

# A case of medically unjustified treatment with multiple mega doses of vitamin C with thyroid hormones that caused serious adverse reactions in a woman

**Mohammed Ali Al-Bayati**

Ph.D., DABT, DABVT  
Toxicologist & Pathologist  
Toxi-Health International  
150 Bloom Dr.  
Dixon, CA 95620

Phone: +1 707 678 4484 Fax: +1707 678 8505  
Email: maalbayati@toxi-health.com Website: <http://www.toxi-health.com>

## Abstract

Christine, a 40-year-old white female, suffered suddenly from a fatigue syndrome in August 2000. Her blood and urine analyses, chest x-ray, and abdominal ultrasound exam were normal. She consulted with several physicians who treated her symptoms and she made progressive recovery in her health. In February 2003, she felt that she had about 80% of her energy back and she was working full time. However, she consulted with a physician in California to get her full energy back and he recommended a detoxification treatment plan with high doses of vitamin C, glutathione, minerals, and vitamins. He gave her 39 intravenous injections of vitamin C (10-35 g per injection), glutathione (0.4-2.0 g per injection), calcium, and other vitamins for about 10 months. In addition, he also treated her with therapeutic doses of levothyroxine and cytomel for more than a year, although she had normal thyroid functions.

Christine's clinical record indicates that she suffered from symptoms of hypercalcaemia and calciurea as a result of her treatment with vitamin C and calcium. The treatment with thyroid hormones also aggravated her condition and she became 100% disabled. I have not found any medical justification for the use of detoxification agents or the treatment with thyroid hormones in this case. Christine's blood, urine, and hair analyses revealed that she was not exposed to chemicals at her workplace or home or ingested toxic chemicals. In addition, Christine's blood thyroid hormones levels were within the normal range and she was not suffering from hypothyroidism.

**Note:** This case illustrates a practitioner's failure to appropriately utilize vitamin and drug products in the practice of medicine. It does not reflect in any way on the safety or on the appropriate use or levels of use of vitamin or drug products in a patient needing them. Dan Burton, Congressman representing the 5th District of Indiana, in a Congressional hearing reported. "106,000 people die a year from prescription drugs, ... with just 16 deaths reported last year [from using a dietary supplement]."

© Copyright 2007. Pearblossom Private School, Inc.—Publishing Division. All rights reserved.

*Keywords:* Calciurea, detoxification, fatigue syndrome, glutathione, hypercalcaemia, hypothyroidism, thyroid hormones, vitamin C

## I. Summary of the case and findings

Christine is a 40-year-old white female from California. She worked as a senior manager for a software company, and traveled a lot. She was in an excellent health and an extremely active and motivated businesswoman until shortly following a business trip to the Far East in July of 2000. In August of 2000, she developed chronic fatigue syndrome. Her blood and urine analyses, chest x-ray, and abdominal ultrasound exam were normal. Christine does not drink alcohol, smoke, or use illicit drugs.

Christine consulted with several physicians who treated her symptoms and she made progressive recovery in her health. In February 2003, she felt that she had about 80% of her energy back and she was working full time. However, she consulted with a physician in California (CPh) to get her full energy back and he recommended a detoxification treatment plan with high doses of vitamin C, glutathione, minerals, and vitamins [Table 1].

In about 10 months, CPh gave Christine 39 intravenous injections of post Myers solution that contains vitamin C (10-35 g per injection), glutathione (0.4-2.0 g per injection), calcium (100 mg/mL) and other ingredients [Table 1]. CPh also treated

Christine with therapeutic doses of levothyroxine and cytomel for more than a year, although she had normal thyroid functions [Tables 3 and 4]. Christine suffered from adverse reactions to these treatments and became a 100% disabled.

Christine consulted with me to review her medical records and case history and to find the possible cause(s) that led to her disability. She also wanted to know about the medical validity of the treatments given by CPh in her case. I reviewed Christine's medical records and the pertinent literature and my investigation revealed the following:

1. Christine's physician (CPh) treated her with high doses of vitamin C, glutathione, calcium, and magnesium IV for about 10 months [Table 1] and his objective was to detoxify her body from toxin(s). My review of Christine's medical records did not lead me to any toxicology-screening tests that CPh did prior to starting his detoxification treatment to indicate that Christine was suffering from acute or chronic intoxication to chemicals. For example, measuring the levels of certain metals and other toxins in Christine's blood, urine, and other biological samples. In addition, my investigation of this case did not reveal that she was exposed to toxic agent(s) in her workplace or at home.

As a toxicologist, I find it medically unjustified that a healthcare provider treats an individual in non-emergency settings with high doses of alleged antidotes for several months without knowing the target toxin(s). My opinion is also supported by the published medical literature in the fields of human toxicology, pharmacology, and medicine [Section 4]. In this case, CPh's plan lacks the specific objectives of achieving his detoxification target and the methodologies to monitor the effectiveness and the efficacy of his treatments.

2. Christine's urine analysis for the presence of 14 chemical agents performed at five days following the starting of CPh's detoxification treatment revealed that the levels of most metals were undetectable or within the reference ranges [Table 7]. Christine was treated with a chelating agent (DMPS) prior to giving a urine sample. The results of her urine test indicate that Christine's body burden of heavy metals was very low. However, CPh continued his detoxification treatment for an additional nine months, even though Christine was suffering from adverse reactions to his treatment with the detoxification agents.

3. Christine's hair analysis for the presence of toxic chemical agents performed at about three weeks following the starting of CPh's detoxification treatment indicated that Christine did not suffer from exposure to toxic levels of metals [Table 8]. However, CPh continued his detoxification treatment for about nine months, even though Christine was suffering from adverse reactions to his treatment.

4. Christine suffered from confusion, forgetfulness, memory loss, gastrointestinal problems, and muscle weakness after receiving the first IV detoxification treatment. Her illness became worse with each additional IV treatment. However, instead of stopping the treatment, CPh increased the dosage. Christine's hair analysis on March 19, 2003 revealed high calcium levels of 2460 µg calcium per gram of hair, which is more than three times the normal average value [Table 5]. Christine's clinical tests and symptoms show that she was probably suffering from hypercalcemia and hypercalciuria [Table 6]. The estimated 24-urine volumes for Christine on March 2, 2003 and November 19, 2003 were 6.8 L and 8.0 L, respectively. Polyuria is one of the clinical signs of hypercalcemia and hypercalciuria.

