Orthomolecular Treatment Response

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Abstract *The following six case studies illustrate assessment and treatment scenarios common to orthomolecular psychiatry. These cases represent a sample of frequently seen mental health pathologies. From case to case, we see not only a common overlap of symptoms but also, an overlap of biochemical-nutrient imbalances. Imbalances depicted herein are widespread in an array of mental health conditions. Targeted lab assessment techniques help to hone in on relevant biochemical-nutrient imbalances. Consistent with treating these imbalances, we observe patients achieving greater mental health and physical well-being.*

Introduction

Two years ago Dr. Abram Hoffer M.D., Ph.D., now deceased, suggested that I publish these cases with the addition of assessment and treatment stratagem. When Dr. Hoffer lectured he commented that "the cases are what people want to hear." So few articles explain treatment strategy in a case series format and I consider this essential in furthering the development of the orthomolecular psychiatry knowledge-base. This article provides a viable treatment rationale for practitioners. The cases are, in my opinion, a typical sample of commonly seen mental health pathologies. From case to case, the histories reveal a commonly seen overlap of symptoms and biochemical imbalances. My clinical strategy implements the use of a targeted battery of tests that establish the biochemical profile at baseline. As treatment progresses, I only assess salient lab trends. I would expect to see consistent continued improvement, and in many cases a complete change in symptoms after the biochemically influential imbalances are reversed. The patients in these case studies have provided consent to publish their stories and findings.

Case Studies

Case 1: Chronic Depression and Anxiety, male, 46 years old
Case 2: Chronic Anxiety and Depression, female, 47 years old
Case 3: Schizophrenia, male, 18 years old
Case 4: Schizoaffective Disorder, male, 41 years old
Case 5: ADHD/Dyslexia, male, 42 years old
Case 6: Chronic Depression, female, 56 years old

Case 1: Chronic depression and anxiety, male, 46 years old

This patient was the mayor of a small town in Northern Ontario. He was struggling with flat stoic depression for 10 years. Anti-depressants had become less effective with time and he was considered treatment refractory (i.e. a non-responder). He had dominant negative thought rumination and occasional suicidal propensity. His mother, sister, and brother had depression. He reacted badly to sweets and alcohol, and skipping meals caused headaches, irritability, and fatigue (hypoglycemic trend). He was dizzy 2 to 3 times a week (sluggish

adrenal), and he had insomnia 1 to 2 times a week. He had prostatitis (inflamed prostate gland) 12 years prior and several courses of antibiotics had been taken. He had an Epstein Barr viral infection in 1972, a candidate trigger for affective disorders,[1] with report of visual illusions. He had significant fatigue and constipation. (**Figure 1,** below)

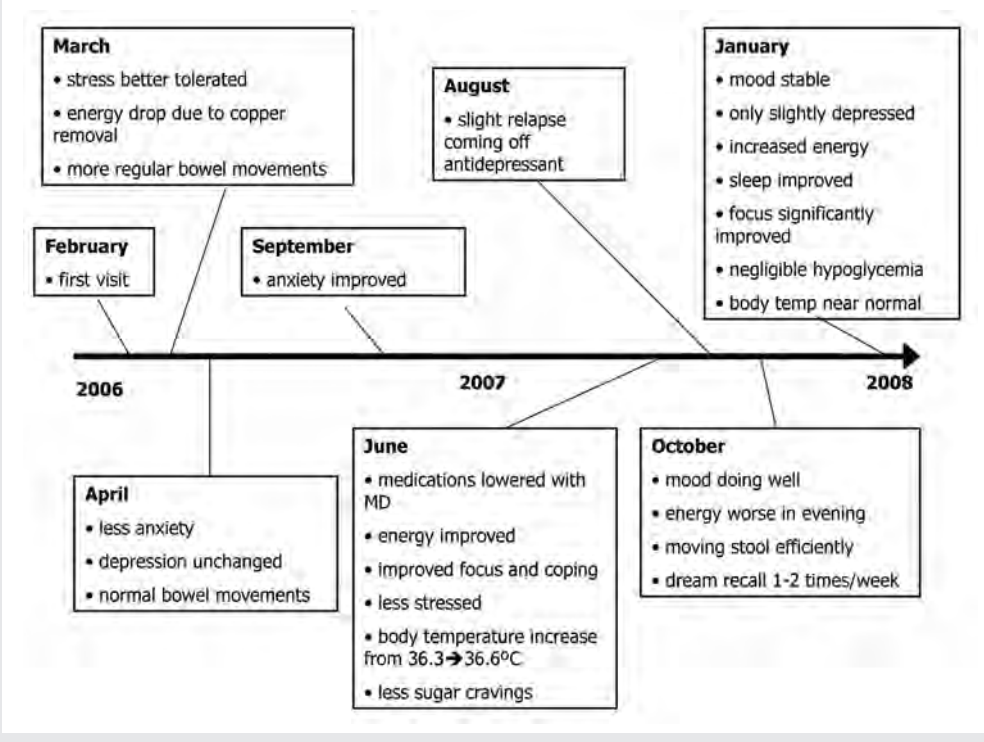
Assessment

• High tissue copper (toxicity) and a low zinc/copper ratio.
• High normal fasting blood homocysteine (mild under-methylation)
• Oral Glucose Tolerance Test (OGTT) blood glucose drops at the 2 and 3 hour mark (hypoglycemia)
• 'Normal' blood thyroid levels with low body temperature and clear hypothyroid symptomology, i.e., Wilson's Temperature Syndrome.

