Chronic Fatigue Syndrome (CFS) and related illnesses: A case history supporting subacute mercury poisoning or "micromercurialism"

Richard F. Miller, ScD
19903 Pinehurst Trail Drive
Humble, Texas 77346-4534
Phone: +1 281 852 4554 Fax: +1 281 852 0879
Email: kmstry@earthlink.net

Abstract

The immunological findings in CFS patients have now been explained, especially chronic T-cell activation as the result of T_H2 cell production in difference to T_H1 cell production. Significant depletion of the sulfhydryl-containing(-SH) amino acid, L-cysteine and the sulfhydryl-containing(-SH) tripeptide, glutathione, concentrations are explained by the body's attempt to excrete the mercury introduced by vaccination and/or diet, resulting in competing T-cell processes not able to downregulate each other. Once these sulfhydryl-containing (-SH) amino acid/tripeptide concentrations are returned to normal, the T_H1/T_H2 ratio will normalize and said chronic T_H2-cell activation would cease. Low selenium levels have also explained by the formation of potentially hydrophobic mercury selenides and this further explains the elevated mercury levels seen in organs like the brain, liver and testicles. The interruption of metabolic pathways, such as carbohydrate metabolism leading to Diabetes Mellitus II in select CFS patients, has also been elucidated and evidence presented which support the influence of heavy metals like mercury playing a significant role.

Keywords: Chronic Fatigue Syndrome (CFS), micromercurialism

Introduction

In a previous report in this journal, it was suggested that subacute mercury poisoning or "micromercurialism", resulting from the administration of Thimerosal-containing vaccines (TCVs); stress and heredity play a significant role in causing Chronic Fatigue Syndrome (CFS) and related illnesses [1]. The focus of this report will be to support the previous immunological observations made; support with animal data and limited human data, the claim that heredity plays a significant role in this and related illnesses and to disclose the mercury levels found in a CFS patient and the results to date with respect to his treatment and recovery. Further, it will be the focus of this article to contrast and compare the symptoms of CFS and related illnesses to autism in children. The latter disorder although deemed more serious, presents striking similarities to CFS with the differences explained by the degree of neurotoxicity in infants and children versus adults [2] and the maturity of the hepatic excretory system [3-5].

1. IMMUNOLOGICAL OBSERVATIONS

1.1. Immunological observations in man

CFS researchers have looked at a variety of immune system anomalies and viral infection possibilities. Since CFS patients exhibit signs of chronic inflammation and/or chronic infection, research has been predominately focused on studies of the immune system and the identification of a potential "infecting agent". In research comparing healthy control patients to CFS patients, the most frequent immunological abnormalities found included chronic T cell activation, decreased Natural Killer (NK) cell function, reduction of the $CD8^+$ suppression cell subset and differences in subsets of $CD4^+$ cells. Further, a significant decrease in intracellular glutathione concentration is observed and a low selenium (Se) concentration in red blood cells with lower concentrations in white blood cells, specifically lymphocytes, has been reported [1].

1.2 Immunological Observations in Animal Models

If one takes the aforementioned observations made in CFS patients and compares them to immunological observations made in Brown Norway (BN) rats, which develop an immunologically mediated disease after injection with nontoxic levels of mercuric chloride ($HgCl_2$), one observes that the immune response is characterized by T-cell dependent B-cell polyclonal activation, responsible for an impressive increase in serum IgE concentrations from <10 μg/ml to ≥5 mg/ml [6-16].

This demonstrates that (a) T-cells from Hg-exposed animals were responsible for autoimmune reactions, (b) they may cooperate with normal syngeneic B-cells for antibody production and (c) they initiate a regulatory circuit employing CD_8^+ suppressor cells. Of interest in animal models, is the fact that methylmercury or pharmaceutical ointments and solutions containing organomercury compounds are effective whether these products are applied to wounds or to normal skin [17]. Further, autoreactive CD_4^+ cells can be detected in the BN rat model as early as 4-6 days. T-cells proliferate in the presence of B-cells either exposed to toxin or to B-cells from normal BN rats.