5. CPh treated Christine with levothyroxine (50 µg/day) and cytomel (10 µg/day) on March 13, 2003 through October of 2004 [Table 3], although her thyroid was producing hormones within the normal range as shown by all the tests performed during the four years prior and during the treatment with thyroid hormones [Table 4]. I have not found any medical justification for the treatment of an individual with normal thyroid functions with therapeutic doses of two thyroid hormones. Furthermore, CPh continued his treatment with these hormones, even though; Christine appeared to be suffering from adverse reactions to these hormones.

6. Christine did urine and hair analyses for heavy metals and other elements in November 2003. Both of these analyses show that Christine did not have abnormal levels of toxic metals and other elements in urine and hair [Tables 7 and 8]. However,

CPh continued his detoxification treatment for additional two months. In November of 2003, Christine was very sick and she was completely disabled as a result of receiving CPh's detoxification treatment.

The clinical data described above indicate that Christine's physician (CPh) did not follow sound medical protocol when he started to treat her with high doses of vitamin C and other agents for about a year. The following general scientific rules should be followed in any detoxification process dealing with the neutralization and/or removing toxin from the body of an individual exposed to toxin(s): (1) know the type and the levels of toxin(s) in blood, tissues, and/ or urine that needs to be neutralized and/or removed from the body; (2) choose a specific detoxifying agent that has been scientifically tested to be effective and it has insignificant toxicity; (3) assess the benefit and the risk of the detoxification procedure prior to use of any detoxifying agent(s); (4) monitor the levels of the toxin(s) in blood, urine, and other compartments of the body during the treatment period with the detoxifying agent(s) to evaluate the efficiency of the detoxification agent(s) and the process; and (5) monitor the adverse reactions of the agent(s) used in the detoxification process. I believe that an individual may suffer from serious illnesses and even death if the treating physician does not follow these scientific rules.

## **2. Christine's illness history and treatments received prior to receiving the detoxifying agents from her California physician (CPh)**

Christine obtained MBA degree from UC-Berkeley, worked as a senior manager for a software company, and traveled a lot. She was in an excellent health and an extremely active and motivated businesswoman until shortly following a business trip to the Far East in July of 2000. In August of 2000, she developed chronic fatigue syndrome. Christine does not drink alcohol, smoke, or use illicit drugs.

Christine consulted with her physician on September 6, 2000 about her fatigue. In September 21, 2000, her fatigue got worse and she was put on short disability for five months. She did not work from September 21, 2000 to February 20, 2001. Christine experienced fatigue/exhaustion, muscles pain, and nocturnal insomnia.

Christine was treated with antidepressant, analgesic, and anti-inflammatory drugs to control her fatigue, associated depression, and muscle pain. She was also given Sinequan to be used at bedtime to help her sleep. At certain time, she suffered from allergic rhinitis and she was treated with short courses of Flonase and other corticosteroid compounds. She developed yeast infection on one occasion and she was treated with Diflucan and felt better within two weeks.

Christine felt better and returned to work a part-time in February 2001. In the Fall 2001, she started to work full-time until April 2003. However, she suffered from mild fatigue during that time. Also, she had Hepatitis-A infection from contaminated food on a business trip in September of 2002.

Christine had several blood tests performed in 2000 through 2002 and her results were normal, except for slightly elevated serum liver enzymes levels on September 12, 2002 as a result of her hepatitis-A infection. Also, she had four urine analyses per-

formed on December 13, 1999 through November 4, 2002 and her results were normal.

In addition, her chest x-ray and abdominal ultrasound were also normal. She did not suffer from organomegaly.

In addition, Christine had seven physical exams between July 21, 2001 and February 11, 2003 and her heart was normal (regular rate and rhythm without murmurs, rubs, or gallops). On February 11, 2003, Christine had a body weight of 128 ½ pounds, blood pressure of 118/71 mmHg, and pulse of 74 per minute.

In addition, Christine had thyroid functions test performed on January 19, 2001 and revealed that her serum levels of T-4 (1.0 ng/dL) and TSH (2.24 mIU/L) were with normal range. Her serum TSH was also measured on September 12, 2002 and it was with the normal range (1.76 mIU/L).

Christine did not use illicit drugs, smoke, or drink alcohol. She was tested for the use of illicit drugs on November 4<sup>th</sup>, 2002 and the result of her urine test was negative. She was tested for the presence of barbiturates; benzodiazepines; cocaine metabolites; opiate metabolites; phencyclidine; and amphetamine.

Christine's health problems showed a significant gradual improvement in February 2001 through February 2003. In February of 2003, Christine had recovered about 80% of her health and since she traveled a lot for work desired 100% of her health. She then sought an alternative treatment to help her to become healthier. She consulted with a California physician (CPh) who recommended a detoxification program and thyroid hormones therapy to make her feel better.

### 3. Treatments given to Christine by her California physician (CPh) and adverse reactions

#### 3.1. Treatments given by CPh

CPh treated Christine on February 26, 2003 through January 7, 2004 with IVs injections of Post Myers solution and glutathione [Table 1]. Post Myers IV solution consist of vitamin C (500 mg/ml); cyanocobalamin (100 µg/mL); pantothenic acid (250 mg/ml); pyridoxine (100 mg/ml); vitamin B. complex, (100 mg/mL); calcium (100 mg/ml); magnesium sulfate (500 mg/mL) [8,9]. Christine received a total of 39 injections of vitamin C in a period of 10-1/2-month (up to 35 g per treatment). Also CPh gave Christine five IV injections of phosphatidylcholine in 2004 to treat her possible neurodegenerative disorder [Table 2].