Treatment

Copper toxicity was addressed initially with zinc 50 mg, sodium alginate, and chlorella. In these cases I typically raise the zinc dose as tolerated by 15 mg increments monthly. Vitamin B_6 was given in the active pyridoxal-5-phosphate (P-5-P) form and I often raise the dose until the patient experiences significant dream recall, at least twice weekly; memory acquisition is in part B_6 dependant.[2] Patients presenting with heavy metals are suspect to having low thyroid metabolisms. They often retain heavy metal toxins if a stressor is significant enough to slow the metabolism down and weaken the organs that would normally eliminate it, i.e., the liver, kidney, and bowel.[3-5] Common stress triggers include not only exogenous sources but also physical stressors such as viral infections or metal toxins. I treated the low thyroid

Figure 1. Symptom changes during treatment of Case 1



state with a botanical protocol (blue flag, guggulipid, fucus, ashwaghanda) and desiccated thyroid (MD prescribed) while monitoring temperatures daily three hours after waking. I like to see temperatures rise into the upper end of the range to determine the effectiveness of the thyroid protocol. I used high dose chromium 600-1,200 mcg and a 40% lean protein diet to address glycemic perturbations. The under-methylated aspect in this case was mild and treatment with oral methylcobalamin (B_{12}-methyl) 0.5-1 mg, folic acid 0.5-1 mg, and betaine HCl 500 mg daily was sufficient. B-complex used here is a standard in most mental health protocols. My essential fatty acid (EFA) bias in adult cases with mood disorders is an eicosapentaenoic acid (EPA) to docosahexaenoic acid (DHA) ratio of 3:1. I do not find EFA's pivotal in reversing mental health pathology but they have a stabilizing effect, and help keep neuron membrane structure intact, which is important in clients who are reducing medications. In these cases I often prescribe Ginkgo to help maintain neuronal receptor balance and electric signaling to prevent medication related receptor decompensation effects. Magnesium glycinate is an excellent bowel regulator and sleep aid and the glycine component is a safe anti-anxiety amino acid. I usually maintain about 600 mg daily in adult mood cases. Magnesium is also useful in slow metabolizers to oppose high tissue calcium; tissue calcium (a sedative mineral) is high in low thyroid states. Vitamin C and niacin, 3 g of each were used initially and he now maintains 1,000 mg of inositol hexanicotinate (B_3-HEXA) and 750 mg of vitamin C. His current dose of zinc is under 60 mg a day. (**Figure 1,** p.5)

Summary

The root problem in this case was copper toxicity and low thyroid metabolism. After treating these factors he became much happier, more in control, and had more energy. Within two years, he experienced almost complete reversal of depression and anxiety. I started him on a custom supplement mix after six months to reduce the number of pills and improve compliance. His current custom supplement dose is three pills, twice daily. His current dose of ginkgo is three pills daily. He is on thyroid medication and I aim to optimize thyroid function further. I am currently assessing his blood and urinary mercury levels, both the ionic/inorganic and the methyl species. [I now assess blood and urinary organic methyl and ionic/inorganic mercury species early on in about 40% of mood disorder cases. Hair tissue levels do not rule out mercury toxicity alone. Hair measures help in determining how efficient the body is at excreting organic mercury at a tissue level when compared to the organic blood mercury load]. His ongoing treatment maintains the "lowest" effective dose of medication; he is off Tarazadone and currently weaning off Effexor from a low 75mg dose.

Case 2: Chronic anxiety and depression– female, 47 years old

This patient had been depressed for many years. She had a family history of depression. She experienced a "nervous breakdown" in 2003 where she lost 30 pounds. She had insomnia since a teenager with rousing at midnight and was unrefreshed on waking. She had been on anti-depressants and sleeping pills for years. She had significant fibromyalgia (often a low thyroid state) related neck and shoulder pain. [Musculoskeletal pain may be associated with the lack of adenosine triphosphate (ATP) in muscle cells; ATP is required to allow muscle cells to relax.[6] Inadequate thyroid function is related to inadequate ATP production at the mitochondrial level].[7] She had a non-malignant bowel tumor removed two years prior. She was constipated with only four bowel movements a week and only four inches of stool per movement. She had a history of intractable headaches. She experienced blurry vision (which may be adrenal related) all day worse in the morning and, she had cold hands and feet (often thyroid related). [Blurred vision is not always adrenal related but adrenal physiology is linked to the maintenance of blood pressure in times of stress and retinal arterial supply fed via cere-

bral blood flow can lag when blood pressure drops].[8] She had a history of anemia, menstrual disruption, premenstrual syndrome (PMS), and post-partum hormonal depression; she was currently peri-menopausal, with irregular menses. (**Figure 2,** below)

Assessment

• Low hair tissue and blood iron
• 'Normal' thyroid stimulating hormone (TSH) result despite low body temperature
• Low adrenal and thyroid hair tissue mineral trends indicative of a slow metabolizer
• No tissue heavy metals; inorganic mercury not ruled out but given her quick response (below) she probably did not have significant blood mercury; the hair mercury ratios showed no warring with important opposing minerals (iron, selenium, zinc).
• High-normal fasting blood homocysteine

