect leukocyte function. Sb can bond to sulfhydryl (-SH) sites on enzymes and may interfere with cellular metabolism. Acute symptoms that may be associated with excessive Sb exposure/retention include: respiratory tissue irritation and pneumoconiosis with (chronic) inhalation of Sb dusts, RBC hemolysis with inhalation of stibine (SbH3) vapor, and gastrointestinal distress if orally ingested. Skin exposure can produce "antimony spots" or rashes which resemble chicken pox. Certain molds can produce the highlyneurotoxic stibine gas from Sb; stibine inhibits acetylcholinestelase activity.

A hair element analysis may be used to further assess Sb exposure. Antimony may be elevated in urine following administration of DMPS or DMSA if exposures to Sb have resulted in net retention; such levels may or may not be associated with overt adversehealth effects.

Lead High

This individual's urine lead (Pb) is higher than expected which means that Pb exposure is higher than that of the general population. A percentage of assimilated Pb is excreted in urine. Therefore the urine Pb level reflects recent or ongoing exposure to Pb and the degree of excretion or endogenous detoxification processes.

Sources of Pb include: old lead-based paints, batteries, industrial smelting and alloying, some types of solders, Ayruvedic herbs, some toys and products from China and Mexico, glazes on(foreign) ceramics, leaded (anti-knock compound) fuels, bullets and fishing sinkers, artist paints with Pb pigments, and leaded joints in municipal water systems. Most Pb contamination occurs via oral ingestion of contaminated food or water or by children mouthing or eating Pb-containing substances. The degree of absorption of oral Pb depends upon stomach contents (empty stomach increases uptake) and upon the essential element intake and Pb status. Deficiency of zinc, calcium or iron increases Pb uptake. Transdermal exposure is significant for Pb-acetate (hair blackening products). Inhalation has decreased significantly with almost universal use of non-leaded automobile fuel.

Lead accumulates in extensively in bone and can inhibit formation of heme and hemoglobin in erythroid precursor cells. Bone Pb is released to soft tissues with bone remodeling that can be accelerated with growth, menopausal hormonal changes, osteoporosis, or skeletal injury. Low levels of Pb may cause impaired vitamin D metabolism, decreased nerve conduction,and developmental problems for children including: decreased IQ, hearing impairment, delayed growth, behavior disorders, and decreased glomerular function. Transplacental transfer of Pb to the fetus can occur at very low Pb concentrations in the body. At relatively low levels, Pb can participate in synergistic toxicity with other toxic elements (e.g. cadmium, mercury).

Excessive Pb exposure can be assessed by comparing urine Pb levels before and after provocation with Ca-EDTA (iv) or oral DMSA. Urine Pb is higher post-provocation to some extent in almost everyone. Whole blood analysis reflects only recent ongoing exposure and does not correlate well with total body retention of Pb. However, elevated blood Pb is the standard of care for diagnosis of Pb poisoning (toxicity).

Mercury High

This individual's urine mercury (Hg) is higher than expected but may not be sufficiently high to be associated with overt pathophysiological effects. Symptomatology depends on many factors: the chemical form of Hg, its accumulation in specific tissues, presence of other toxicants, presence of disease that depletes glutathione or inactivates lymphocytes or is immunosuppressive, and the concentration of protective nutrients, (e.g. zinc, selenium).

Early signs of excessive Hg exposure include: decreased senses of touch, hearing, vision and taste,metallic taste in mouth, fatigue or lack of physical endurance, and increased salivation. Symptoms may progress with moderate or chronic exposure to include: anorexia, numbness and paresthesias, headaches,hypertension, irritability and excitability, and immune suppression/ dysregulation. Advanced disease processes from excessive Hg assimilation include: tremors and incoordination, anemia, psychoses, manic behaviors, possibly autoimmune disorders and renal dysfunction or failure. Note that in Hg exposure of long duration, renal excretion of Hg (and normal metabolites) may become impaired, and the urine level of Hg might be only mildly elevated or not elevated at all due to renal failure.

Mercury is used in: dental amalgams (50% by weight), explosive detonators; some vaccines, pure liquid form in thermometers, barometers, and laboratory equipment; batteries and electrodes,some medications and Ayurveic herbs, fungicides and pesticides, and in the paper industry. The fungicide/pesticide use of mercury has declined due to environmental concerns, but Hg residues persist in the environment. Emissions from coalfired power plants and hospital/municipal incinerators are significant sources of mercury pollution.

Methylmercury, the most common, organic form of Hg, occurs by methylation of inorganic Hg in aquatic biota or sediments (both freshwater and ocean sediments). Methylmercury accumulates in aquatic animals and fish and is concentrated up the food chain reaching highest concentrations in large fish and predatory birds. Except for fish, the human intake of dietary mercury is negligible unless the food is contaminated with one of the previously listed forms/sources. Daily ingestion of fish can result in the assimilation of 1 to 10 micrograms of mercury/day.