Furthermore, CPh treated Christine with levothyroxine (50 µg/day) and cytomel (10 µg/day) on March 13, 2003 through March of 2004 [Table 3]. Although, her thyroid was producing hormones within the normal range as shown by all the tests performed during the last four years. The blood levels of T3-free, T4-free, total thyroxine, and TSH were measured and found to be within the normal range [Table 4]. The average TSH value for Christine was 1.58 mIU/L (n=7), which is similar to the mean values in disease-free population in the USA and Europe [10, 11]. Her T3 uptake percentage was also normal.

**Table 1. Christine's treatment with vitamin C and other agents IV**

Treatment Date (2003)	Days between Treatments	IV Solution Given	Vitamin C Injected (g)	Glutathione injected (mg)
02/26	00 <sup>a</sup>	Post Myers <sup>b</sup>	10	400
03/12	16	Post Myers	10	400
03/24	12	Post Myers	10	400
04/04	11	Post Myers	10	400
04/18	14	Post Myers	12	400
04/28	10	Post Myers	12	400
05/02	05	Post Myers	12	400
05/06	04	Post Myers	12	400
05/08	2	Post Myers	20	400
05/12	5	Post Myers	20	400
05/15	3	Post Myers	25	400
05/20	5	Post Myers	25	400
05/30	10	Post Myers	25	400
06/10	11	Post Myers	25	600
06/17	7	Post Myers	35	800
06/30	13	Post Myers	30	800
07/07	7	Post Myers	25	800
07/22	15	Post Myers	15	800
08/06	13	Post Myers	15	800
08/19	12	Post Myers	20	1000
08/27	8	Post Myers	18	1400
09/04	8	Post Myers	18	1600
09/12	6	Post Myers	18	1600
09/16	4	Post Myers	18	1800
09/23	7	Post Myers	18	1900
09/30	7	Post Myers	18	1800
10/08	8	Post Myers	18	1600
10/14	6	Post Myers	20	1900
10/21	7	Post Myers	20	1800
10/27	7	Post Myers	20	1800
11/04	7	Post Myers	20	1900
11/11	7	Post Myers	20	1900
11/20	9	Post Myers	20	2000
11/21	1	Post Myers	20	2000
12/02	10	Post Myers	20	1900
12/11	9	Post Myers	25	1800
12/19	8	Post Myers	20	1800
12/30	11	Post Myers	20	1900
01/07	8	Post Myers	25	1900

<sup>a</sup>First Injection

<sup>b</sup>Post Myers IV solution contains vitamin C (500 mg/ml); cyanocobalamin (100 µg/mL); pantothenic acid (250 mg/ml); pyridoxine (100 mg/ml); vitamin B. complex, (100 mg/mL); calcium (100 mg/ml); magnesium sulfate (500 mg/mL) [8, 9].

**Table 2. Christine's treatment with phosphatidylcholine and glutathione IV**

Treatment date	Days between Treatments	Phosphatidylcholine Dose (mg)	Glutathione Dose (mg)
01/28/04	20*	5000	2000
02/11/04	14	5000	2000
02/18/04	7	5000	2000
02/25/04	7	5000	2000
03/16/04	18	5000	2000

\* Treatment with glutathione on January 7, 2003 [Table 1].

**Table 3. Doses of thyroid hormones given to Christine (2003-2004)**

Prescription date	Medications	Dose (µg/day)
03/13/2003	Levothyroxine (Unithroid)	50
05/06/2003	Cytomel, T3 (Liothyronine)	10
05/12/2003	Levothyroxine (Unithroid)	50
06/17/2003	Cytomel, T3 (Liothyronine)	10
06/18/2003	Levothyroxine (Unithroid)	50
08/23/2003	Levothyroxine (Unithroid)	50
08/27/2003	Cytomel, T3 (Liothyronine)	10
11/01/2003	Levothyroxine (Unithroid)	50
12/29/2003	Levothyroxine (Unithroid)	50
02/11/2004	Levothyroxine (Unithroid)	50
03/13/2004	Levothyroxine (Unithroid)	50
03/16/2004	Cytomel, T3 (Liothyronine)	10

**Table 4. Christine's thyroid functions tests results (2001-2004)**

Date	T3, free (pg/dL)	T4, free (ng/dL)	TSH (mIU/L)	T3 up-take %	T4 (Thyroxine total) (µg/dl)
01/19/2001	- <sup>a</sup>	1.0	2.24	-	-
09/12/2002	-	-	1.76	-	-
02/21/2003	299	1.2	1.31	28	6.9
07/21/2003	270	1.2	1.18	-	-
11/03/2003	260	1.1	1.71	-	-
01/27/2004	-	-	1.83	-	-
10/19/2004	320	-	1.04	-	8.0
Average	287	1.1	1.58	28	7.5
Reference range	230-420	0.8-1.8	0.4-5.5	22-35	4.5-12.5

<sup>a</sup>Not measured.

### 3.2. Christine's adverse reactions to treatments given by CPh

After receiving the first IV treatment of CPh's detoxification agents on February 26, 2003 [Table 1], Christine's ability to concentrate at work became extremely diminished and her fatigue and her other health problems became worse. Her muscle weakness increased and she suffered from confusion, forgetfulness, and memory loss. Her gastrointestinal problems become much worse than ever and she was forced to work from home. She was also unable to go out to eat. Her health condition became worse with each additional IV treatment with high doses of vitamin C, glutathione, and Post Myers solution [Table 1].

Christine's treatment with levothyroxine and cytomel aggravated her symptoms [Table 3]. Although Christine repeatedly asked her CPh for reasons for her severe negative reactions, his only response was that it was part of the detoxification process.

In April of 2003, Christine's fatigue became so unbearable that she could not even get out of bed. It happened after receiving the fifth IV injection of the detoxification agents [Table 1] and a month of treatment with the thyroid hormones [Table 3]. She was unable to walk and she communicated using the walkie-talkie from her bed to someone in the kitchen preparing her food.

