Test Requisition: # Physician:

MOSAIC DIAGNOSTICS
Formerly Great Plains Laboratory

オーダー:
テスト
クライアント　名:
患者:
ID:
性別:　　年月日:

クライアント #:
Mosaic Diagnostics Llc/japanese
9221 Quivira Road
Overland Park, KS 66215 U.S.A.

毒性金属; 尿

毒性金属				
		結果 µg/g Creat	基準値	基準値内 / 高い
アルミニウム	(Al)	2.8	< 15	
アンチモン	(Sb)	0.030	< 0.18	
ヒ素	(As)	44	< 40	
バリウム	(Ba)	0.43	< 5	
ベリリウム	(Be)	<dl	< 0.01	
ビスマス	(Bi)	0.032	< 0.8	
カドミウム	(Cd)	0.36	< 0.6	
セシウム	(Cs)	5.7	< 9	
ガドリニウム	(Gd)	<dl	< 0.5	
鉛	(Pb)	9.8	< 1.1	
水銀	(Hg)	15	< 0.8	
ニッケル	(Ni)	<dl	< 4	
パラジウム	(Pd)	<dl	< 0.3	
プラチナ	(Pt)	<dl	< 0.1	
テルル	(Te)	0.10	< 0.5	
タリウム	(Tl)	0.62	< 0.4	
トリウム	(Th)	<dl	< 0.015	
錫	(Sn)	0.32	< 3	
タングステン	(W)	0.15	< 0.4	
ウラン	(U)	0.011	< 0.03	

クレアチニン			
	結果 mg/dL	基準値	-2SD -1SD MEAN +1SD +2SD
クレアチニン	54.9	35 - 240	

標本データ

コメント:
採取日:
受領日:
報告日:
方法論: ICP-MS QQQ, ヤッフェ反応によるクレアチニン

誘発因子: DMSA 500MG
誘発試験: Post Provocative
採取期間: 6 hours

受入時pH: Acceptable

< dl: less than detection limit
測定結果は、尿の希釈による変動に対処する為に、クレアチニン濃度で値が修正されています。基準範囲とそれに対応するグラフ、非負荷試験下の健常者の集団の値表しています。キレーション(負荷)物質によって金属元素の尿中排泄が増加する事があります。

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必須要素; 尿

必須要素

	結果 mEq/g Creat	基準値	PERCENTILE 2.5th 16th 50th 84th 97.5th
ナトリウム (Na)	171	40 – 200	
カリウム (K)	42.9	20 – 90	
	結果 µg/mg Creat		
リン (P)	648	150 – 1000	
カルシウム (Ca)	70	20 – 250	
マグネシウム (Mg)	40.5	20 – 200	
亜鉛 (Zn)	0.29	0.09 – 1.3	
銅 (Cu)	0.0196	0.003 – 0.022	
硫黄 (S)	522	250 – 900	
モリブデン (Mo)	0.140	0.01 – 0.11	
ホウ素 (B)	1.0	0.5 – 3.8	
リチウム (Li)	0.0082	0.008 – 0.18	
セレン (Se)	0.023	0.03 – 0.2	
ストロンチウム (Sr)	0.145	0.035 – 0.26	

	結果 µg/g Creat	基準値	68th 95th
コバルト (Co)	0.13	< 1	
鉄 (Fe)	6	< 50	
マンガン (Mn)	<dl	< 0.4	
クロム (Cr)	<dl	< 1.5	
バナジウム (V)	0.13	< 0.6	

クレアチニン

	結果 mg/dL	基準値	-2SD -1SD MEAN +1SD +2SD
クレアチニン	54.9	35 – 240	

標本データ

コメント:

採取日:
受領日:
報告日:

誘発因子: DMSA 500MG
誘発試験: Post Provocative
採取期間: 6 hours

受入時pH: Acceptable

方法論: ISE, Spectrophotometry, ICP-MS QQQ, ヤッフェ反応によるクレアチニン

< dl: less than detection limit
測定結果は、尿の希釈による変動に対処する為に、クレアチニン濃度で値が修正されています。基準範囲とそれに対応するグラフ、非負荷試験下の健常者の集団の値表しています。キレーション(負荷)物質によって金属元素の尿中排泄が増加する事があります。

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.

Arsenic High

This individual's urine arsenic (As) is higher than expected. Because urine is the major mode of excretion for arsenic, an elevated level reflects increased assimilation of As. Ingestion of organic species of As in seafood is not uncommon and may be associated with very elevated urine As. Arsenobetaine and arsinocholine, commonly found in shellfish are relatively non-toxic and 90% is excreted in the urine with a half-life of about 48 hours.