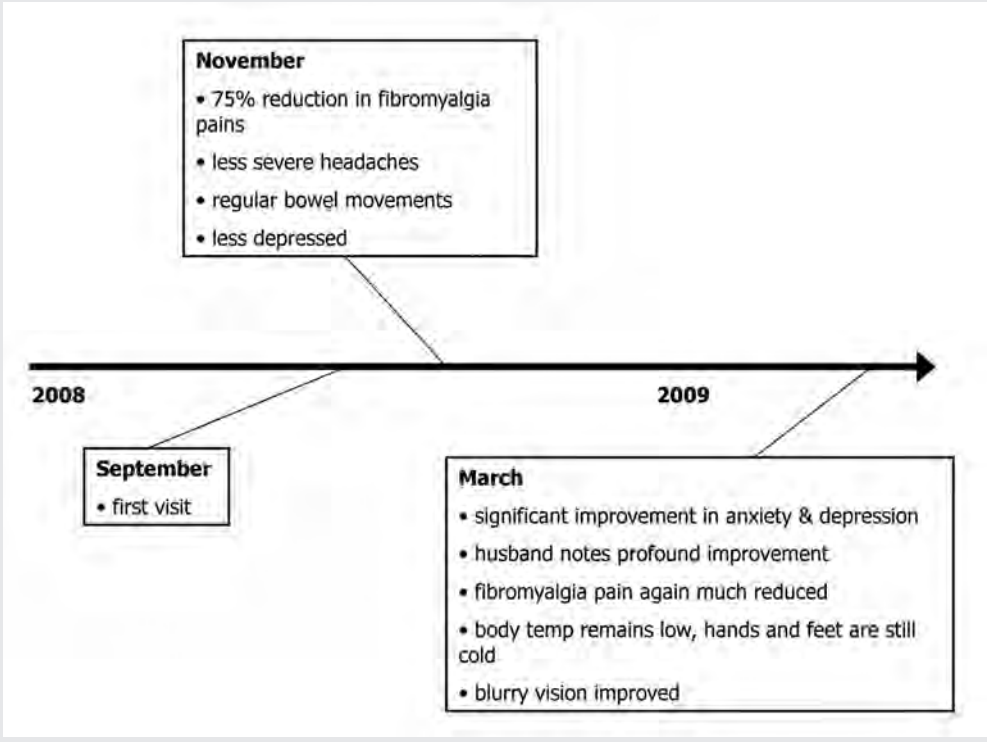
Treatment

Initially I gave her a strong multi-mineral/vitamin, vitamin C 1,000 mg three times daily, vitamin A 20,000 IU, a 3:1 EPA/DHA supplement, an adrenal botanical (eleutherococcus, sarsaparilla, rose hip, hawthorne, alfalfa) without licorice twice daily and magnesium glycinate at bedtime. She did a gluten-free trial. Iron complex was added in November 2008 and 5-hydroxy-tryptophan (5-HTP) 50-100 mg at bedtime. A thyroid botanical protocol (blue flag, guggulipid, fucus) was added in March 2009. (**Figure 2**, below)

Summary

The root cause of her condition was likely adrenal stress and inadequate vitamin-mineral nutriture leading to secondary thyroid compromise. Note that some people have compromised thyroid function that re-

Figure 2. Symptom changes during treatment of Case 2



covers with adrenal treatment.[3,9] By addressing these issues she experienced significant improvement in anxiety and depression. Her Hoffer-Osmond Diagnostic (HOD) depression score (DS) dropped from eight to three (normal range) and her husband noted she was a "different person." She had a 75% reduction in fibromyalgia-related pain symptoms within two months. She was better off gluten. Her maintenance treatment at last visit was minimal and ongoing.

Case 3: Schizophrenia, male, 18 years old

This athletic 18 year old had experienced auditory hallucinations for six months. This was a clear medically diagnosed case of first-episode schizophrenia. The visual hallucinations had recent high intensity suggesting progression of schizophrenic pathology and associated neuronal degeneration. He was withdrawing from society. His history showed Epstein Barr Virus (EBV) infection two years prior; a potential trigger for schizophrenia. [Viral infections from EBV and other viruses have been implicated as potential triggers of schizophrenia].[10] His diet was carbohydrate dominant. He was constipated with only one movement per day and low volume. The neuroleptic Risperdal was provided in hospital two weeks prior to the first visit and discontinued. (**Figure 3,** p.9)

Assessment

• Vitamin B_3 dependant trend – the default of all schizophrenias due to unregulated spikes in brain neurotransmitter with residual transient oxidized species similar to mescaline or LSD-25; the "Adrenochrome Hypothesis" was the first biochemical theory ever presented in the history of psychiatry.[11]
• No tissue heavy metals; inorganic and organic blood mercury toxicity was not ruled out
• Poor sugar regulation
• High liver enzymes, a transient issue that was present pre-treatment and probably due to medication
• High fasting blood homocysteine (undermethylated)
• Low body temperature (possibly due to thyroid weakening secondary to post-viral stress)
• Significant systolic blood pressure drop from 108 lying to 94 on standing (sluggish adrenal; typical in weakened thyroid metabolisms) with a resting pulse of 60.