CPh put Christine on full time disability in April of 2003. He described Christine's health condition in his letter dated June 20, 2003 to her insurance company as follows:

*"Because of the severe fatigue, memory loss, fainting spells and insomnia she was placed on disability. Her condition stabilized during the next several weeks. On April 29<sup>th</sup> she was examined for increased abdominal cramps and pain, severe fatigue, dysuria and syncopal episodes. Further testing revealed an acute E. coli urinary tract infection. The patient was treated with a course of Cipro for ten days. A subsequent overgrowth of yeast was treated with Nystatin.*

*Presently, she continues on a program to improve her digestion, nutrition, and detoxification. She continues to be extremely fatigued. Several times in my office she almost has passed out from exhaustion and abdominal pain. She is so physically exhausted that she is unable to perform even the daily tasks of homework, cooking or laundry. She has to hire a part-time caregiver to assist her at home. Her memory and thinking are extremely compromised and she is not able to perform the mental tasks associated with her work. Due to patient's debilitated condition she can tolerate only limited therapy and her prognosis for significant improvement in the near future is limited. The patient should be considered completely disabled."*

As indicated above in April of 2003, Christine was diagnosed as having a kidney infection and she was treated with Cipro. Her urine culture for bacteria showed an Escherichia Coli count of 100,000 organisms per mL of urine or greater. Christine stated that she developed very severe pain after eating that forced her to lie down. She also had her first convulsion in her life and she suffered from severe headaches. Headaches were so rare for Christine that she had only taken aspirin less than ten times in the last twenty years of her life.

Christine's weight dropped from 128 pounds in January 2003 to 108 pounds in late April 2003. She stated that she has never lost so much weight in her life. Yet, she made every effort on her part to maintain the weight that she has had over the last decade (i.e., 125 to 132 pounds). In addition, her insomnia had gotten so bad that she could sleep only three to four hours per night despite being in bed over 10 hours per night. She would wake almost every hour in extreme pain. She also stated that her heart would race at night and her liver (upper right side) would hurt.

In May 2003, Christine got a cold feeling after receiving the IVs and her temperature dropped to 92.3 degrees. In addition,

she suffered from severe pain and fell over after receiving the IVs. On three occasions in CPh's office, Christine needed to smell salts to be revived. Christine was so physically exhausted that she was unable to perform even the daily tasks of housework such as cooking and laundry. She had to have a part-time caregiver to assist her at home. Her memory and thinking were extremely compromised and she was not able to perform the mental tasks associated with her work. She found it difficult to perform such as simple tasks as paying her bills.

In her doctor's visit of February 2, 2004, Christine noted that she still cannot sleep for more than six hours per night and she tolerated only two hours of activity per day. She left the house only for necessary errands such as grocery shopping and doctor's visit. She had approximately three bad days per week when she was either bedridden or housebound due to fatigue and ill health.

Furthermore, Christine stated that at times, she became paralyzed with pain, can barely talk, and was fed in bed. Her head must be held up in order to drink water. After taking a walk or do anything active, she would start sweating, her eyes would glaze over and then she would black out and go unconscious for a while. Sometimes daily to bi-weekly, Christine would fall over holding her abdomen from gripping pain. In addition, her body temperature would fluctuate from 94 degrees to 99 degrees and in reverse within a 30-minute period. In February to March 2004, Christine required over 60 enzymes a day in order to digest her food and would lie in bed in severe pain holding her abdomen after eating. She also had a lot of hair loss.

Christine's physician (CPh) put her on full time disability in April of 2003 and is still disabled although she is recovering slowly. He described Christine's health condition in his letter of February 2004 to the Department of Social Services as follows:

*"She continues to be extremely fatigued. Several times in my office she has nearly passed out from exhaustion and abdominal pain. She has been so physically exhausted that she is unable to perform even the daily tasks of homework, cooking or laundry. She has had to hire a part-time caregiver to assist her at home. Her memory and thinking are extremely compromised and she is not able to perform the mental tasks associated with her work. Until recently she found it difficult to perform such as simple tasks as paying her bills."*

*On her most recent visit (2/2/04) she notes that she can still not sleep for more than six hours per night; she tolerates two hours of activity per day; she leaves the house for necessary errands such as grocery shopping and doctor's visit three times per week; and she can only tolerate walking up to six blocks per day. She has approximately three bad days per week when she is either bedridden or housebound due to fatigue and ill health."*

*Furthermore, CPh stated that due to patient's debilitated condition she can tolerate only limited therapy and her prognosis for significant improvement in the near future is limited. The patient should be considered completely disabled and will not be able to return to work in any capacity for approximately six months. The patient is seen in the office every 4-6 weeks. She is not a candidate for job retraining."*

Christine reported that she asked her physician (CPh) on several occasions to explain to her the causes of her suffering from the adverse reactions to his treatments. She stated that his response was that is part of the detoxification process and she had a weak liver. He was also adamant that she continued her treatment or she would never get better. He saw her for a total of 24 office visits from February 2003 to October 2004 for a total of approximately 14 hours. She was not satisfied with CPh's explanation and decided to stop taking the IV's treatment. Within months of not receiving any more IVs treatment from CPh, Christine's need for enzymes to digest her food decreased from over 60 enzymes down to eight enzymes a day and eventually none. Her ability to move and to do work around the house improved. She started to cook her own meals and went grocery shopping.

#### **4. Possible explanations for Christine medical problems caused by CPh's treatments**

Christine consulted with me as a toxicologist and pathologist to review her medical records and case history. She is trying to find the possible scientific and medical answers for the following questions: (1) Did she suffer from adverse reactions to her physician's treatment with high doses of vitamin C, calcium, magnesium, and post Myers's solutions? (2) Did she suffer from adverse reactions to the treatment with levothyroxine and cytomel? (3) Are there medical justifications for the treatments stated in questions 1 and 2?

My review of Christine's medical records and the pertinent medical literature revealed that Christine's adverse reactions to CPh's treatment regimen could be explained by at least three main mechanisms. These include: (1) The development of hypercalcemia and hypercalciurea resulted from her treatment with high doses of vitamin C and calcium; (2) the conversion of ascorbic acid to oxalate in kidney; (3) the adverse reactions to her treatment with levothyroxine and cytomel [Table 3].