1.3 Selenium depletion

In experimental animals and in studies on nerve tissue cultures [18], selenium has been shown to interfere with the metabolic and toxic effects of methylmercury (MeHg). Selenium (Se) and MeHg create in the blood, under the potential influence of glutathione (GSH), bismethylmercuryselenide ($[MeHg]_2Se$) [19]. This compound seems to penetrate the

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blood-brain barrier, giving rise to mercury accumulation to a greater extent than MeHg alone [20].

Selenium or selenohydryl ligands have a greater stability as compared to that of sulfhydryl groups (-SH), however, several authors have reported changes in the distribution of MeHg after administration of selenium with an increase in the accumulation of selenium in the brain, liver, kidneys and blood. Selenium although found to be lower in both red and white blood cells, appears to increase the mercury retention in adult and fetal brains [21, 22].

1.4 Chronic fatigue syndrome compared to Autism

It was first suggested by Bernard et al. [23, 24] that autism was a novel form of mercury poisoning and although substantial numbers of articles have been published which warn of the potential ill-effects of mercury in infants and children [25-44] limited data are available, until recently, disclosing the potential effects of mercury restricted to post-natal life and the effects of heredity [45].

This article will prove the ill-effects of mercury in adults and will hope to extend the successful treatment of a CFS patient to children suffering from autism. The remaining question is not whether mercury plays a significant role in the cause of CFS but whether the administration of Thimerosal-containing vaccines (TCVs) is the sole cause of CFS and related illnesses like Gulf War Syndrome (GWS) or whether TCVs simply raise mercury levels above a threshold level which trigger the disease state?

2. CASE HISTORY

2.1. Vaccinations

In 1996, a forty-six year old, white, male was asked to travel to South America on short notice. Having traveled internationally on several occasions before, he had been vaccinated against Yellow Fever, Cholera and Typhoid with no ill effects. The patient was advised to update his flu and DT vaccinations and to obtain vaccinations against Hepatitis A and B. At the beginning of October 1996, the patient received 1cc each of influenza, Hepatitis A and B and DT vaccines. After two weeks, he received an additional 1cc each of Hepatitis A and B vaccine and was told to complete his vaccination against Hepatitis B at the end of one year. Within one month, the patient received the equivalent of 150 μg of mercury [46]. He also received prophylactic medication for malaria.

2.2. Stress Factor(s)

The trip to multiple countries in South America was very successful; however, upon returning home in early November 1996, he was informed that his father was very ill, requiring hospitalization. His father died four days after entering the hospital, diagnosed with cancer of the pancreas, liver, bowel and lungs. Prior to his father's death the patient had to obtain his father's signature on a do not resuscitate order (DNR), plan his father's funeral and appear before a probate judge to get permission to enter his parents' safe deposit box to locate his father's will. His mother had been wheel chair bound for several years prior to his father's death and had never signed the safe deposit box documents.

After his father's funeral, came the Thanksgiving, Christmas and New Year's holidays when he had hoped to relax and recover, however, his mother collapsed, do in part to losing her husband of over fifty years and had to be hospitalized for several weeks.

2.3. Flu Symptoms and CFS Diagnosis

In February 1997, while watching his son's soccer game, the patient "caught a chill" and after the game went home spending the next three days in bed with the "flu". After this time, he returned to work where his temperature spiked, reaching 105˚F. The care of a physician was immediately sought. The initial diagnosis was indeed the flu but after an additional three days, the patient showed no improvement. On day six of the "flu", the patient asked his physician to run an Epstein-Barr titer, having recently read about a new illness wherein flu-like symptoms followed by extreme fatigue had been reported and linked to a chronic Epstein-Barr viral (EBV) infection.