Treatment

At intake I started this patient immediately on 3 g of B_3-HEXA and 3 g of vitamin C. I also provided a potent multi-B complex, multi-mineral, EFA with 2g of EPA, an adrenal botanical twice daily, and a 40% protein diet. At the second visit I added a thyroid botanical protocol with iodine, B_{12}/folic acid (1 mg of each) trans-dermal gel, 2,000 mg of betaine HCl, and 30 mg of zinc. A gluten-free trial was recommended as well. (**Figure 3**, p.9)

Summary

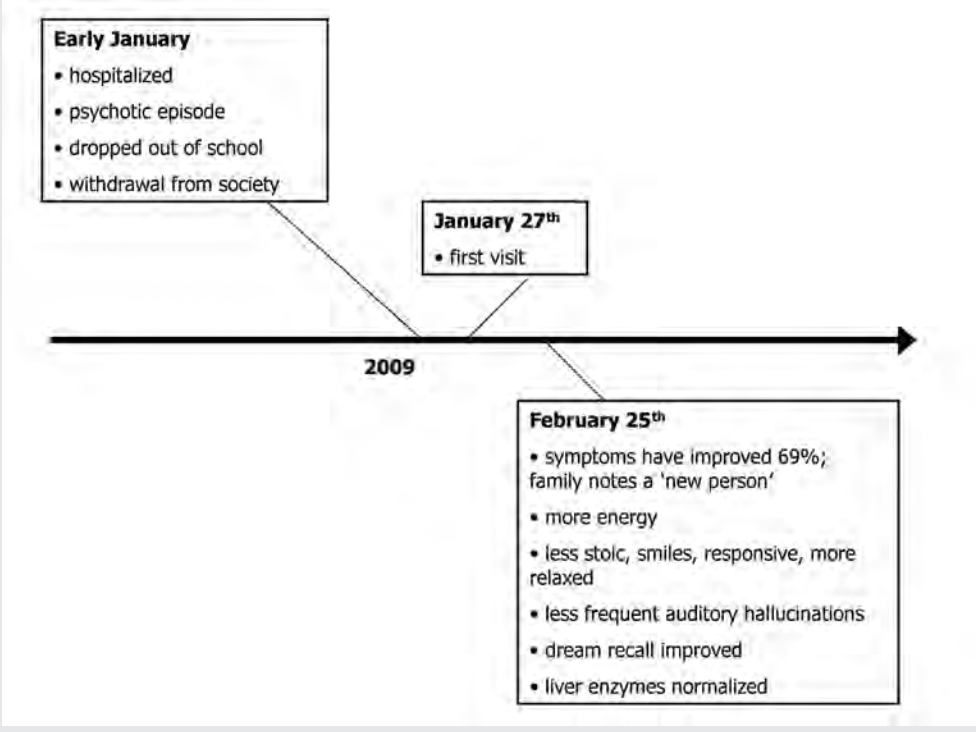
The root cause of the biochemical imbalance in this case is foremost a vitamin B_3 dependency followed by under-methylation syndrome, protein deficiency contributing to glycemic imbalance, hypoadrenia, and low thyroid metabolism. This patient experienced a 69% reduction of symptoms in one month as was indicated by a HOD Total Score (TS) drop from 59 to 18. His HOD Perception Score (PerS), indicating hallucinatory intensity, dropped from 18 to 3. Within one month he only heard voices 1 to 2 times a day versus constantly. His HOD DS dropped by 100% and he was smiling! Niacin (the B_3-HEXA form in this case) addressed the 'negative' symptoms, something rarely seen in schizophrenics sedated on neuroleptics. The results seen here are not uncommon in first-episode cases that are not exposed to neuroleptics (i.e. neuroleptic-naïve) for a significant period. We call these patients "good niacin responders."

Note that the response is more dramatic early in the course of the disease when brain tissue structure is not as compromised. The prognosis in such cases is good when treated with optimal niacin-therapy, and recovery approaches 90% in the first year of treatment.[12] Recovery here is defined as becoming free of symptoms; being able to integrate well with

family, friends, and society; and returning to previous levels of functioning in terms of vocation or career.[13] Ultimately these healthy and often bright people will recover and contribute to society by paying taxes and supporting our countries' Gross Domestic Product. There is nothing better than helping one of these patients and seeing changes unfold first-hand with a gradual fading of family turmoil. Very few people realize how much the family suffers and yearns for that former person to reveal themselves again.

Schizophrenia need not be a life sentence of relapses, "remissions" and drug side effects. Poor conventional prognosis repeatedly delays diagnosis as psychiatrists are often loath to make such conclusions without first exhausting all other differential diagnoses–schizoaffective disorder, personality disorder, bipolar disorder, mood disorder with psychotic break, etc. It is not remarkably difficult to diagnose schizophrenia and there should not be a cloud of skepticism in doing so. We see so many schizophrenics presenting often too late on one or more psychotropics with minimal if any improvement. In my experience, chronic patients can recover up to 40-60% with targeted orthomolecular therapy after which further improvement is dependant on their ability to withdraw from medications as tolerated.[14-17] If they had been treated early on with appropriate niacin therapy this would not be the case in the vast majority.[18,19] I consider optimal orthomolecular protocol as a first-line treatment in first-episode neuroleptic-naïve schizophrenia. It can reverse over the next 70 years upwards of 55 million cases; that is, 80-90% of the approximate 70 million cases worldwide at our current population size, which is now fast approaching the 7 billion mark.

