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The Pitfalls of Hair Analysis for Toxicants in Clinical Practice: Three Case Reports

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Hair analysis is used to assess exposure to heavy metals in patients presenting with nonspecific symptoms and is a commonly used procedure in patients referred to our clinic. We are frequently called on to evaluate patients who have health-related concerns as a result of hair analysis. Three patients first presented to outside physicians with nonspecific, multisystemic symptoms. A panel of analytes was measured in hair, and one or more values were interpreted as elevated. As a result of the hair analysis and other unconventional diagnostic tests, the patients presented to us believing they suffered from metal toxicity. In this paper we review the clinical efficacy of this procedure within the context of a patient population with somatic disorders and no clear risk factors for metal intoxication. We also review limitations of hair analysis in this setting; these limitations include patient factors such as low pretest probability of disease and test factors such as the lack of validation of analytic techniques, the inability to discern between exogenous contaminants and endogenous toxicants in hair, the variability of analytic procedures, low interlaboratory reliability, and the increased likelihood of false positive test results in the measurement of panels of analytes. *Key words*: **hair analysis, lead, mercury, toxicant.** *Environ Health Perspect* **110:433–436 (2002). [Online 12 March 2002]**
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Case Presentation

We present three separate cases in which patients came to our clinic believing that they needed treatment for metal toxicity. In each case, the patient's beliefs were due to the results of hair analysis and other diagnostic tests.

Case 1. Patient 1, a 41-year-old white female, came to our clinic because she was concerned about heavy metal intoxication, particularly mercury. She was an organizational consultant with a Ph.D. in organizational behavior, and she was a self-described "hands-on healer." She stated that she had been sick for the last 10 years, she constantly felt very tired and suffered from body aches and joint pain, she constantly felt as though she was getting the flu, and she had noticed significant deterioration in her mental functioning. Her past occupational history revealed no potential sources of mercury exposure; other possible sources of exposure included frequent seafood consumption and 12 mercury amalgam dental fillings. She reported that her past medical history was significant for depression, anxiety, and Wilson's syndrome. She did not drink alcohol to excess or smoke tobacco. Her medications were nefazodone, dextroamphetamine, estrogen/testosterone combination, testosterone, progesterone, and vitamins and supplements. A physical examination of the patient, including a neurologic examination, was within normal limits. The patient stated that she had no concern about environmental causes of her symptoms until a specialist in alternative medicine, who felt her symptoms suggested metal toxicity, ordered an elemental hair analysis, which screened for 34 different elements described as "toxic," "nutritional" and "other." The report did not indicate which method was used to measure these analytes in the hair. The hair analysis report indicated a mercury level of 2.79 ppm (the laboratory considered 0–0.95 ppm to be within normal limits). The same health care provider also ordered a urine test, which involved oral administration of 500 mg of *meso*-2,3-dimercaptosuccinic acid (DMSA) and urine collection over an unspecified period of time. This resulted in a urine mercury level of 11.5 μg/g of creatinine. The other metals collected in the urine were lead, arsenic, cadmium, and nickel. The laboratory reported that all metals in the urine were within normal limits, except mercury, which was elevated. The patient came to our clinic requesting treatment for mercury toxicity. Based on the hair analysis and provocative urine test, the patient was convinced that her symptoms were due to mercury toxicity. In our clinic, we ordered a blood test, which revealed a mercury blood level of 11 μg/L (1.1 μg/100 mL).

Case 2. Patient 2 was a 54-year-old white female who presented for an evaluation of elevated mercury levels. She came to our clinic because she was convinced that she was suffering from mercury toxicity, which caused "brain fog" and neccessitated chelation therapy. She stated that she was diagnosed with trichomoniasis in January 1990. The trichomoniasis proved to be Flagyl-resistant, and she was eventually treated with intravenous medications. According to her history, the trichomoniasis was finally cured in July 1998, but she continued to have vaginal pain. The patient was eventually diagnosed with fibromyalgia and chronic fatigue syndrome. She stated that she had difficulty dealing with stress and had poor mental functioning, fatigue, and low energy. She smoked two packs of cigarettes per day and had done so for 30 years. She had not consumed alcoholic beverages in 10 years and denied drug use or hobbies associated with toxic exposure. Her medications were estrogen and B_{12}, though no prior history of B_{12} deficiency was noted. Her physical examination, including neurologic examination, was within normal limits. She reported no occupational exposures to mercury, but reported eating tuna and sardines four to six times per week. Her medical records revealed that through the years she had extensive nontraditional medical testing, including evaluations for intestinal parasites and allergy testing for "snapper, sole, dietary components, and preservatives." Over the years, she had at least four blood mercury tests performed, resulting in concentrations ranging from 11 μg/L to