On day ten, the physician disclosed to the patient that indeed he had an elevated EBV titer which couldn't be possible because the patient had suffered from mononucleosis as a teenager and that it was essentially impossible to experience the same infection twice unless something drastic was occurring with respect to the immune system. The patient was referred to a Houston physician for treatment at which time a physical was completed and blood work drawn. The patient was later told of the diagnosis of Chronic Fatigue and Fibromyalgia Syndromes. Apart from having an elevated EBV titer, extreme fatigue, muscle soreness, chronic low-grade temperature, etc. blood work showed an extremely low glutathione level, approaching zero; very low levels of natural killer (NK) cells and an abnormal lymphocyte proliferation index. The patient was placed on glutathione (GSH) replacement therapy in March 1997.

2.4. Glutathione Replacement Therapy

After eighteen months, the patient's glutathione concentration was re-tested and found to have returned to 89% of normal, concentrations of the sulfhydryl-containing (-SH) amino acid, L-cysteine, where found, however, to be in the lower 25% percentile of the acceptable range (21.3%) and a lymphocyte proliferation index was determined to be 34.1% (average range = 25 to 75%). This data was deemed interesting but no connection was made to subacute mercury poisoning or "micromercurialism" at this point.

2.5. Chelation Therapy

After reading about mercury poisoning cases in Iraq and Japan, autism and Thimerosal-containing vaccines (TCVs), the question raised was whether CFS and GWS were related to the others and whether chelation therapy would be beneficial? Initially, chelation therapy with tetrasodium edetate (Na_4EDTA) or calcium disodium edetate ($CaNa_2EDTA$) was considered but

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there was a concern that these chelants were too indiscriminate, able to chelate and promote the elimination of necessary metals like potassium, calcium, magnesium and zinc and that long-term chelation therapy with these compounds would require constant electrolyte/mineral supplement replacement. Although chelation therapy employing these additives would theoretically lower mercury levels, it was decided that a chelant containing at least one sulfhydryl (-SH) moiety would be preferred because of its relative specificity for mercury (Hg). Hence, N-acetyl cysteine was employed and Succimer, (Chemet® / DMSA, 2, 3-dimercaptosuccinic acid) was employed to challenge the system to determine residual levels of heavy metals. Results are shown in Table 1.

3. Experimental

Heavy metals concentrations were determined by Coupled Plasma-Atomic Emission Spectroscopy (ICP-AES) by Great Smokies Diagnostic Laboratory of Asheville, North Carolina and/or King James Medical Laboratory of Cleveland, Ohio.

N-acetyl cysteine is an over-the-counter (OTC) dietary supplement and was used at a dose of 500 mg at bedtime for six weeks. Chemet® is available by prescription. Although Chemet's® primary indication is for acute lead poisoning in children > 1 year of age, used at a dose of 10 mg per kg body weight, it is also recommended for treatment of poisoning cases caused by heavy metals such as mercury.

Glutathione concentrations, Natural Killer (NK) cell counts, physical symptoms and physician observations were completed every three months or as required, from March 1997 until the present. Pain surveys and fatigue questionnaires where completed at each visit. The data are available through the aforementioned physician of record, Dr. Patricia D. Salvato.

Spectrox™ total antioxidant function, which included analyses for L-cysteine and glutathione and lymphocyte proliferation indices were conducted by SpectraCell Laboratories, Inc. of Houston, Texas.

4. RESULTS AND DISCUSSION

4.1. Pre-trial observations

Several observations of interest were made before and after chelation therapy and during GSH replacement therapy, which have a direct bearing on the hypotheses aforementioned [1]. Prior to chelation therapy but during the time that GSH replacement therapy was being conducted, the following observations were made:

1. Pain seemed to be moderated by serotonin levels. When a serotonin re-uptake inhibitor (SSRI) was not taken routinely, pain was observed to be more intense.
2. Although both Actos and Starlix were being taken to control blood sugar levels and a diet was being followed, blood sugar levels were not easily controlled.
3. Acetaminophen-containing pharmaceuticals were avoided as they are reported and were found to accelerate the deletion of GSH from the body [47].
4. Testosterone seems to intensify the ill effects of CFS as is reported in autistic males.