Figure 3. Symptom changes during treatment of Case 3



Is 'Recovery' Possible with Neuroleptic Treatment?

In my discussions with psychiatrists, I have never heard them say that conventional neuroleptic treatment will enable more than 10-25% of patients to 'recover'. Sixty-six percent of schizophrenic cases do not achieve a full remission of symptoms with neuroleptic treatment.[20] Thirty-three percent of cases suffer "persistent cognitive disturbance and associated deficits of mood, motivation, and behavioural responses."[21] After a psychotic episode, 30-40+% of compliant neuroleptic-treated schizophrenics who have had a "positive" response will relapse within 1 year.[22,23] The highest suicide risk occurs in the first 5 yrs – the "critical period."[24] Fifteen percent of cases will successfully complete suicide.[25] The best case scenario is first episode early intervention with the lowest effective neuroleptic dose and adjunct psycho-education, relapse prevention, and psycho-social intervention.[26] These "patients are more likely to be taking medication at the end of three years… more compliant… more likely to be prescribed atypical medication… more likely to have returned to work or education… more likely to remain living with their families… less likely to suffer depression to the extent of requiring anti-depressants… commit less suicide attempts… less likely to suffer relapse and re-hospitalisation… less likely to have involuntary admission to hospital." A double blind study in the USA (the PRIME study) allocated prodromal schizophrenic patients to olanzapine versus placebo groups. After 1 year of treatment, researchers found olanzapine to be of no advantage over placebo; both groups experienced similar progressions and worsening of psychotic symptoms.[27] Patients commonly have poor quality of life and the majority remain dependent on parents and few have skilled or paying jobs.[28]

Case 4: Schizoaffective disorder, male, 41 years old

This patient experienced three nervous breakdowns with paranoia (1982) and mental confusion (1985, 1987). He was on and off neuroleptics and anti-depressants. In 1988, he was given a diagnosis of schizoaffective disorder. He reported he had been unable to work since 2006 while on Olanzapine; Olanzapine interfered with his memory and executive processes including problem solving.[16,17] He had Olanzapine-induced diabetes and was overweight. His insomnia was destabilizing. He felt better taking niacin three weeks prior to the first visit. His HOD Ratio Score (RS) revealed greater dominance in perceptual (receipt of sensory information) versus mood dysfunction which is a typical niacin dependant trend. He had glucose-6-phosphate dehydrogenase deficiency. (**Figure 4,** p.11)

Assessment

- I consider this patient vitamin B_3 dependant until proven otherwise (i.e. if niacin treatment does not produce a profound response with sequentially higher doses)
- High fasting glucose, iatrogenic dysglycemia
- High cholesterol and triglycerides
- Gluten-intolerant symptoms
- Copper deficient trend from hair and blood assessment
- Protein catabolism trend from hair and blood assessment
- No tissue heavy metals; blood inorganic and methyl mercury had not been ruled out
- Normal body temperature

Treatment

Pure niacin was provided at 2 g, three times daily at the outset along with a B-complex and a multi-mineral, a botanical complex for glucose regulation (syzygium, oplopanax, sylibum, nopal), an EFA with 2 g of EPA, magnesium 400 mg, and a 40% lean protein diet. In the second visit I raised the B_3 dose to 4 g three times a day (pill then later powder form). [Sometimes clients need and want to be maintained at high B_3 dose levels]. Blood copper-zinc status, liver enzymes, and uric acid levels are checked annually in these cases. He had no liver enzyme elevations on high dose B_3 and his uric acid (which competes with niacin for urinary excretion) levels remained normal.[29] I see liver

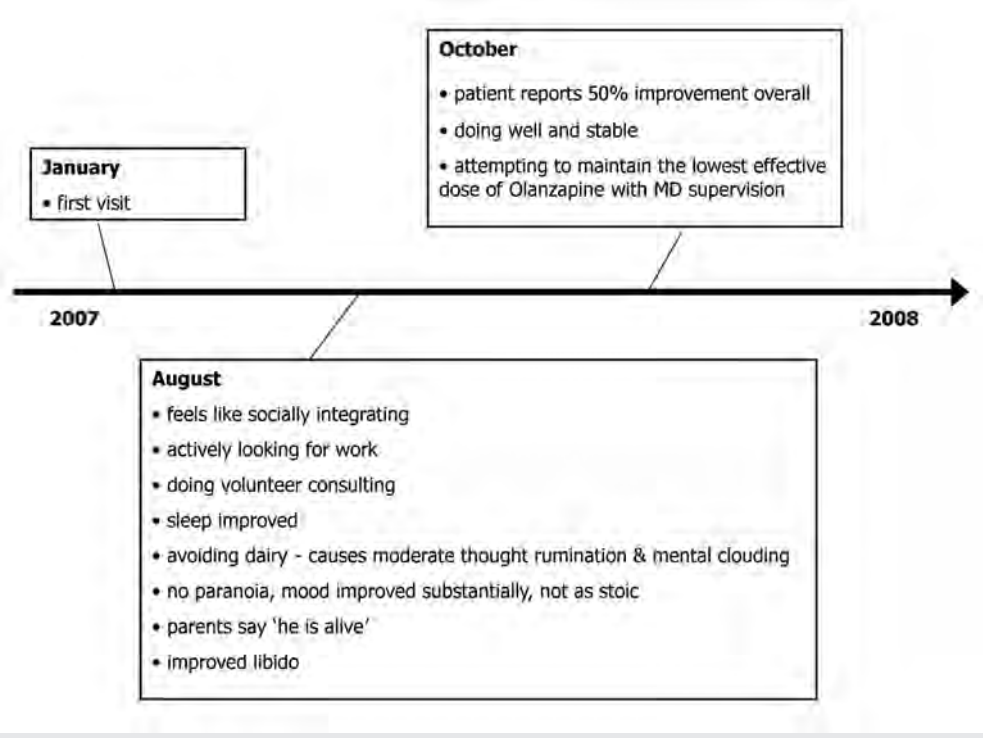
enzyme elevations in less than 5% of niacin treated schizophrenics and in all cases, levels normalized after a proper 1 to 2 week washout; this confirms what other researchers have found, that vitamin B_3 does not cause structural damage but merely speeds liver function and sometimes in that process spills excess microsomal liver enzymes. In contrast, medication-related enzyme elevations can linger at high levels for months. Lecithin and betaine are excellent at addressing liver enzyme elevations when and if that occurs.[29]

