

ORDER: 241029-2002 TEST: U241029-2002-1 PATIENT: Michael Pontidis ID: PONTIDIS-M-00001

SEX: Male

AGE: 50 DOB: 01/10/1974

CLIENT #: 47223

DOCTOR: Michael Bauerschmidt MD Deeper Healing Medical Wellness 234 Seven Farms Drive Ste 110 Charleston, SC 29492 U.S.A.

Toxic Metals; urine

TOXIC METALS					
	RESULT μg/g Creat	REFERENCE INTERVAL	WITHIN REFERENCE	OUTSIDE REFERENCE	
Aluminum (AI)	5.0	< 15			
Antimony (Sb)	0.067	< 0.18			
Arsenic (As)	3.9	< 40			
Barium (Ba)	2.0	< 5			
Beryllium (Be)	<dl< td=""><td>< 0.01</td><td></td><td></td></dl<>	< 0.01			
Bismuth (Bi)	0.026	< 0.8	ı		
Cadmium (Cd)	0.41	< 0.6			
Cesium (Cs)	3.3	< 9			
Gadolinium (Gd)	1.7	< 0.5			
Lead (Pb)	5.5	< 1.1			
Mercury (Hg)	0.84	< 0.8			
Nickel (Ni)	1.5	< 4			
Palladium (Pd)	<dl< td=""><td>< 0.3</td><td></td><td></td></dl<>	< 0.3			
Platinum (Pt)	<dl< td=""><td>< 0.1</td><td></td><td></td></dl<>	< 0.1			
Tellurium (Te)	<dl< td=""><td>< 0.5</td><td></td><td></td></dl<>	< 0.5			
Thallium (TI)	0.35	< 0.4			
Thorium (Th)	<dl< td=""><td>< 0.015</td><td></td><td></td></dl<>	< 0.015			
Tin (Sn)	3.9	<3			
Tungsten (W)	<dl< td=""><td>< 0.4</td><td></td><td></td></dl<>	< 0.4			
Uranium (U)	0.009	< 0.03			

URINE CREATININE						
	RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD			
Creatinine	70.0	35 – 240				

SPECIMEN DATA

Comments:

Date Collected: 10/28/2024 Provoking Agent: DMPS 250MG pH upon receipt: Acceptable

Date Received:10/29/2024Provocation: Post ProvocativeDate Reported:10/30/2024Collection Period: 6 hours

Methodology: ICP-MS QQQ, Creatinine by Jaffe Reaction

< 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.

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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 μ g/24 h; μ g element/urine volume (L) is equivalent to ppb.

Timed Samples (< 24 hour collections)

All "Potentially Toxic Elements" are reported as $\mu g/g$ creatinine; all other elements are reported as $\mu g/mg$ 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 $\mu g/g$ 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 $\mu g/24$ h. Results are also reported as $\mu g/24$ 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.

Gadolinium High

This individual's urine level of Gadolinium (Gd) is higher than expected. Gadolinium is one of the most abundant "rare-earth" elements but is never found as a free element in nature. Gadolinium has no known biological role in humans.

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Toxicity is rarely associated with Gd due to its poor gastrointestinal absorption (it is suspected that very little Gd is absorbed from the gastrointestinal tract (<0.05%). If exposure to high enough doses and/or if absorption does occur, symptoms of acute toxicity may develop, including abdominal cramps, diarrhea, lethargy, muscular spasms, and even eventual death due to respiratory collapse. Gadolinium salts can cause irritation of the skin and eyes and are suspected to be possible carcinogens. As reported by Perazella (2009) Gadollinium-based contrast (GBC) agents have been linked on occasion with a rare systemic fibrosing condition called nephrogenic systemic fibrosis (NSF) and their use in patients with even mild kidney disease should be avoided (parenteral administration).

Gd is often used in alloys (e.g. chromium, iron). Other technical uses include the phosphors of color television tubes and in making magnets and electronic components such as recording heads for video recorders and in the manufacture of compact disks and computer memory. In medicine Gd, chelated with diethylenetriaminepentaacetic acid (DTPA), is used as a non-radioactive contrasting agent in magnetic resonance imaging and has a half-life in blood of about 90 minutes. However, residual Gd is retained in tissues for quite some time. It is also used in control rods for nuclear reactors and power plants, in making garnets for microwave applications.

EDTA effectively chelates Gd therefore urinary Gd might be higher than average post-Ca-EDTA provocation, particularly in patients who have had Gd-enhanced MRIs.

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.

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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.