##### **4.1 The development of hypercalcemia and hypercalciurea**

Christine was given a 39 injection (IV) of high doses of vitamin C (10-35 g per injection) in 10.5 months [Table 1]. High doses of vitamin C can cause decalcification of the bone and mobilize other elements deposited in bone such as metals. Table 5 contains the results of Christine's hair analyses performed in 2003 and 2004. The first analysis was done on March 19, 2003, which was one week following the second IV injection of vitamin C [Table 1]. The calcium concentration in Christine hair was about 3.28 times higher than the average value in normal person.

I believe that the excess calcium in Christine's hair was resulted from the mobilization of calcium from her bone by vitamin C and the calcium present in Post Myers IV solution (100 mg/ml) given to Christine. The level of magnesium in Christine's hair was also elevated (2.3 times higher than the average concentration in normal individual), which was resulted from her treatment with magnesium. Post Myers IV solution contains 500 mg magnesium sulfate per mL.

Tsao *et al.* collected urine samples from 46 healthy subjects during a period of eight hours after the ingestion of 2 g of ascorbic acid. A significant increase in mean urinary calcium

excretion from 48.2 +/- 25.1 mg to 58.3 +/- 28.0 mg in the 8-h time period was observed. They concluded that ascorbic acid has a short-term effect on the regulation of the absorption and metabolism of calcium in humans [12]. Christine was given doses of vitamin C by IV injection that were 5 to 17.5 times higher than those given to the individuals treated in this study [Table 1].

The mobilization of calcium from bones by the high doses of vitamin C and the treatment with high doses of calcium IV caused Christine to suffer from hypercalcemia and hypercalciuria. The levels of calcium in Christine's blood and urine were not measured post her treatments with vitamin C and calcium. However, her symptoms, the concentrations of calcium in her hair, and her urine measurements indicated that she was suffering from hypercalcemia and hypercalciuria [Tables 5 and 6].

Christine's urine measurements presented in Table 6 show that she suffered from polyurea. Her estimated 24-hour urine volumes collected on Mar. 2 and Nov. 19 of 2003 were 6.8 and 8.0 L, respectively. These two measurements were taken following the first and the 33 injections with high doses of vitamin C and calcium, respectively [Tables 1 and 6]. A person will be considered to suffer from a polyurea, if he or she has a 24-hour urine output more than 3 L. Hypercalcemia usually causes reversible nephrogenic diabetes insipidus due to the reduction in water reabsorption along the distal nephron [6:261-2].

Miller and Stapleton measured the urinary volume in 24-hour urine collections in 50 children with hypercalciuria and urolithiasis or hematuria and 36 healthy children. Urinary volume was 22.2 +/- 2.0 ml/kg/day in healthy children and 25.4 +/- 2.0 ml/kg/day in children with hypercalciuria [13].

Christine received her last injection of vitamin C and calcium on Jan. 7, 2004 from CPh [Table 1]. Her average volume of the 24-hour urine collected in Nov. of 2004 and Feb. of 2005 was 2.62 L, which was within the normal range, when receiving treatment from an Illinois physician [Table 6]. These data indicate that Christine was suffering from hypercalciuria as a result of her treatment with high doses of vitamin C and calcium.

The symptoms of hypercalcemia may include anorexia, nausea, vomiting, constipation, hypotonia, depression, and occasionally lethargy and coma. Hypercalcemia per se alters renal function in addition to the pathologic effects of calcium phosphate deposition [6:2217]. Christine suffered from some of these symptoms described above.

**Table 5. Abnormal concentration ( $\mu\text{g/g}$ ) of elements in Christine's hair**

Elements <sup>a</sup>	( $\mu\text{g/g}$ ) on 03/19/03	( $\mu\text{g/g}$ ) on 11/13/03	( $\mu\text{g/g}$ ) on 11/15/04	Reference range ( $\mu\text{g/g}$ )
calcium	2460	1640	2290	300-1200
magnesium	280	200	150	35-120
cadmium	0.45	<0.009	0.021	<0.1
lead	4.1	0.15	0.16	<1.0
mercury	1.7	0.45	0.60	<1.0
strontium	14	7.3	4.6	0.5-7.6
titanium	1.1	0.77	0.75	<1.0

<sup>a</sup>Hair analyses were performed by Doctor's Data Inc. Sample size: 0.198 g.

**Table 6. Christine's urine volume and creatinine concentrations, 2003-2005**

Measurements	Values on 03/02/03	Values on 11/19/03	Values on 11/15/04	Values on 02/24/05	Reference Range
Urine volume collected (mL)	1700	2000	2300	2400	2000-3000 [6]
Collection Duration (hr)	6	6	24	24	24
Collection rate (mL/hr)	283	333	96	100	83-125
Volume (mL) Per 24 hr	6800	8000	2300	2400	2000-3000 [6]
Creatinine (mg/dL)	18	17	55	55	60-160
Creatinine (mg/24 hr)	1224	1360	1260	1325	1000-1600 [6]

#### 4.2 Conversion of vitamin C to oxalate and kidney damage

The second adverse reaction to the treatment with high doses of vitamin C is the conversion of vitamin C to oxalate. High level of oxalate can enhance stone formation in kidney and causes kidney damage and infection as shown in the studies conducted in humans described below. Christine suffered from kidney infections and severe pain during her course of treatment with high doses of vitamin C [Table 1].

Hatch et al. measured serum and urinary oxalate in 9 normal subjects, ingested 8 g of ascorbic acid daily. Serum oxalate levels increased to 310% of control values during supplementation. Urinary oxalate gradually increased during ascorbate intake and 7 days post cessation of ascorbate, rose unexpectedly for all subjects into the hyperoxaluric range [14]. The doses of vitamin C given to Christine was up to 4 times higher than those given to the subjects in this study [Table 1].