I added P-5-P 50 mg and pyridoxine hydrochloride (B_6-HCl) 200 mg, vitamin E 400 IU, methyl-B_{12} 1.5 mg and folic acid 2.4 mg daily. His dream recall returned within a month of B_6-HCl and P-5-P dosing; dream recall had been absent for a decade. He felt better off gluten and I recommended he maintain this. In the third visit I switched him to intramuscular B_{12} and folic acid, added iron and increased manganese. Also, I started a custom supplement series to improve compliance and the average dose was seven pills twice daily. I included vitamin D 2,000 IU, betaine (TMG) 1.8g, chromium 1,200 mcg, 6 mg copper, a multi-mineral base, and R-lipoic acid 150 mg daily. In the forth visit I added 1g vitamin C twice daily. I added a thyroid botanical protocol (blue flag, guggulipid, fucus, ashwaghanda) in the fifth visit. In the sixth visit I upped the vanadium dose. I recently recommended gymnema for sugar regulation and he is maintained well on the custom supplement. (**Figure 4**, below)

Summary

This is a vitamin B_3 dependant case. The RS score dropped from a high seven to five between August and June of 2007 which shows

Figure 4. Symptom changes during treatment of Case 4



the progression of reversal of perceptual dysfunction as the dominant symptom. Within seven months his paranoia totally dissipated, his mood improved substantially, and he was no longer as stoic. He was able to work and became a functional member of society within one year of treatment. His physical improvements were also consistent with orthomolecular treatment which addresses all body systems. Medication side effects were much reduced and the patient continued to improve. A maintenance protocol is in effect. He continues to do well and is active in the work force.

Risks of Neuroleptic Sedation

Neuroleptics sedate brain areas in need of correction and dilute effect of nutrients by reducing the amount of viable tissue available to be acted upon. In my experience, orthomolecular treated patients who have been on neuroleptics longer than six to twelve months plateau at the landmark 40-60% improvement level. Further improvement is dependant on maintaining lower effective doses of medication.[30,31] Many of these patients have difficulty remaining stabile on lower neuroleptic doses due to symptom reemergence.

Olanzapine and other neuroleptics are associated with rebound psychosis or super sensitivity psychosis (SSP) which is a receptor decompensating effect secondary to neuroleptic withdrawal.[32-35] Many psychiatrists argue that symptom reemergence after withdrawal of neuroleptics is due to dropping the dose of a drug that is stabilizing. That being said, you cannot rule out the addictive hold secondary to altered follower neuron receptor numbers. That is, with neuroleptic follower neuron blockade there is a natural compensation to upregulate follower neuron receptors and produce greater numbers of receptors that are also blocked by the neurolpetic if provided in sufficient amounts. When the neuroleptic drug dose is later dropped the receptor blockade is lifted and neurotransmitter in the cleft is left free to bind to the follower neuron with greater receptor numbers. The end result is excess firing of the follower neuron which results in classic denervation super-sensitivity and sometimes neuronal death; two excellent papers by Gur et al and Chakos et al discuss the finding of brain tissue loss.[36,37] More researchers need to do studies like this to explore further the potential for neuroleptics to cause iatrogenic brain damage and psychosis secondary to withdrawal. Neuroleptic rebound potential should be part of the pretreatment discussion between the psychiatrist, patient and family members.

With prolonged neuroleptic administration tardive dyskinesia can appear. This is not uncommon. Symptoms involve tongue darting, mouth smacking and extremity jerking or writhing. This can be permanent if cholinergic interneurons of the striatum are damaged. This is thought to be concurrent with episodes of SSP.[38]

Neuroleptics are associated with metabolic syndrome and premature mortality. Several reviews describe neuroleptics (especially atypicals) causing metabolic syndromes including diabetes, weight gain, hyperglycemia, dyslipidemia and hypertension. In discussions on neuroleptic outcome some researchers mention "[p]ersons with major mental disorders lose 25 to 30 years of potential life in comparison with the general population, primarily due to premature cardiovascular mortality."[39,40]