Depending upon the extent of cumulative Hg exposure, elevated levels of urine Hg may occur after administration of DMPS, DMSA or D-penicillamine. Blood and especially red blood cell elemental analyses are useful for assessing recent or ongoing exposure to organic (methyl) Hg.

Thallium High

This individual's urine thallium (Tl) is higher than expected, but associated symptoms or toxic effects may or may not be presented. Presentation of symptoms can depend upon several factors including: chemical form of the Tl, mode of assimilation, severity and duration of exposure, and organ levels of metabolites and nutrients that effect the action of Tl in the body.

Thallium can be assimilated transdermally, by inhalation, or by oral ingestion. Both valence states can have harmful effects: Tl+1 may displace potassium from binding sites and influences enzyme activities; Tl+3 affects RNA and protein synthesis. Tl is rapidly cleared from blood and is readily taken up by tissues. It can be deposited in kidneys, pancreas, spleen, liver, lungs, muscles, neurons and the brain. Blood is not a reliable indicator of Tl exposure.

Symptoms that may be associated with excessive Tl exposure are often delayed. Early signs of chronic, low-level Tl exposure and retention may include: mental confusion, fatigue, and peripheral neurological signs: paresthesias, myalgias, tremor and ataxia. After 3 to 4 weeks, diffuse hair losswith sparing of pubic and body hair and a lateral fraction of eye- brows usually occurs. Increased salivation occurs less commonly. Longer term or residual symptoms may include: alopecia, ataxia, tremor, memory loss, weight loss, proteinuria (albuminuria), and possibly psychoses. Ophthalmologic neuritis and strabismus may be presented.

Environmental and occupational sources of Tl include: contaminated drinking water, airborne plumes or waste streams from lead and zinc smelting, photoelectric, electrochemical and electronic components (photoelectric cells, semiconductors, infrared detectors, switches), pigments and paints, colored glass and synthetic gem manufacture, and industrial catalysts used in some polymer chemistry processes. Thallium is present in some "weight loss" supplements (e.g. Active 8) at undisclosed levels ("trade secret").

Hair (pubic or scalp) element analysis may be used to test for suspected Tl exposure.Although urine is the primary natural route for excretion of thallium, the biliary/fecal route also contributes. Therefore, fecal metals analysis provides a confirmatory test for chronic ongoing exposure to Tl. Clinical findings that might be associated with excessive Tl are: albuminuria,EEG with diffuse abnormalities, hypertension, and elevated urine creatinine phosphokinase (CPK).No provocation agents are currently available to estimate Tl retention by means of urinalysis.

Tin High

Tin is elevated in this individual's urine, and urine accounts for at least 80% of excreted tin that is ingested and absorbed from the gastrointestinal tract. Ingested tin is not significantly absorbed if it is an inorganic form. Oxide coatings readily form on metallic tin, and salts can quickly oxidize making them insoluble. Organic tin, however, is bioavailable and more readily absorbed. Some organic tin compounds such as short-chain alkyltins can be absorbed transdermally and can causedegeneration of myelin.

Food and drink usually provide small daily intakes of (nontoxic) tin, with amounts depending upon type of food, packaging, quality of drinking water and water piping materials. Total daily intake is expected to vary from about 0.1 to 15 milligrams. Tin is present in many metal alloys and solders; bronze, brass and pewter contain the element. Dyes, pigments and bleaching agents often contain tin. Anticorrosion plating of steel and electrical components may also use tin. "Tin cans" are tin-plated steel with a thin outer oxide layer allowing the surface to be shiny but inert. Modern food-containing cans usually have polymer coatings that prevent food-metal contact. In the pastsome toothpastes contained stannous fluoride, a soluble fluoride source for strengthening tooth enamel. Currently most brands of fluoridated toothpastes contain sodium fluoride. Organic tins, theusually toxic forms, are: biocides (triphenyltin and alkyltins) used against rodents, fungi, insects andmites; curing agents for rubbers and silicones (dialkyltin); and methyltin formed bacteriologically(similar to methylmercury).

Mildly elevated levels of tin in urine may reflect sporadic dietary intake and excretion; there may be no associated symptoms. A two- or three-fold increase in urine tin levels is not uncommon following administration of EDTA or with sulfhydryl agents (DMSA, D-penicillamine, DMPS). Early signsof chronic organic tin excess can be: reduced sense of smell, headaches, fatigue and muscle aches, ataxiaand vertigo. Hyperglycemia and glucosuria are reported. Also, for organic tin exposure, there can beirritation of contacted tissues (eyes, skin, bronchial tubes, or GI tract). Later, immune dysfunctionmay occur with reduced lymphocytes and leukocytes; mild anemia may occur. A hair element analysis can beused to corroborate tin excess. Tin is commonly elevated in urine from autistic patients followingadministration of DMSA or DMPS.