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37 µg/L. In addition, a hair analysis was performed using inductively coupled plasma-mass spectrometry (ICP-MS). The laboratory provided results for 39 elements divided into three categories: "elements regarded as toxic," "elements regarded as nutrients," and "other elements." Her results indicated a mercury level of 3.46 ppm, which the laboratory reported was more than 2 standard deviations above the mean. She was also evaluated by an alternative dentist who placed a probe in her mouth and concluded that her dental amalgams were contributing to an elevated mercury body burden. Based on these results, the patient started taking DMSA in the form of Captomer (Thorne Research, Inc., Dover, ID), available at that time on the Internet, as recommended by her nutritionist. She also had 12 amalgam fillings removed and replaced with caps. She presented with continued concerns about her elevated mercury levels.

Case 3. Patient 3, a 46-year-old white male graduate student, came to our clinic seeking continuation of lead chelation therapy, which he had received from prior physicians. He had been receiving chelation therapy with intravenous ethylene diamine tetraacetic acid (EDTA) for the past 2.5 years from two separate physicians. He stated that his symptoms began 3 years ago with constipation, gas, and mood swings. His problems reached a crisis point, and he was diagnosed with attention deficit hyperactivity disorder, irritable bowel syndrome, bipolar disease, and "narcissistic complex." Finally, a physician who took a nutritional approach tested both stool and hair samples. According to the patient, the stool testing led to the diagnosis of "five bugs," and hair analysis revealed an elevated lead concentration. A provocative challenge was reported as "very high" and the patient stated that he was eventually treated with intravenous EDTA chelation therapy two times per week. The patient reported a dramatic improvement in brain functioning since treatments; he stated that "my IQ soared." His medications were citalopram and ibuprofen. He did not smoke tobacco, drink alcohol, or use recreational drugs. His physical examination was within normal limits. On the first visit, the patient requested continuation of his chelation therapy. We requested his laboratory results, which he sent. The patient's laboratory results included hair analyses performed on four different occasions over a 26-month period, all of which were purported to reveal substantially elevated lead concentrations. His hair lead-level results were 27.66 ppm in June 1998 (laboratory reference range, 0–0.8 ppm), 10.42 ppm in April 1999 (laboratory reference range, 0–0.8 ppm), 5.3 µg/g in January 2000 (laboratory reference range, 0–2.0 µg/g), and 8.26 ppm in August 2000 (laboratory reference range, 0–1.4 ppm). The method of analysis for three laboratories was not indicated, and the method used by the fourth laboratory was ICP-MS. The patient also had three provocative challenges in which urine was collected for 24 hr and the amount of lead was measured after administration of an unspecified dose of EDTA or DMSA (it was not indicated which was used). These test results were 19.3 µg/24 hr in August 1999, 17 µg/24 hr in November 1999, and 22 µg/24 hr in October 2000. In addition, the patient had a spot urine analysis for lead as well as a lead concentration in packed erythrocytes, both of which were stated to be elevated based on the reference ranges provided by the laboratories.

Discussion

Many commercial laboratories, chiropractors, nutrition consultants, practitioners of alternative medicine, and others promote the use of hair analysis. Some have set up Web sites on the Internet that state or imply that hair analysis can help diagnose a variety of diseases and conditions, including metal toxicity, and that the results of hair analysis can be used as the basis for taking dietary supplements and other therapies (*1*). The Web sites commonly market and sell dietary supplements and other treatments that are purported to be necessary therapy for patients with "abnormal" hair analysis results.