Furthermore, Urivetzky et al. conducted study with a total of 15 subjects who received 0 (placebo), 100, 500, 1,000 or 2,000 mg of ascorbic acid on days 2 and 3 postoperatively. These subjects had unilateral nephrostomy tubes after extracorporeal shock wave lithotripsy. Before and after administration of ascorbic acid, successive 6-hour urine specimens were collected from the nephrostomy tube and from the contralateral kidney directly into a preservative to stabilize ascorbic acid and oxalate. At doses of 500 mg or more of ascorbic acid there was a statistically significant increase in urinary oxalate equivalent to 1.2 to 1.8% of the millimoles of ascorbate administered. This represented an increase in urinary oxalate excretion of 6 to 13 mg per day per 1,000 mg of ascorbic acid supplement. This amount would increase the risk of calcium oxalate urolithiasis [15]. Christine was given doses of vitamin C up to 35 g per IV injection [Table 1].

In addition, Baxmann et al. evaluated forty-seven adult calcium stone-forming patients received either 1 g (n=23) or 2 g (n=24) of vitamin C supplement for 3 days and 20 healthy subjects received 1 g. A 24-hour urine sample was obtained before and after vitamin C for calcium, oxalate, magnesium, citrate, sodium, potassium, and creatinine determination. The Tiselius index was used as a calcium oxalate crystallization index. A significant increase in mean urinary oxalate was observed in

calcium stone-forming patients receiving either 1 g (50 +/- 16 vs. 31 +/- 12 mg/24 hours) or 2 g (48 +/- 21 vs. 34 +/- 12 mg/24 hours) of vitamin C and in healthy subjects (25 +/- 12 vs. 39 +/- 13 mg/24 hours).

A significant increase in mean Tiselius index was observed in calcium stone-forming patients after 1 g (1.43 +/- 0.70 vs. 0.92 +/- 0.65) or 2 g vitamin C (1.61 +/- 1.05 vs. 0.99 +/- 0.55) and in healthy subjects (1.50 +/- 0.69 vs. 0.91 +/- 0.46). These data suggest that vitamin C supplementation may increase urinary oxalate excretion and the risk of calcium oxalate crystallization in calcium stone-forming patients [16].

Furthermore, Auer *et al.* evaluated a male subject who developed hematuria and calcium oxalate crystalluria after ingestion of large doses of ascorbic acid for 8 consecutive days. Twenty-four-hour urine samples were collected before and during the ascorbic acid ingestion period as well as after the detection of hematuria. Oxalate excretion increased by about 350% during ascorbate ingestion before hematuria. Increasing calcium excretion was accompanied by decreasing potassium and phosphate values. The calcium oxalate relative supersaturation and Tiselius risk index increased during vitamin C ingestion and large aggregates of calcium oxalate dihydrate crystals were observed by scanning electron microscopy immediately after the detection of hematuria [17].

They concluded that the high percentage metabolic conversion of ascorbate to oxalate in this subject caused relative hyperoxaluria and crystalluria, the latter manifesting itself as hematuria. They stated that clinicians need to be alerted to the potential dangers of large dose ingestion of vitamin C in some individuals [17]. Christine suffered from severe pain and developed kidney bacterial infections in April of 2003 following a few weeks of receiving the vitamin C treatment.

Also, it has been found that the level of oxalate in urine was increased following the treatment of individuals with therapeutic doses of vitamin C by IV injection. Pena de la Vega *et al.* compared the urinary oxalate excretion level in thirteen patients who had no history of nephrolithiasis and had received 100 mg or 200 mg vitamin C IV per day. Each participant provided a 24-hour urine sample for oxalate determination on the vitamin C dose (100 mg/d), and again after at least 1 month on the increased vitamin C dose (200 mg/d). Urinary oxalate excretion increased on the 200-mg vitamin C dose, from 0.34 +/- 0.13 to 0.44 +/- 0.17 mmol/d (mean increase = 0.10 mmol/d;  $p = .04$ ; 95% confidence interval). They concluded that in therapeutically used doses, IV vitamin C increases urinary oxalate excretion, potentially predisposing susceptible individuals to nephrolithiasis [18]. The doses of vitamin C given to Christine by IV injection were many folds higher than those given to the subjects in this study [Table 1].

### 4.3 Levothyroxine and cytomel treatments and adverse reactions

Christine was treated with levothyroxine (50 µg/day) and cytomel (10 µg/day) on March 13, 2003 through March of 2004 [Table 3]. Although, her thyroid was producing hormones within the normal range as shown by all the tests performed during the last four years. Her blood levels of T3-free, T4-free, total thyroxine, and TSH were measured and found to be within

the normal range [Table 4]. Her T3 uptake percentage was also normal.

The average TSH value for Christine was 1.58 mIU/L ( $n=7$ ), which is similar to the mean values in disease-free population in the USA and Europe. In the USA, the mean serum TSH was 1.50 (95% confidence interval, 1.46-1.54) mIU/L as determined in a study using 16,533 healthy subjects [10]. In Norway, the average values for the TSH in blood of females and males were 1.80 (mU/l) and 1.50 (mU/l), respectively [11].

The US Centers for Disease Control conducted study in the United States of America that included 16,533 people who did not report thyroid disease, goiter, or taking thyroid medications (disease-free population). The mean serum TSH was found to be 1.50 mIU/liter (95% confidence interval, 1.46-1.54 mIU/L). It was higher in females than males [10].

Furthermore, Bjoro *et al.* examined the prevalence of thyroid disease and dysfunction including thyroid autoimmunity in Norway. All inhabitants 20 years and older (94009) in Nord-Trondelag were invited to participate in a health survey with a questionnaire and blood samples. In individuals without a history of thyroid disease, the TSH levels were 0.49-5.70 mIU/L for females and 0.56-4.60 mIU/L for males. The average values for the TSH in blood of females and males were 1.80 (mIU/L) and 1.50 (mIU/L), respectively [11].

In Christine's case, the blood levels of antibodies against thyroglobulin and thyroid peroxidase were measured in February of 2003 and January of 2004. The level of thyroglobulin antibody was found within the normal range. However, the thyroid peroxidase antibody was found to be elevated (58-159 IU/mL), which is a common finding in healthy people and has no clinical significant in health people. For example, Bjoro *et al.* conducted a health survey study in all inhabitants 20 years and older (94009) in Nord-Trondelag. They found that the prevalence of positive TPOAb (>200IU/ml) was 13.9% in females and 2.8% in males [11].

