



ORDER: 241030-2022
 TEST: U241030-2022-1
 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	(Al)	1.1	< 15		
Antimony	(Sb)	0.031	< 0.18		
Arsenic	(As)	2.7	< 40		
Barium	(Ba)	1.2	< 5		
Beryllium	(Be)	<dl	< 0.01		
Bismuth	(Bi)	0.005	< 0.8		
Cadmium	(Cd)	0.17	< 0.6		
Cesium	(Cs)	2.8	< 9		
Gadolinium	(Gd)	0.016	< 0.5		
Lead	(Pb)	0.13	< 1.1		
Mercury	(Hg)	0.17	< 0.8		
Nickel	(Ni)	0.6	< 4		
Palladium	(Pd)	0.03	< 0.3		
Platinum	(Pt)	<dl	< 0.1		
Tellurium	(Te)	<dl	< 0.5		
Thallium	(Tl)	0.34	< 0.4		
Thorium	(Th)	<dl	< 0.015		
Tin	(Sn)	4.0	< 3		
Tungsten	(W)	<dl	< 0.4		
Uranium	(U)	<dl	< 0.03		

URINE CREATININE							
	RESULT mg/dL	REFERENCE INTERVAL	-2SD	-1SD	MEAN	+1SD	+2SD
Creatinine	257	35 – 240					

SPECIMEN DATA	
Comments: Date Collected: 10/28/2024 Date Received: 10/30/2024 Date Reported: 10/31/2024 Methodology: ICP-MS QQQ, Creatinine by Jaffe Reaction	Provocation: Pre Provocative Collection Period: Random 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.



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Essential Elements; urine

ESSENTIAL ELEMENTS							
	RESULT mEq/g Creat	REFERENCE INTERVAL	PERCENTILE				
			2.5 th	16 th	50 th	84 th	97.5 th
Sodium (Na)	31.7	40 – 200					
Potassium (K)	25.7	20 – 90					
	RESULT µg/mg Creat						
Phosphorus (P)	242	150 – 1000					
Calcium (Ca)	107	20 – 250					
Magnesium (Mg)	74.3	20 – 200					
Zinc (Zn)	1.9	0.09 – 1.3					
Copper (Cu)	0.0062	0.003 – 0.022					
Sulfur (S)	403	250 – 900					
Molybdenum (Mo)	0.0391	0.01 – 0.11					
Boron (B)	0.62	0.5 – 3.8					
Lithium (Li)	0.115	0.008 – 0.18					
Selenium (Se)	0.206	0.03 – 0.2					
Strontium (Sr)	0.085	0.035 – 0.26					

	RESULT µg/g Creat	REFERENCE INTERVAL	68 th 95 th	
Cobalt (Co)	0.58	< 1		
Iron (Fe)	3	< 50		
Manganese (Mn)	0.02	< 0.4		
Chromium (Cr)	0.13	< 1.5		
Vanadium (V)	0.017	< 0.6		

URINE CREATININE							
	RESULT mg/dL	REFERENCE INTERVAL	-2SD	-1SD	MEAN	+1SD	+2SD
Creatinine	257	35 – 240					

SPECIMEN DATA	
Comments:	
Date Collected: 10/28/2024	Provocation: Pre Provocative
Date Received: 10/30/2024	Collection Period: Random
Date Reported: 10/31/2024	pH upon receipt: Acceptable
Methodology: ISE, Spectrophotometry, 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.

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 essential elements was performed by ICP-Mass Spectroscopy. Analysis of essential and other elements in urine is used primarily to identify and characterize renal wasting conditions. Analysis of essential elements in urine is not a direct approach for assessing nutritional status or adequacy. Blood, cell, and other assimilation and retention parameters are optimal direct indicators of essential element status.

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 24 hour urine collections essential elements are reported as mg/24 h. For timed or first morning urine collections, elements are normalized per gram creatinine to avoid the potentially great margin of error which 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.

If an essential element is sufficiently abnormal per urine measurement, a descriptive text is included with the report. If there are no descriptive texts following this introduction, all essential element levels are within acceptable range. All reference ranges are age and sex specific.

This analysis of urinary toxic metals and essential elements was performed by ICP-Mass Spectroscopy. Analysis of metals in urine 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.

Analysis of essential and other elements in urine is used primarily to identify and characterize renal wasting conditions. Analysis of essential elements in urine is not a direct approach for assessing nutritional status or adequacy. Blood, cell, and other assimilation and retention parameters are optimal direct indicators of essential element status.