4.2 Chelant trial observations

Approximately eight months after chelation therapy supplemented by GSH•ATP injections, 200 mg per week, was completed [1]; several significant observations were again made which have a potential direct bearing on the future treatment of CFS and GWS patients and potentially autistic children. They were:

1. Pain control became very manageable; as time progressed pain was controlled in most cases by Naproxen or aspirin. Prior to this time, ≈ 250 mg of morphine sulfate was needed daily to control pain.
2. Sleep cycles normalized to the extent that Ambien was no longer needed to experience a good night's rest.
3. Control of blood sugar levels became very manageable. Actos was discontinued because blood sugar levels were dropping to as low as 80. Starlix was discontinued after activity levels increased because blood sugar concentrations began falling into the normal range of 80 – 120 mg/dl.
4. Neurontin was completely discontinued, not needed for pain control.
5. Bowel activity and bowel sounds returned to normal.
6. Provigil was completely discontinued because energy levels started returning to normal. The patient was able to start working around his home and in the yard, four to six hours a day; which in Houston, Texas, is an accomplishment for healthy individuals because of temperatures and humidity. He further started looking for a full-time job
7. The patient's drug sensitivity increased to levels experienced before becoming ill. As little as 100 mg of Neurontin would put the patient to sleep for hours. Prior to this time, the patient was taking morphine sulfate, Neurontin, Flexeril, etc. with no prolonged sedation.
8. The patient's environmental allergies returned to pre-illness levels. During the height of the illness, the patient was tested for previously known environmental allergies and showed no reaction to sub-cutaneously injected allergen test solutions indicating that his immune system was essentially not functioning.
9. Bladder irritation decreased thereby allowing for the discontinuation of Detrol-LA.

4.3. HERITABLE TRAITS

4.3.1. Heritable traits in animal model

The role of T-cells in targeting autoimmunity has been well documented; BN rats deprived of T-cells, either genetically or after adult thymectomy, lethal radiation and reconstitution with fetal liver cells are completely protected from mercury (Hg)-induced autoimmunity [48]. In addition, transfer of T-cells from diseased BN rats into naïve syngeneic BN rats induces a mild form of autoimmunity. If recipients are also depleted of CD_8^+ suppressor/cytotoxic cells with an anti-CD_8 monoclonal antibody, the disease is marked with an increase in IgE concentration to ≈ 700 μg/ml [49].

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It must be noted that some strains such as Lewis (LEW) rats are completely resistant even when injected with increased doses (400 μg/100 g body weight) [50-52]. This observation has recently been supported by Hornig et al. [45].

4.3.2. Heritable traits in humans

A significant point which requires introduction at this time is the fact that the patient's two female siblings, ages 45 and 40 at the time, having also been vaccinated for Hepatitis A and B; one an international traveler and one a first responder, both developed what was diagnosed as CFS at around the same time as the patient, however, both seemed to have less severe symptoms as might be expected since estrogen has been reported to protect females, lessening the ill effects of subacute mercury poisoning which has also been seen in autistic children where male to female ratios are approximately 4-5:1 [53]. Later, the younger sister's diagnosis was changed to Raynaud's phenomenon wherein, a concern was whether Thimerosal's cometabolite, thiosalicyclic acid, played a role in the ultimate diagnosis of the younger sister who was a known "aspirin allergic".

4.4 IMMUNOLGOCIAL OBSERVATIONS EXPLAINED

4.4.1. Induced autoimmunity in animal model

Mercury-induced autoimmunity in BN rats is easily prevented and cured by introduction of immunosuppressive agents such as cyclosporine A [54, 55] and cyclophosphamide [56, 57] even administered for a short time (0-10 days) after initial $HgCl_2$ injections. In addition, cyclosporine A pretreated rats become unresponsive to $HgCl_2$ injections for up to five weeks [55]; lymphoid cells cannot transfer this protective effect and unresponsiveness can be broken by injection of naïve syngeneic lymphoid cells. It has also been shown that it is possible to vaccinate against autoimmunity by injecting "attenuated" autoreactive T-cells. In both cases, proliferation is blocked when stimulator cells are pre-incubated with an anti-class II monoclonal antibody [58, 59].