Although we cannot trace this development to the Internet and have not attempted to quantify the number, we have observed an increase in the number of patients coming to our clinic with concern for their health following hair analysis. These three cases are typical. In each of the three cases, the patient presented with nonspecific complaints such as joint pain, muscle aches, fatigue, flu-like symptoms, constipation, loss of appetite, and headache, but lacked objective evidence of illness. They had diagnoses such as fibromyalgia, chronic fatigue syndrome, irritable bowel syndrome, anxiety, and depression. In addition, the patients had been diagnosed with metal toxicity based on hair analysis and other diagnostic tests, and were either treated with chelation therapy or believed that chelation therapy was required. In each case, hair analysis results obtained before coming to our practice were reported to be elevated, whereas the additional tests used were within normal limits (e.g., lead chelation challenge) or were difficult to interpret (e.g., lead level in packed erythrocytes, spot urine samples for lead).

In case 1, despite the patient's elevated hair mercury level and DMSA challenge, we did not feel that this patient was suffering from mercury toxicity. The patient's history revealed a lack of significant environmental or occupational exposure to mercury, and her physical exam was within normal limits. We also did not believe that the urine test following a DMSA challenge (11.5 µg/g creatinine) was consistent with toxicity. Although there is a lack of normative data for provocative DMSA challenges for mercury, which makes her results difficult to interpret (*2*), data regarding unstimulated urine tests suggest that her results did not reflect toxicity. In unexposed populations, a normal mercury urine concentration is < 10 µg/g creatinine. Subtle neurologic effects are observed at urinary mercury levels > 20 µg/L and early renal effects are observed at levels > 50 µg/g creatinine (*3,4*). Therefore, we felt that the result of this patient's provocative challenge was relatively low and did not reflect mercury toxicity. The diagnosis of mercury toxicity is typically made from testing blood or urine (*5*). In this patient, a blood test obtained in our clinic resulted in a mercury level of 11 µg/L (1.1 µg/100 mL). According to the Agency for Toxic Substances and Disease Registry, normal levels for mercury in blood range from 0.5 to 2.0 µg/100 mL (*6*).

In case 2, the patient was also concerned about mercury toxicity and had begun chelation therapy in the form of Captomer. Captomer is sold in 100-mg tablets, of which 65 mg is succinic acid (*7*). Captomer or other forms of DMSA can be purchased over the Internet (*8*). Many alternative medicine Web sites offer DMSA for "detoxification" (*9*). Patient 2 also had 12 dental amalgam fillings removed. We assessed that, although this patient's mercury levels were higher than would be expected in the general population, these levels did not warrant chelation therapy or removal of amalgams. Rather, we believed that this patient's mercury elevation was most likely due to her fish consumption, which ranged from four to six servings per week. In studies of persons who reported eating fish more than four times per week, the mean whole blood mercury level was 4.44 µg/100 mL (44.4 µg/L) (*10*), a level entirely consistent with this patient's history. In addition, symptoms relating to mercury toxicity in persons with occupational exposure are more likely to occur at blood levels in excess of 200 µg/L (*10*), which is considerably higher than this patient's results. We therefore recommended to this patient that she decrease her fish intake to no more than one serving per week and that she have another blood level measured in 1–2 months. She did not return for a repeat measurement.

In case 3, the patient had received intravenous EDTA for 2.5 years. It was our assessment that the patient was not suffering from lead toxicity and that chelation was not warranted. His physical examination was within normal limits, and his laboratory

results did not support lead toxicity. A provocative challenge, such as the patient received, is appropriate for determining chelatable lead burden. Typically, values > 600 µg/24 hr are considered evidence of an elevated chelatable-lead burden (*11*). The patient's values never exceeded 22 µg/24 hr, however. In our view, spot urine samples and packed erythrocytes for lead, as performed on this patient, are less useful than measurement of blood lead and chelatable-lead in evaluating patients (*10,11*), mainly because of inherent difficulties with interpretation of these measures.