Sources of As include: contaminated foods (e.g. chicken), water or medications. Industrial sources are: ore smelting/refining/processing plants, galvanizing, etching plating processes. Tailing from or river bottoms near gold mining areas (past or present) may contain arsenic. Insecticides, rodenticides and fungicides (Na-, K- arsenites, arsenates, also oxides are commercially available). Commercial As-containing products include: sodium arsenite, calcium arsenate, lead arsenate and "Paris green" which is cupric acetoarsenite, a wood preservative (pressure treated wood). Undesirable levels of As have been found in many Ayruvedic herbs.

Chronic exposure to or ingestion of inorganic As causes tissue levels to gradually increase as the element binds to sulfur, phosphorus and selenium. An important detrimental effect is inactivation of lipoic acid,a vitamin cofactor needed for metabolism of pyruvate and alpha-ketoglutarate.

Symptoms consistent with mild or moderate As exposure include: fatigue, malaise, eczema or allergic-like dermatitis, and garlic-like breath. Increased salivation may occur. Hair element analysis may provide further evidence of As exposure to inorganic As. Blood As levels are not dose related and may or may not reflect As exposure or net retention of As. Levels of As may exceed the expected range after administration of DMPS or DMSA depending upon cumulative exposures. This does not necessarily indicate As excess to the point of toxic effects or physiological impairment.

Copper High

Significantly elevated copper in urine can be secondary to provocative challenge with sulfhydryl (-SH) bearing agents such as D-penicillamine ("Cuprimine"), DMSA, or DMPS. Large, multi-gram doses of vitamin C (ascorbic acid), administered orally or intravenously, may slightlyor moderately increase excretion of copper.

Increased urinary copper can be an artifact of nutritional supplementation with copper or come from drinking water that is high in copper content. Acidic water carried in copper pipes can dissolve some copper which increases the copper intake if used for drinking or cooking. Molybdenum supplementation at high levels or if inappropriate may cause increased copper excretion; molybdenum and copper are mutually antagonistic in terms of body retention.

Bacterial or other infections may cause hypercupremia with attendant or delayed hypercuprinuria. This is transient and follows the inflammatory stage of the disease. Published studies such as Vivoli, Sci Total Environ, 66 p. 55-64, 1987 have correlated increased urinary copper with increased blood pressures in hypertensives. Biliary obstruction or insufficiency can decrease normal excretion of copper via the bile while increasing blood and urinary levels. Proteinuria also may feature increased copper levels.

Hyperaminoacidurias that include histidinuria can result in urinary copper wasting because histidine is a powerful chelator of copper. Hyperaminoacidurias that include histidine can be of many origins including:genetic factors, chemical or elemental toxicities, infectious agents, hyperthyroidism, sugar intolerances,nephrotic syndromes, etc.

In Wilson's disease, urinary copper is generally increased (above 100 micrograms/24 hours) without provocation or chelation. Use of D-penicillamine or DMPS as a provocative diagnosticprocedure can yield a 5 - 10X increase in urinary copper levels in normal individuals. Incontrast, Wilson's disease patients may then excrete 50-100 times the normal levels or 1000 to2000 mcg/24 hr. (Walshe, J. Rheumatology (supp/7) 8 p.3-8, 1981).

Urine analysis (unprovoked) is not an adequate procedure to assess copper stores or copper metabolism. Blood levels, erythrocyte copper content, erythrocyte superoxide dismutase activity, and serum ceruloplasmin are other more indicative measurements for copper status.

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.

Molybdenum High

This individual's molybdenum level exceeds one standard deviation above the mean of the reference population which means that this individual's urine molybdenum level corresponds to the highest 17% (approximately) of that population.

Molybdenum is an essential activator of some important enzymes in the body: sulfite oxidase (catalyzes formation of sulfate from sulfite), xanthine oxidase (formation of uric acid and superoxide ion from xanthine), and aldehyde oxidase (processes aldehydes). Over 50% of absorbed Mo is normally excreted in urine; the remainder is excreted via bile to the intestines or is excreted in sweat.

Administration of EDTA is not observed to raise molybdenum levels in the urine. Significant urine Mo levels in molybdenum normal individuals (adults) may occur with D-penicillamine administration and up to 300 micrograms/24 hours is commonly observed (Doctor's Data). Similar increases with DMSA administration would be expected. For DMPS (administered slow-push intravenously) up to 250 micrograms Mo/24 hours is commonly seen, and prolonged use of dithiolchelators can deplete molybdenum stores.