Case 5: ADD/dyslexia, male, 42 years old

The patient was a scattered unfocused adult. He had moderate to severe adult attention deficit disorder (ADD) with a high HOD TS of 60, a high DS of 10, and a high PerS of 13. He was an idea person, good at business strategy. He was dyslexic with sequential information tasks as required for following legal contracts. He had good visuo-spatial memory and math skills (common in right-brained dominant ADD, children and adult), but poor short term memory and critical reasoning. He had restless legs with sleeplessness that was worse with ADD medication. He was using a proton pump inhibitor for the gastric reflux of a hiatal hernia. (**Figure 5,** p.13)

Assessment

• Normal fasting blood homocysteine
• Low blood iron with low normal saturation
• No tissue heavy metals; inorganic and organic blood mercury not ruled out
• Hypoglycemic trend

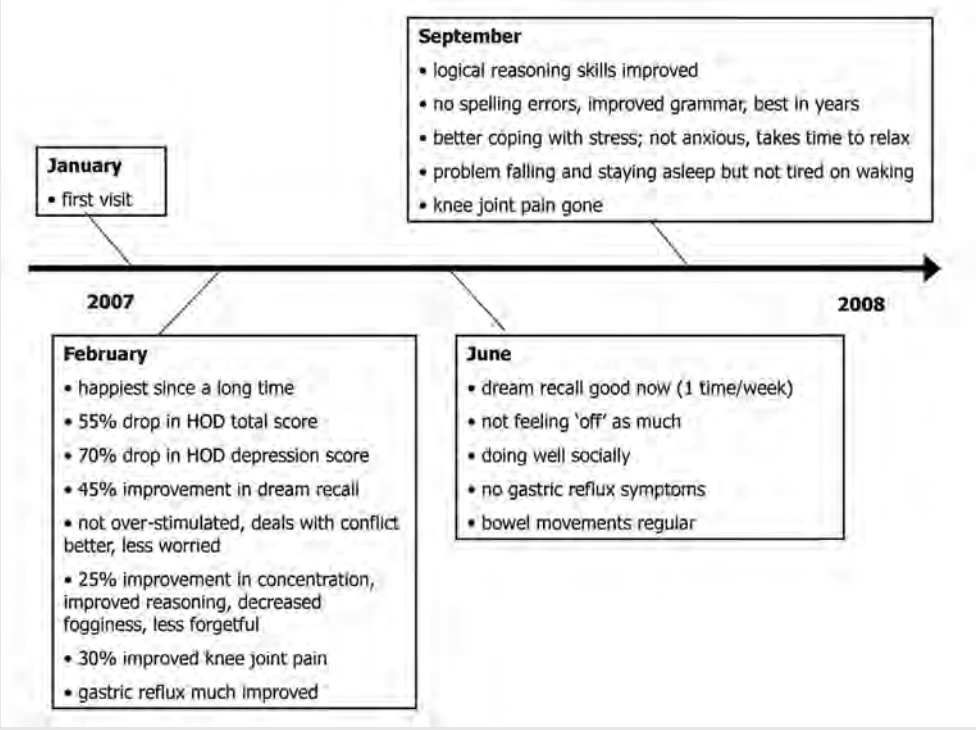
Treatment

I placed this patient on a 40% lean protein diet and a daily regimen of B-complex 100's, B_3-HEXA 1.5 g, vitamin C 3 g, P-5-P 50 mg, B_6-HCl 200 mg, zinc 30 mg, chromium 800 µg, magnesium 800 mg and an EFA with 3:1 EPA/DHA. On the second visit I added an adrenal glandular and betaine HCl. On the third visit I added vitamin E 400 IU, a low dose iron complex and had him maintain a gluten-free diet which he felt better on. By the fourth visit his metabolism picked up and I tailored back his supplementation by omitting the B-complex, zinc, B_6 HCl, betaine HCl and vitamin C, and implementing a custom multi-mineral-vitamin (five pills a day) for fast metabolizers with a B-complex weighted in B_{12}-methyl 2 mg (no folic acid), copper 2 mg, chromium polynicotinate 600 µg, and calcium hydroxyappatite 200 mg. On the fifth visit after a lengthy trip overseas I provided him with a potent probiotic to normalize digestion. On the sixth visit I added a botanical for glucose regulation (Syzygium, Oplopanax, Sylibum, Nopal). (**Figure 5**)

Symptom Changes Summary

This ADD adult experienced significant improvement in thinking and mood. The over-stimulated ADD symptoms including his dyslexia were no longer present. Orthomolecular treatment allowed him greater work performance and greater overall well-

Figure 5. Symptom changes during treatment of Case 5



being. His gastric reflux improved quickly and was reversed within six months. The root imbalance in this case was likely adrenal and glycemic imbalance with gluten intolerance.[4] He was doing well enough at the last visit to continue on a maintenance regimen. Many ADD cases present with copper toxicity which in the initial stages forces a metabolism to shift from fast to slow. Fast metabolizing ADD cases tolerate Ritalin or Concerta but eventually become refractory to, and aggravated by these stimulants.