In addition to concerns about the diagnoses these patients had received, we were concerned about their treatment in these settings. These patients all sought or were already receiving chelation therapy. Chelation therapy has not been shown to improve outcomes in patients with the clinical presentations described and the low levels of mercury seen in cases 1 and 2 (*12*). There is some evidence that chelation in patients with renal insufficiency and low-to-moderate chelatable-lead levels improves creatinine clearance over time (*13*), but patient 3 did not have renal insufficiency and his lead level was lower than those described by Lin et al. (*13*). In view of the lack of evidence on the efficacy and safety of chelation treatment in patients with low-to-moderate metal exposures, removal of the individual from the exposure is the therapeutic approach most favored (*12*). This is the approach we followed with patient 2 when we recommended reduction in fish consumption.

In these three cases, the treatments provided were homeopathic at best (DMSA) and possibly detrimental at worst (intravenous EDTA). The homeopathic nature of patient 2's treatment is demonstrated by comparing the dose of Captomer that she was taking with the recommended dose of succimer (also contains DMSA) for patients with mercury toxicity. One Captomer pill contains 65 mg of succinic acid. Patient 2 was taking three Captomer pills per day initially, and later three pills per week. By contrast, the recommended dose of Succimer for mercury intoxication is 30 mg/kg/day or 1,500 mg/day for a 50 kg woman for 5 days (*14*). Patient 3 received intravenous EDTA for 2.5 years. This treatment exposed him to the potential side effects of EDTA without evidence of the possibility of clear clinical benefit.

As discussed above, a common theme to these patients' case histories was the role of hair analysis in the diagnosis of metal toxicity. It should be noted that hair analysis can be useful in certain settings. Research studies using validated methods can effectively assess methymercury levels of a population (*15*). Mercury and arsenic poisoning have also been documented with the use of hair analysis (*16,17*).

It is important to distinguish the use of hair metal analysis in a research setting (in which one or a few analytes are measured in individuals in a defined population to make inferences in that population) from the use of a panel of hair metal measurements to make a diagnosis in an individual patient. This is particularly true with patients whose symptoms and exposure history may suggest a low likelihood of metal toxicity; if the pretest probability of disease is low, the probability that a positive test is a false positive result can become substantial (*18*). In addition, each of our patients had an extensive panel of mineral and toxicant levels (consisting of 34–39 analytes) measured in their hair. Assuming that test results are independent of one another, testing 34–39 different minerals and toxicants instead of 1 greatly increases the chances that any one of these test results will be elevated by chance alone. For example, if the 95th percentile of the distribution is selected as the criterion for an abnormal test and the analyte results are independent, then the chance that at least 1 analyte in a panel of 34 will be elevated based on chance alone is approximately 83% {i.e., $[1 - (0.95)^{34}]$} (*19*).

Several studies over the past few decades have demonstrated the limited utility of hair analysis as a diagnostic tool. Seidel et al. (*17*) recently reported a comparison of hair analysis results in six commercial laboratories. In their paper they concluded that "hair mineral analysis from these laboratories was unreliable," and recommended "that health care practitioners refrain from using such analyses to assess individual nutritional status or suspected environmental exposures" (p. 67). Indeed, the medical literature makes plain that physicians should not rely solely on hair analysis to diagnose or treat heavy metal toxicity or nutritional deficiencies because these tests are unreliable and have poorly established reference ranges (*17,20,21*). Accordingly, occupational and environmental physicians have cautioned that making treatment decisions on the basis of hair analysis can result in adverse medical and public health consequences, such as unnecessary and potentially dangerous treatments and needless worry on the part of patients (*22*).

Clinicians examining patients who believe they are suffering from metal toxicity because of hair analysis results should be prepared to discuss with their patients the reasons why the hair analysis may not serve as a reliable diagnostic tool in individual cases. Some of the concerns that researchers have raised regarding hair analysis as a diagnostic tool include lack of validation of analytical techniques, presence of exogenous contaminants, and variable analytic procedures and low interlaboratory reliability. These factors, coupled with the fact that reference values are not well-established, mitigate against the use of such testing in individual patients presenting with nonspecific, multisystemic symptoms.