4.4.2. Suspected immune response in humans

In humans, CD_4^+ cells can divide into two subsets of T-helper (T_H) cells: T_H1 and T_H2 [60, 61]. T_H1 cells produce interlukin-2 (IL-2), interferon-γ (IFN-γ) and tumor necrosis factor-β (TNF-β), employing IL-2 as a growth factor. These factors may be mainly responsible for delayed hypersensitivity reactions and can cooperate with B-cells for antibodies of the IgG2 isotype in mice.

T_H2 cells produce IL-4, IL-5, IL-6, and IL-10 and can use IL-2 and IL-4 as growth factors. These factors are mainly involved in B-cell help and particularly in antibodies of IgA, IgG1 and IgE isotypes. Each subset downregulates the other one. It should be noted that in mice susceptible to mercury-induced autoimmunity can be treated with an anti-IL-4 monoclonal antibody which prevents the disease. One potential explanation and an important conclusion reached in Miller [1] is the preferential activation of T_H2 cells to the detriment of T_H1 cells occurs because mercury (Hg) depletes cells of free cysteine and of the reduced form of glutathione (GSH); both play an important role in the production and the responsiveness to IL-2 by T-cells [62]. Thus T_H2 cells that can be IL-2 independent would be favored. See Figure 1.

5. CONCLUSIONS

The data are conclusive in this case history; however, there is at least one caution to be voiced. Although the patient had shown improvement while on glutathione replacement therapy and continued said therapy without any ill-effects, the addition of a mercury chelant interrupts the enterohepatic steady-state circulation of a cysteine-mercury complex promoting the elimination of mercury [1]. As a result the patient will feel as ill as before beginning treatment which seems to be common sense, in hindsight. After chelation therapy, the patient's health and physical stamina start returning in a dramatic fashion. An unrelated specialist seeing the patient approximately one year before and six months after chelation therapy made the unsolicited comment that the patient seemed "much brighter (Shu, S. to Miller, R., 2005).

The metabolic by-product of Thimerosal hydrolysis *in vivo*, thiosalicyclic acid should now also be of concern when considering its affect on aspirin allergics and particularly in children. Is "micromercurialism" further complicated by this sulfur-containing derivative of aspirin? Could this explain reports of additional multiple etiologies characterized by an encephalitic-like state with fever, vomiting, disturbance of consciousness and/or convulsions? If thiosalicyclic acid can indeed trigger Reye's syndrome-like symptoms then Thimerosal is "a sharp two-edged sword."

If symptoms of micromercurialism and Reye's syndrome overlap, the potential of Reye's syndrome can be determined by analyzing the sugar levels of the cerebrospinal fluid because they are found to be consistently low. Further, the liver and kidneys could be evaluated for massive fatty infiltration without necrosis. The patient would also exhibit cerebral edema and deposition of fat droplets in the endothelium of cerebral blood vessels; however, several of these analyses are made more difficult to impossible because of the age and size of the autistic patient.

Several immunological findings in CFS patients have now been explained, especially chronic T-cell activation which results from T_H2 cell production in deference to T_H1 cell production. If levels of L-cysteine and glutathione weren't depleted by the body's attempt to excrete the mercury introduced by vaccination or diet, the two processes would downregulate each other and said chronic T_H2-cell activation would cease. Low selenium levels have also explained by the formation of potentially hydrophobic mercury selenides and this further explains the elevated mercury levels seen in organs like the brain. The interruption of metabolic pathways, such as carbohydrate metabolism leading to Diabetes Mellitus II in select CFS patients, has also been elucidated and evidence presented which support the influence of heavy metals like mercury playing a significant role.