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 24 hour urine collections essential elements are reported as mg/24 h, and toxic metals are reported as µg/24 h. For timed, random or first morning urine collections, elements and metals are normalized per gram creatinine to avoid 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.

If an essential element is sufficiently abnormal per urine measurement, a descriptive text is included with the report. For potentially toxic elements, a descriptive text is provided whenever levels are measured to be higher than the unprovoked reference values. If there are no descriptive texts following this introduction, all essential element levels are within acceptable range and all potentially toxic metals are at levels below the unprovoked reference values. All reference ranges and reference values are age and sex specific.

Boron Low

Boron (B) is introduced to the body mainly through food (noncitrus fruits, leafy vegetables, nuts, legumes, wine, cider, beer) and drinking water but is also found in food preservatives (sodium borate), and insecticides (boric acid). Although there is an absolute requirement for B in vascular plants, evidence for biological essentiality in animals (including man) is limited. It has been proposed that boron contributes to living systems by acting indirectly as a proton donor and that it exerts a particular influence on cell membrane and structure and function. Boron is rapidly absorbed and excreted largely in the urine. Based on urinary recovery findings, more than 90% of ingested B is usually absorbed. Boron is distributed throughout the tissues and organs of animals and humans at concentrations mostly between 4.6 and 55.5 nmol (0.05 and 0.6 µg)/g fresh weight. Among the organs that contain the highest amounts of B are bone, spleen, and thyroid.

Boron influences macromineral metabolism and steroid hormone metabolism (testosterone, estrogen, DHEA, and 1,25 dihydroxycholecalciferol). A B deficient diet may also affect calcium metabolism and thus affects the composition, structure, and strength of bone. Signs of B deficiency in animals vary in nature and severity as the diet varies in its content of calcium, phosphorus, magnesium, potassium, cholecalciferol, aluminum, and methionine. Boron is also thought to have an estrogenic effect. In postmenopausal women consuming a very low B diet, B supplementation reduced the total plasma concentration of calcium and the urinary excretions of calcium and magnesium, and elevated the serum concentrations of 17β-estradiol, testosterone, and ionized calcium, mimicking the effects of estrogen ingestion in postmenopausal women. In another study of magnesium and B deprivation among 13 healthy postmenopausal women (aged 50-78 years), it was found that marginal magnesium and B deprivation may also affect brain function as measured by EEG. It seems there may be increased CNS activity following boron deprivation. In long term hemodialysis patients serum boron levels may be excessively decreased.

No B requirements have been set as of 1998. Estimates are that between 1-2 mg/day may be required. Average intake in the U.S. has been estimated at between 1.7-4.3 mg/day.

Cobalt High

Urinary cobalt (Co) provides an indication of recent or ongoing exposure to the metallic element, and B-12 vitamers. It should be noted that benign high urine Co levels may be associated with recent high B-12 supplementation. That urinary Co is associated with intact B-12, not free Co ions. All forms of B-12 contain Co safely entrapped in the core of the structure. B-12 vitamers are water soluble and are considered safe even at high doses.

Urinary Co is most commonly used as an indicator of occupational exposure to the metal, because urine is the primary elimination route of Co after respiratory exposure. Exposure to Co has been reported for hard metal workers, gas turbine and space propulsion workers, base metal refinery workers, dental technicians, construction workers and workers in the electronics industry.

Individuals bearing metal-on-metal (M-O-M) prosthetic implants (hip or knee replacements) may excrete higher than normal amounts of Co, albeit typically much lower than that associated with occupational exposure. Co is the most abundant metal released by physical wear from the bearing surfaces, but chromium may also be elevated. For patients with M-O-M prosthetics exhibiting abnormal Co excretion, consider the Implant Profile which assesses the blood levels of the six metals that are most commonly associated with orthopedic devices. Signs and symptoms that may be associated with high exposure to metallic Co include visual and auditory impairment, tinnitus, vertigo, impaired immune and renal function, cardiomyopathy, cognitive dysfunction/dementia, mood disorders, hypothyroidism, peripheral neuropathy and skin rashes.

Chelation may acutely increase urinary excretion of free Co ions, but not Co associated with B-12.

Kettelarij J, et al. Neglected exposure route: cobalt on skin and its associations with urinary cobalt levels *Occup Environ Med* 2018;75:837–842. doi:10.1136/oemed-2018-105099

Copper Low

Low urinary copper may or may not correspond to subnormal copper levels in body tissues, and other laboratory tests are more indicative of copper status. Such tests include measurement of: whole blood or blood cell copper, hair copper, erythrocyte superoxide dismutase activity, and serum ceruloplasmin. Because the major route of copper excretion is via bile and feces, urinary levels may fluctuate without reflecting or influencing body stores.