Lack of validation of analytic techniques. Seidel et al. (*17*) reported that laboratories vary greatly with regard to the standards used for validation, quality assurance, and quality control. Calibration standards include the use of non-hair mineral standards, pooled in-house hair samples, and the use of "Chinese commercial hair standard(s) certified for up to 17 elements" (p. 68). There is no standard certification for metals analyzed.

Presence of exogenous contaminants. Hair provides an effective medium for binding material such as dust and sweat (*23*). Exogenous contaminants can make hair analysis highly inaccurate because it is unclear whether the source of the mineral is endogenous or exogenous. Sources of contamination include sweat and sebacious secretions, dust, and beauty treatments such as shampoos, conditioners, permanent waves, bleaches, and hair spray (*24*). Sky-Peck (*25*) compared natural hair to hair treated with peroxide or with permanents and showed that treatments significantly altered the concentration of sulfur, calcium, iron, and nickel. In addition, hair treatments affect zinc, copper, and arsenic concentrations (*25*). Others have shown significant effects of beauty treatments on zinc and copper concentration (*21*). Klevay et al. (*26*) reported a list of 16 elements whose concentrations in hair can be affected by grooming products.

Many different washing procedures have been described and numerous studies have been carried out to determine which method is most effective. These procedures include the use of organic solvents, ionic and non-ionic detergents, chelating agents, rinses with deionized water, hot solutions, ultrasonification, dilute acid, and cold distilled water (*23*). Seidel et al. (*17*) reported that the washing methods differ greatly among laboratories, with some laboratories not washing at all; this illustrates the lack of consensus on the best washing technique (*17*). Despite efforts by the American Medical Association (*27*), the International Atomic Agency (*23*), individual laboratories, and groups of laboratories (Society for Elemental Laboratory Testing), approaches to washing among laboratories continue to vary (*27*).

Variable analytic procedure and low interlaboratory reliability. Differing analytic procedures contribute to the discrepancies observed among different laboratories. Although many analytic procedures have been described, the two most commonly used today, based on a recent survey, are ICP-MS

and inductively coupled plasma-atomic emission spectrometry (ICP-AES) (*17*). ICP-MS has been shown to be more sensitive at detecting the lower limits of trace elements and less likely to provide discrepant reference levels than ICP-AES (*28*).

Several studies over the years have performed interlaboratory comparisons. In 1982, Mason and Zlotkin (*29*) reported a study in which hair samples from three healthy men were sent to three laboratories. In 1985, Barrett (*20*) reported on samples from two healthy teenagers that were sent to 13 laboratories. Sixteen years later, Seidel et al. (*17*) described a study in which a hair sample from a healthy individual was submitted for analysis to six different laboratories. All of these studies found substantial interlaboratory variability.

It has been estimated that 225,000 hair mineral tests are performed each year in the United States by the nine leading commercial laboratories, at an average annual cost of approximately $9.6 million (*17*). However, this estimate undervalues the costs to society because it does not include the cost of additional unnecessary diagnostic tests, unnecessary treatment, and adverse outcomes that ensue. The three patients described above, for example, received unnecessary diagnostic tests and unnecessary treatment, although fortunately, there were no apparent adverse outcomes.

Conclusion

We have presented three patients who came to our clinic after having received diagnoses of metal toxicity based, at least in part, on the results of hair analysis. All three patients initially presented to outside physicians for evaluation of nonspecific, multisystemic symptoms. Occupational and environmental history, medical history, and physical examination suggested that the likelihood of metal toxicity as the cause of the symptoms was low. In the case of these three patients, laboratories measured a panel of analytes in hair and reported that one or more analytes were elevated. Environmental and occupational physicians should be aware that some patients who are concerned about metal toxicity may ask about hair analysis or may already have had hair analysis performed. Hair analysis should be used with caution to make decisions regarding overexposure or need for treatment in patients with nonspecific symptoms and a low pretest probability of metal toxicity based on medical history, exposure history, and physical examination. Physicians should be prepared to explain to patients the reasons that hair analysis is not a reliable indicator of exposure in this setting. Finally, physicians should counsel patients to stop treatment if they are already being treated for metal toxicity based on hair analysis and other unconventional diagnostic tests.

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