The adverse reactions associated with levothyroxine therapy are primarily those of hyperthyroidism due to their overdosage. They may include the following: (1) general symptoms (fatigue, increased appetite, weight loss, heat intolerance, fever, and excessive sweating); (2) central nervous system disturbance (headache, hyperactivity, nervousness, anxiety, irritability, emotional problem, and insomnia); (3) musculoskeletal problems (tremors and muscle weakness); (4) gastrointestinal tract disturbance (diarrhea, vomiting, and abdominal cramps); (5) cardiac disturbance (palpitation, tachycardia, arrhythmias, increased pulse and blood pressure, angina, infection, and heart failure); (6) dyspnea; (7) reproductive disturbance (menstrual irregularities, and infertility); (8) hair loss; and (9) hypersensitivity reactions [6,7,19,20].

In addition, the adverse reactions of cytomel in some individuals taking this medication may include heart palpitation, trembling, irregular heartbeat, agitation, shortness of breath, excretion of sugar through the urine, excessive perspiration, diarrhea, weight loss, and psychic disorder. Christine suffered from some of these symptoms described in individuals suffering from adverse reactions to high levels of thyroid hormones.

## 5. Testing the scientific validity of Christine's physician's therapeutic approaches used in Christine's case

In February of 2003 through January 2004, Christine was treated with high doses of vitamin C, glutathione, calcium, and magnesium IV [Table 1]. Christine's physician's objective was to detoxify her body from toxin(s). My review of Christine's medical records did not lead me to any toxicology-screening test that her physician did prior to starting his detoxification treatment. For example, proper protocol includes measuring the levels of certain metals and other toxins in Christine's blood, urine, and/or any other biological samples before engaging in a treatment. In addition, my investigation of this case did not reveal that she was exposed to toxic agent(s) in her workplace or at home.

Plus if a treatment is started, that treatment should be periodically monitored to evaluate progress or in this case, why the patient became extremely ill.

As a toxicologist, I find it medically unjustified that a healthcare provider treats a patient in non-emergency settings with high doses of antidotes for several months without knowing the target toxin(s). My opinion is also supported by the published medical literature in the fields of human toxicology, pharmacology, and medicine [1-7]. In this case, his plan lacks the specific objective of achieving his detoxification target and the methodologies to monitor the effectiveness and the efficacy of his treatment.

Furthermore, Christine was treated with levothyroxine (50 µg/day) and cytomel (10 µg/day) on March 13, 2003 through March of 2004 [Table 3]. Although, her thyroid was producing hormones within the normal range as shown by all the tests performed during the last four years [Table 4]. I have not found any medical justification for the treatment of a normal person with therapeutic doses of two thyroid hormones [1-7]. Furthermore, Christine's physician (CPh) continued his treatment with these hormones, even though; she was suffering from the adverse reactions to these hormones.

Below is a list of additional specific clinical observations that support my concern about CPh's treatment plan used in Christine's case:

1. Christine did a urine analysis for the presence of toxic elements at five days following the starting of CPh's detoxification treatment. The levels of most metals were undetectable or within the reference ranges. Christine was treated with chelating agent (DMPS) prior to giving urine sample [Table 7]. The results of this test indicate that the majority of Christine body burden of heavy metals and other toxicants was low. However, CPh's continued his detoxification treatment for additional nine months, even though; Christine was suffering from severe adverse reactions to this treatment.

**Table 7. The concentrations of elements detected in Christine's urine post the use of provoking agent (DMPS)**

Elements	µg/g creatinine <sup>a</sup> on 3/02/2003	µg/g creatinine <sup>a</sup> on 11/19/2003	Reference range <sup>b</sup> µg/g creatinine
aluminum	< detection limit	< detection limit	<35
antimony	< detection limit	< detection limit	<1
arsenic	33	40	<130
beryllium	< detection limit	< detection limit	<0.5
bismuth	0.6	< detection limit	<15
cadmium	1.1	< detection limit	<2
lead	3.5	< detection limit	<18
mercury	32	5.6	<4
nickel	< detection limit	< detection limit	<12
platinum	< detection limit	0.5	<1
thorium	0.3	0.3	<0.8
tin	18	4.6	<10
tungsten	< detection limit	< detection limit	<0.1
uranium	< detection limit	< detection limit	<0.2

<sup>a</sup>Method of analysis: ICP-MS; <dI: less than detection limit; provoking agent: Oral DMPS

<sup>b</sup>Values without the use of provoking agent.

2. Christine did hair analysis for the presence of toxic metals at about three weeks following the starting of CPh's detoxification treatment. The results of this test indicated that Christine did not suffer from exposure to metals and arsenic [Table 8]. However, CPh continued his detoxification treatment for about nine months more, even though; Christine was suffering from adverse reactions to his treatment.

**Table 8. The concentrations of elements detected in Christine's hair**

Elements	µg/g of hair on 03/19/2003	µg/g of hair on 11/13/2003	Reference range µg/g hair
aluminum	5.1	2.6	<7.0
antimony	0.09	0.01	<0.05
arsenic	0.037	0.079	<0.06
beryllium	<0.01	<0.01	<0.02
bismuth	0.026	0.019	<0.01
cadmium	0.45	<0.009	<0.1
lead	4.1	0.15	<1.0
mercury	1.7	0.41	<1.1
nickel	<0.45	0.19	<0.4
platinum	0.003	<0.003	<0.005
silver	0.25	0.06	<0.15
thallium	<0.001	<0.001	<0.01
thorium	<0.001	<0.001	<0.005
tin	0.22	0.05	<0.03
titanium	1.1	0.77	<1.0
uranium	0.017	0.074	<0.06

\*Method of analysis: ICP-MS; sample size: 0.198 g of hair (head)

3. After receiving the first IV detoxification treatment, Christine suffered from confusion, forgetfulness, memory loss, gastrointestinal problems, and muscle weakness. Her illness became worse with each additional IV treatment. However, CPh did not stop the treatment, but instead increased the dosage. Christine's clinical tests and symptoms show that she was suffering from hypercalcemia and hypercalciurea. Christine's hair analysis on March 19, 2003 revealed high calcium levels of 2460 µg calcium per gram of hair, which is more than three times the normal average value (Table 5). In addition, the estimated 24-urine volumes for Christine on March 2, 2003 and November 19, 2003 were 6.8 L and 8.0 L, respectively (Table 6). Polyurea is one of the clinical signs of hypercalcemia and hypercalciurea [6].