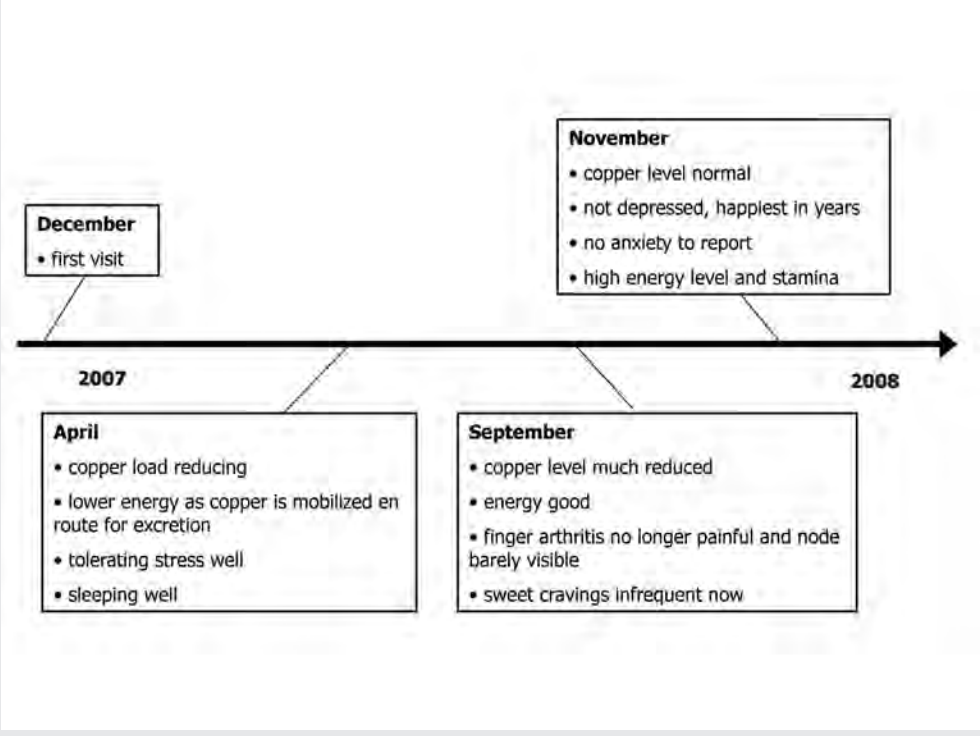
Case 6: Chronic depression, female, 56 years old

This client had struggled with depression since her teens. She had negative thought rumination at age 17. Since age 10 she noted having PMS mood swings as well as headaches and migraines. She had menstrual disruptions that suggest potential estrogen progesterone imbalance. She was stoic with suppressed emotions and irritability. She had a long-standing lack of appetite history which I determined, with laboratory results, was due to zinc and iron deficiency. She was light-headed without breakfast. She had gestational iron deficiency unremitting and post-partum changes including anxiety, negative thoughts, inability to tolerate stress, weeping, screaming, depression, fatigue, apathy (worse winter, worse premenstrual), and sweet cravings. In her mid-40's she developed osteoarthritis of the fingers and knees and severe insomnia with less than four hours of sleep a night. She had occasional suicidal propensity and significant despair. (**Figure 6,** below)

Assessment

• Severe high hair tissue copper with low zinc/copper ratio and iron/copper ratio.

Figure 6. Symptom changes during treatment of Case 6



[Note that copper dominance is linked to estrogen dominance and consequent relative progesterone deficiency trends.[41,42]]
• High normal fasting blood homocysteine
• Low thyroid and adrenal slow metabolizer trend as indicated by hair tissue ratios
• Low normal body temperature despite 'normal' TSH of 1.7
• Hypoglycemic hair tissue trend with normal fasting blood sugar
• Protein catabolism confirmed in the blood with elevated protein waste ratios
• Low ferritin iron levels

Treatment

I treated this patient with daily supplemental B-complex 100s, sequentially higher doses of zinc (up to 120 mg daily), manganese 30 mg, chromium 1,200 mcg, an adrenal glandular, magnesium 600 mg, iron 25 mg, vitamin C 1,500 mg, potassium 300 mg (to oppose calcium), betaine HCl 1,500 mg and vitamin E 400 IU. I also recommended a 40% lean protein diet. In the third visit I added a thyroid botanical protocol (blue flag, guggulipid, fucus, ashwaghanda). Her metabolism had sped up nicely and we cut back to a maintenance supplement regimen. I recommended she stay off gluten as she felt much better and had higher energy without it. Her maintenance regimen was zinc 30 mg daily, a B-complex, low dose adrenal and thyroid botanicals, and a low dose multi-mineral (without copper), iron, and magnesium. (See **Figure 6**, p.11)

Summary

This is a clear simple case of uncomplicated hormonal copper toxicity.[4] Consistent with copper normalization I saw significant improvements in mood stability and stress tolerance. Copper excess is associated with estrogen dominance and a relative progesterone (natural anti-depressant) deficiency which contributes to PMS, irritability and depression. Physical improvements include reduced arthritic pain, and increased energy and stamina. Recently she went off zinc and the low dose thyroid botanical and her hair tissue copper level crept back up with resultant resurfacing of depression. The maintenance protocol is needed to keep copper at bay.

Discussion on Orthomolecular Treatment Response

Realigning specific biochemical-nutrient imbalances is a foundational tenant of orthomolecular treatment. Herein we see the use of targeted lab tests and other eclectic naturopathic assessment techniques. This approach is advanced and helps us define biochemical-nutrient imbalances that are in dire need of being corrected. By reversing targeted biochemical-nutrient imbalances we see a direct positive correlation with patient improvement. These imbalances are widespread in mental health pathology. Medication effects have important ramifications on outcome as discussed herein.

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