It only remains for a physician treating CFS or GWS to study the L-cysteine/glutathione therapy in large numbers of patients and more importantly for a physician treating autism to

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try this orthomolecular therapy in children. The sulfhydryl-containing (-SH) amino acids, L-cysteine and L-methionine, could be selectively added to an autistic child's diet and GSH·ATP therapy initiated. Having been on GSH·ATP therapy for eight years, the author has experienced no ill effects, however, it has been noticed that wound healing/remodeling is positively affected by glutathione. A potential study of Cyclosporine A in CFS and/or GWS patients should also be rigorously evaluated.

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Table 1: "Provocative" Urinalysis Results[1]

Element	Acceptable	Found (µg/g Creatinine)	After Chelation Therapy
Arsenic	0 - 45	29.7	< 0.42
Barium	< 5.9	2.22	N.D.[2]
Cadmium	0 - 4	0.29	< 0.21
Lead	0 - 20	0.90	< 0.42[3]
Mercury	- 0[4] -	1.48	< 0.15
Nickel	0 – 10	9.37	< 0.42
Selenium	20 – 200	128.6	96.76
Zinc	70 – 700	1889.5[5]	692.45

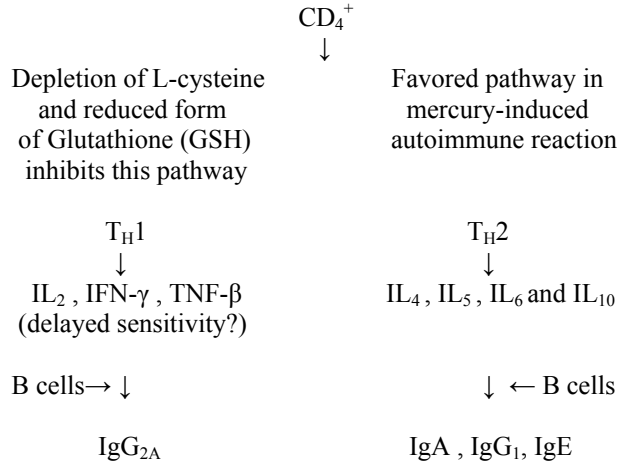
[1]Mercury concentrations were determined in the fifth year of glutathione replacement therapy.
[2]N.D. = Not Determined.
[3]If values presented are <, it reflects that the concentration was below detectable levels.
[4]Although acceptable concentrations are reported by some literature references to be less than 2.31 µg/g creatinine, Thimerosal allergics should have a mercury concentration of 0 µg/g creatinine.
[5]The zinc concentration was elevated due to taking mineral supplements.

Table 2: Medication Requirements Before and After Chelation Therapy

Medication	Before Chelation Therapy (Cumulative Daily Dosage and/or Range)	After Chelation Therapy (Cumulative Daily Dosage and/or Range)
Actos	60 mg	0
Ambien	10 mg	0
Avinza[1]	0	30 - 60 mg prn
Bextra	20 mg	0
Clonazepam	1.0 mg	0
Detrol-LA	4.0 mg	0
Elavil	5.0 mg	0
Flexeril	5- 10 mg	0 mg
Levothyroxine	0.088 mcg	0.088 mcg
MS Contin[2]	180 - 240 mg	0
MS IR[3]	45 - 60 mg	0
Neurontin	4800 – 7200 mg	0
Provigil	20 mg	0
Prozac	20 mg	20 mg[4]
Starlix	360 mg	240 mg[5]

[1] Avinza is a sustained-release capsule and was taken primarily to withdraw the patient from morphine slowly. It was later replaced with Darvocet N-100.
[2] Morphine sulfate continuous release.
[3] Morphine sulfate immediate release.
[4] The patient continued on Prozac because of a previous diagnosis of PTSS.
[5] After activity levels increased, blood sugar concentrations normalized and Starlix was discontinued.

Figure 1: CD_4^+ Differentiation into T_H1 and T_H2 Cells



1. As one recovers, the ratio of T_H1/ T_H2 should change, exhibiting an increase in [T_H1].
2. As [T_H1] increases, there should also be an increase in [IFN-γ].
3. Further, as both [T_H1] and [IFN-γ] increase, there should be an increased capacity to produce IL_2 which potentially explains the results of Salvato and Thompson [1, 64].

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