4. Christine did urine and hair analyses for heavy metals and other elements in November of 2003. Both of these analyses show that Christine did not have abnormal levels of toxic metals and other elements in urine and hair (Tables 7 and 8). However, CPh continued with his detoxification treatment for an additional two months (Table 1). In November of 2003, Christine was still very sick and she was completely disabled as a result of receiving CPh's detoxification treatment.

## 6. Conclusions

My investigation of this case revealed that Christine suffered from serious adverse reactions to her physician's treatments with high doses of vitamin C and calcium and other elements. I also believe that there is no medical justification for treating Christine with these agents. Furthermore, the treatment with therapeutic doses of levothyroxine and cytomel aggravated Christine's health problems caused by the detoxification agents used. In addition, Christine's thyroid has normal functions and I have not found a medical justification of treating a person with normal thyroid functions with therapeutic doses of levothyroxine and cytomel.

**Editorial Note:** This case illustrates a practitioner's failure to appropriately utilize vitamin and drug products in the practice of medicine. It does not reflect in any way on the safety or on the appropriate use or levels of use of vitamin or drug products in a patient needing them. Dan Burton, Congressman representing the 5th District of Indiana, in a Congressional hearing reported. "106,000 people die a year from prescription drugs, ... with just 16 deaths reported last year [from using a dietary supplement]."

## References

- [1] Casarett & Doull's. Toxicology. The basic science of poisons. 6th ed. Klassen CD, ed. McGraw-Hill, New York, 2001.
- [2] The Pharmacological Basis of Therapeutics (9th edition), Hardman JG, Gilman AG, Limbird LE, eds. McGraw-Hill, New York, 1995.
- [3] Principles and Methods of Toxicology. Editor: Hayes WA. 4<sup>th</sup> ed. Taylor & Francis, London, 2001.
- [4] Handbook on the toxicology of metals. Friberg L, Nordberg GF, Vouk VB, eds. 2<sup>nd</sup> ed., Vol. II. Elsevier, New York, 1986.
- [5] Handbook of Toxicologic Pathology. Haschek WM, Rousseaux CG, eds. Academic Press Inc. San Diego, CA, 1991.
- [6] Harrison's Principles of Internal Medicine. 14th ed. Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, Hauser SL, Longo DL, eds. McGraw-Hill, New York, 1998.
- [7] Harrison's Principles of Internal Medicine. 15th ed.. Braunwald E, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL, eds. McGraw-Hill, New York, 2001.
- [8] Gaby AR. Intravenous Nutrient Therapy: the "Myers' Cocktail." Available online at [http://www.mcguiffmedical.com/Customer\\_Knowledgemyers\\_cocktail\\_iv.htm](http://www.mcguiffmedical.com/Customer_Knowledgemyers_cocktail_iv.htm)
- [9] Nutrient Cocktail for Schizophrenia and Bipolar Disorder. Available online at <http://www.alternativementalhealth.com/articles/cocktail.htm>
- [10] Hollowell JG, Staehling NW, Flanders WD, Hannon WH, Gunter EW, Spencer CA, Braverman LE. Serum TSH, T(4), and thyroid antibodies in the United States population (1988 to 1994): National Health and Nutrition Examination Survey (NHANES III). *J Clin Endocrinol Metab.* 2002; 87(2):489–99.
- [11] Bjoro T, Holmen J, Kruger O, Midthjell K, Hunstad K, Schreiner T, Sandnes L, Brochmann H. Prevalence of thyroid disease, thyroid dysfunction and thyroid peroxidase antibodies in a large, unselected population. The Health Study of Nord-Trondelag (HUNT). *Eur J Endocrinol*, 2000; 143(5):639–47.
- [12] Tsao CS, Miyashita K, Leung PY. Effect of ascorbic acid on calcium elimination in humans. *J Nutr Sci Vitaminol (Tokyo)*, 1986;32(4):437–46.
- [13] Miller LA, Stapleton FB. Urinary volume in children with urolithiasis. *J Urol*, 1989; 141(4):918–20.
- [14] Hatch M, Mulgrew S, Bourke E, Keogh B, Costello J. Effect of megadoses of ascorbic acid on serum and urinary oxalate. *Eur Urol*, 1980; 6(3):166–9.
- [15] Urivetzky M, Kessaris D, Smith AD. Ascorbic acid overdosing: a risk factor for calcium oxalate nephrolithiasis. *J Urol*, 1992;147(5):1215–8.
- [16] Baxmann AC, De O G Mendonca C, Heilberg IP. Effect of vitamin C supplements on urinary oxalate and pH in calcium stone-forming patients. *Kidney Int*, 2003; 63(3):1066–71.
- [17] Auer BL, Auer D, Rodgers AL. Relative hyperoxaluria, crystalluria and haematuria after megadose ingestion of vitamin C. *Eur J Clin Invest*, 1998; 28(9):695–700.
- [18] Pena de la Vega L, Lieske JC, Milliner D, Gonyea J, Kelly DG. Urinary oxalate excretion increases in home parenteral nutrition patients on a higher intravenous ascorbic acid dose. *JPEN J Parenter Enteral Nutr*, 2004; 28(6):435–8.
- [19] Physicians' Desk Reference, 53<sup>rd</sup> ed., 1999. Medical Economics Company, Inc, Montavale, NJ, USA..
- [20] Physicians' Desk Reference, 57<sup>th</sup> ed., 2003. Published by Thomson PDR at Montavale, NJ, USA.