Lower than normal excretion of copper (and other elements) can occur in renal insufficiency; in which case blood levels may be normal or elevated. Inadequate levels of molybdenum or zinc allow increased retention of copper, and transient hypocuprinuria may occur during periods when copper stores are accumulating.

Low urinary copper may also correspond to copper deficiency of nutritional or gastrointestinal origins. The richest dietary sources of copper are: nuts, shellfish, liver, raisins and legumes. Dairy products generally are low in copper content. Gastric hypochlorhydria, sprue, and pancreatic dysfunction may inhibit copper uptake.

Lithium High

The concentration of lithium (Li) in this urine specimen is unexpectedly high. Li occurs almost universally at low concentrations in water and in plant and animal food products. Li has important functions in the nervous system, and possibly the immune system. Assimilation of Li from food, water and even commonly available organic Li supplements (when taken as directed) would not be expected to be associated with abnormally high levels of Li in urine. In contrast, much higher doses of inorganic Li carbonate, which are often prescribed for specific mood disorders, would be expected to be associated with markedly elevated urine Li if ingestion was recent or chronic.

Occupational/accidental assimilation of excessive amounts of Li could possibly be associated with the manufacture or improper handling of lightweight metal alloys, glass, lubrication greases, and batteries.

Li, when assimilated in excessive quantities, may cause dermatitis, nausea, confusion, coarse hand tremor, slurred speech, edema, or hypotension. Li toxicity may be more pronounced with low sodium intake. Point-in-time Li doses/exposure are rapidly excreted in urine, and blood analysis may not be indicative of exposure after 5 to 7 days.

Phosphorus Low

The level of phosphorus (P) in this sample is lower than expected. Phosphates also are present in every cell of the body where they are involved in chemical energy transfer and enzyme regulation. Phosphorylation chemistry is part of carbohydrate, amino acid, and lipid metabolism. Along with calcium, P assimilation into bone is regulated by vitamin D. P levels may be affected by abnormal calcium, P or vitamin D. P is a major component of mineralized tissue such as bone and teeth.

Phosphorus is found in most food sources and is a common ingredient of food additives. P deficiency is uncommon except in starvation or inherited genetic disorders of kidney function that waste P into the urine. Symptoms of true P deficiency include rickets, loss of appetite, muscle weakness, bone fragility and numbness in the extremities.

Phosphorus deficiency may be confirmed by serum, packed blood cell (RBC) element analysis, or whole blood elements. If clinically indicated by patient symptoms or history, vitamin D levels may be assessed.

Potassium Low

The level of potassium (K) is lower than expected in this sample. K is an electrolyte and a potentiator of enzyme functions in cells. K can be low in the body as the result of gastrointestinal or renal dysfunction, or as a side effect of some diuretics. In adrenocortical hyperactivity, blood levels of K are depressed, while urinary K is increased. Diabetic acidosis and other medical conditions may result in severe K loss. Symptoms of true K deficiency include: muscle weakness, fatigue, and tachycardia. An electrocardiogram may show abnormalities when K is low in serum/plasma or whole blood.

Appropriate tests to confirm low K in body tissues may include measurements of packed red blood cell K; serum or whole blood K and sodium/K ratio.

Selenium High

Urine accounts for about one-half of the total body excretion of dietary selenium when normal amounts are ingested. Seafood, organ meats, cereal grains, and seleniferous vegetables (garlic, onions) are good dietary sources. Selenium is also excreted in sweat, and lesser amounts are present in fecal matter. Because diets are highly variable in selenium content, urine is not a reliable indicator of selenium adequacy or function. However, selenium excess or overload can feature high urinary levels. Without occupational or environmental exposure, or excessive dietary intake, urinary selenium is expected to be below 100 micrograms per liter.

Selenium can be toxic with long-term intake as low as 750 mcg/day. Essential daily selenium requirements range from 10 micrograms (infants) to 50-70 micrograms (adults). Some manifestations of chronic selenium exposure are: fatigue, jaundice, hyperpigmentation of skin, unstable blood pressure, reddish discoloration and structural degeneration of nails and teeth, and dizziness. A garlic-like breath odor usually occurs and there may be a metallic taste in the mouth. Acute selenium contamination generally occurs from inhalation of selenium fumes which inflame mucous membranes and cause coughing and irritation of eyes and nasal passages.

Packed red blood cell elements analysis is a more definite test for selenium status. Hair analysis may provide confirmation of selenium excess if exogenous sources of contamination (antidandruff shampoos) are eliminated.