Elevated Mo in urine can occur in renal wasting syndromes, nephritis, and biliary dysfunction or blockage. Other elements would then be relatively more increased (Mn, Fe, Cu). Administration of supplemental copper in high doses can result in elevated urine molybdenum; copper and molybdenum are mutually antagonistic with respect to body retention. Tungsten is a more powerful antagonist. Individuals doing tungsten-inert-gas ("TIG") welding may episodically excrete high amounts of molybdenum (but may actually be subnormal in body tissue levels). Increased dietary sulfate levels reduce intestinal absorption and increase renal excretion of molybdenum(eg. MSM).

Molybdenum is relatively nontoxic. Studies with animals show that huge oral does are required to produce clinical symptoms which are those of copper deficiency: loss of appetite, anemia, arthritic signs, diminished glucose tolerance, loss of skin pigmentation. Moderately excessive molybdenum uptake can produce gout-like symptoms and elevated blood/urine levels of uric acid.

If molybdenum excess is suspected, the following laboratory tests could be informative: serum and urine uric acid levels, hair multielement analysis including copper and molybdenum, packed blood cell molybdenum and copper levels, erythrocyte SOD activity.

Selenium Low

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.

Low urinary selenium may be a consequence of: junk food diet or highly-processed food diet, gastrointestinal dysfunctions, renal insufficiency (in which case other elements will be subnormal in urine but possibly elevated in blood), and long-term parenteral nutrition or special diets that are low in selenium.

Selenium is a necessary element for proper activity of two enzymes in human metabolism: glutathione peroxidase (GPx) and iodothyronine deiodinase (ITD). Selenium deficiency may cause weakness or rate limitation for one or both of these enzymes. GPx oxidizes glutathione while reducing oxidized lipids. Weak GPx activity may allow excessive inflammation to occur. ITD deiodinates thyroxine prohormone and catalyzes T4 _ T3. Selenium deficiency may be a cause of insufficient T3 and thyroid dysfunction (Berry J.M. Nature 349, 1991 pp.438-40).

Symptoms consistent with selenium deficiency include: myalgia, increased inflammatory responses, hypothyroidism with low T3. Cardiomyopathy and Keshan disease can occur in cases of severe, chronic Se deficiency. Subnormal selenium may accentuate the effects of cadmium, mercury or arsenic overload. Confirmatory tests for selenium status include packed red bloodcell elements, and hair elemental analysis (provided that antidandruff shampoos have not been used.

Sodium High

The concentration of sodium in this urine sample is higher than expected and is more than two standard deviations above the mean. A high urine sodium concentration can indicate that the kidney's capacity to reabsorb sodium might be impaired and/or that some stimulus to excrete 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 more one ingests (e.g. added dietary salt, drinking and cooking with softened water, salt poisoning, etc.) the more one excretes. High urine sodium may be associated, for example, with diuretic use or conditions such as Addison's disease (primary adrenal insufficiency).

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.

Vanadium High

A high level of Vanadium (V) was found in this urine sample. Increased V, especially in an unprovoked urine sample, reflects recent excessive exposure/intake and absorption to V.

Vanadium can be highly toxic. Excess levels of V can result from over-zealous V supplementation. It may also result from chronic consumption of fish, shrimp, crabs, and oysters that have been harvested near offshore oil rigs. Industrial/environmental sources of V include: processing of mineral ores, phosphate fertilizers, combustion of oil and coal, production of steel, and chemicals used in the fixation of dyes and print (Metals in Clinical and Analytical Chemistry, 1994). V is used in producing rust-resistant, spring and high speed tool steels. Vanadium pentoxide and other vanadates are used as catalysts in the production of sulfuric acid and formaldehyde. Urban air in industrialized areas may have higher levels of V than in rural areas.

Symptoms of V toxicity vary with chemical form and route of assimilation. Inhalation of excess V may produce respiratory irritation and bronchitis. Excess ingestion of V can result in decreased appetite, depressed growth, diarrhea/gastrointestinal disturbances, nephrotoxic and hematotoxic effects. Pallor, diarrhea, and green tongue are early signs of excess V and have been reported in human subjects consuming about 20 mg V/day (Modern Nutrition in Health and Disease, 8th edition, eds. Shils, M., Olson, J., and Mosha, S., 1994).

A confirmatory test for excess exposure to V is the Doctor's Data the whole blood vanadium test. EDTA (but not DMPS or DMSA) is an effective chelator of V. Therefore excessive retention (body burden) of V can be assessed by comparing pre- and post-Ca-Na2-EDTA urine V levels.