Sodium Low

The concentration of sodium in this urine sample is lower than expected and is more than two standard deviations below the mean. Low urine sodium levels are uncommon but may be seen, for example, with severe vomiting and/or diarrhea. Further, a low urine sodium concentration implies that the kidney's capacity to reabsorb sodium must be intact and that some stimulus to conserve sodium is present. Urine sodium can vary from day to day depending on the degree of water reabsorption. To get an accurate assessment of renal clearance of sodium, both urine and serum sodium can be compared - this can be done with the fractional excretion of sodium (FENa) calculation (1).

Most of the sodium in the human body can be found either in blood or lymphatic fluid. Sodium levels are regulated by aldosterone (from the adrenal cortex) which acts on the proximal tubules of the nephron to increase reabsorption of sodium and water and to increase the excretion of potassium. Urine sodium testing has a role in the assessment of sodium concentration in the extracellular fluid (ECF) - urine sodium test results should be correlated clinically with sodium and water intake, observation for clinical signs of ECF volume contraction or expansion, serum sodium levels, estimation of renal function and GFR as well as with urine osmolality.

In a normal individual, urine sodium excretion generally reflects dietary intake - the less one ingests (e.g. low salt diet, etc.) the less one excretes. In dehydration (e.g. vomiting, diarrhea, etc.) sodium may be retained (less sodium output in urine) in efforts to retain water. Decreased urine sodium concentration also may be associated with disease states such as Conn's syndrome (primary hyperaldosteronism due to an aldosterone-producing adenoma), congestive heart failure, liver disease and/or nephrotic syndrome. Low urine sodium has been associated with greater risk of myocardial infarction in males with high blood pressure (2).

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 cause degeneration 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 past some toothpastes contained stannous fluoride, a soluble fluoride source for strengthening tooth enamel. Currently most brands of fluoridated toothpastes contain sodium fluoride. Organic tins, the usually toxic forms, are: biocides (triphenyltin and alkyltins) used against rodents, fungi, insects and mites; 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 signs of chronic organic tin excess can be: reduced sense of smell, headaches, fatigue and muscle aches, ataxia and vertigo. Hyperglycemia and glucosuria are reported. Also, for organic tin exposure, there can be irritation of contacted tissues (eyes, skin, bronchial tubes, or GI tract). Later, immune dysfunction may occur with reduced lymphocytes and leukocytes; mild anemia may occur. A hair element analysis can be used to corroborate tin excess. Tin is commonly elevated in urine from autistic patients following administration of DMSA or DMPS.

Zinc High

High urinary zinc may or may not correspond to global zinc excess or to zinc loss from body tissues, because the major route for zinc excretion is via the bile, intestinal transport and feces. Typically, from two to ten percent of total zinc excretion occurs via urine; a similar amount occurs in sweat; the remainder (about 80 to 95%) occurs via biliary secretion to the intestine and is excreted in feces. Urine levels may fluctuate without reflecting or influencing body stores.

Very high urinary zinc levels are expected to result from EDTA detoxification therapy; 3 to 20 mg/L is commonly measured in the 12 hours following intravenous administration of EDTA. Lesser elevations of urine zinc also are expected to result from sulfhydryl agent detoxification therapy (DMPS, DMSA, D-penicillamine). One to five mg/L is commonly found in the 24 hours following administration of these agents. Zinc repletion may be beneficial or required during such therapies.

Breakdown of tissue releases zinc into extracellular fluids and increases urinary zinc levels. This may be observed following or in conjunction with: accidental injury, surgery, catabolism of diseased/disordered tissue, starvation (ketosis) and diabetes. Zinc wasting may occur in alcoholic cirrhosis.

Zinc overload or toxicity can occur from ingestion of zinc contaminated food or drink; galvanized pipes or pails can be sources. Occupational or environmental exposure to zinc fumes may produce an acute contamination or poisoning. Elevated urinary zinc beyond two standard deviations high (without provocation) warrants investigation of possible sources of zinc excess, or of tissue catabolism or injury.

Excessive amounts of zinc in body tissues may displace copper and/or iron from tissue binding sites and may provoke anemia. Symptoms consistent with chronic zinc toxicity include: lethargy, difficulty writing and with fine motor skills, light-headedness, and renal failure. Immediate symptoms (within 12 hours) of acute zinc excess via ingestion include: nausea, vomiting, diarrhea, exhaustion, headache, dizziness, and myalgia. Other laboratory findings consistent with zinc toxicity would be: elevated leukocyte count, elevated serum amylase and lipase, elevated whole blood zinc concentration, elevated hair zinc level (if the zinc excess is chronic).