

Chronic Sinusitis: A Major Perpetuating Factor?

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Chronic Sinusitis: A Common Perpetuating Factor?

In far too many instances, in my opinion, researchers have found one potential initiating or perpetuating factor associated with fibromyalgia syndrome (FMS) and assume that they have found THE CAUSE or THE CURE. In most cases, the development of FMS or full-blown bodywide chronic myofascial pain (CMP) is multifactorial. There may be a heterogeneous collection of factors that initiate and/or perpetuate the central sensitization if it is FMS. I have found that the key to success in dealing with FMS, just as in dealing with CMP, is to identify as many perpetuating factors as possible and control them as much as possible. I think I have found a hitherto unsuspected cause of, or at least contributor to, some cases of central sensitization, and perhaps a successful therapy for it.

Research indicates that FMS may be central nervous system (CNS) sensitization resulting from an immune response leading to spinal glial activation (Staud 2004). At the Focus on Pain (Travell) Seminar in Orlando, Florida, in 2003, Dr. Linda Watkins, Director of the Interdepartmental Neuroscience PhD program, explained her research at the University of Colorado in Boulder. Her team is investigating the onset of chronic pain and the mechanism causing central sensitization. They have found that the CNS can be sensitized by many factors, including infection and trauma, but the key to the sensitization is the activation of spinal glial cells. [More on Dr. Watkins' presentation is found elsewhere on the Focus on Pain handout on this website.]

A team lead by Joyce DeLeo, MD, at Dartmouth-Hitchcock Medical Center in Lebanon, NH, found that the CNS neuroimmunological cascade response leading to chronic pain states may also be linked to opioid tolerance (DeLeo, Tanga, Tawfik 2004). They found that the changes in CNS glial cells and proinflammatory cytokines that contribute to central sensitization can decrease the effectiveness of opioid medications. This team also found that neuroinflammation and interstitial swelling can be integral parts of central sensitization. I have found that interstitial swelling can be tied to pain levels in some patients with FMS and CMP. Also of interest is their observation of cellular adhesion molecules in the lumbar spinal cord following peripheral inflammatory stimuli. This may indicate a similar process occurring in the central nervous system similar to the myofascial cellular adhesion in response to mechanical or biochemical trauma. This team provided another piece of the puzzle.

Another piece, and a big one, came with the presentation of a paper on March 23, 2004, at the annual meeting of the American Academy of Allergy, Asthma and Immunology in San Francisco.

A Mayo Clinic team of physicians lead by David A. Sherris found that airborne fungi commonly found in the mucus linings of the sinuses can adversely affect individuals prone to chronic sinusitis. These fungi provoke an immune response, which in turn attacks the fungi, resulting in symptoms of chronic sinusitis. Could this immune response provoke central sensitization? The team ran a placebo controlled, double blind pilot study using Amphotericin-B intranasally. Seventy percent of the linings of the sinus membranes of those patients on the drug decreased in thickness, and the symptoms abated. Approaching chronic sinusitis as an immune disorder creates a different perspective.

Dr. Sherris, now interim chair of the University of Buffalo Department of Otolaryngology, is using the Amphotericin B nasal spray the team used on his patients. He reports on WebMD that this study may indicate the first ever treatment for the cause of chronic sinusitis, rather than a symptomatic approach.

In 1992, an article linked chronic rhinitis to FMS (Cleveland, Fisher, Brestel, et al. 1992). This team studied 47 consecutive patients with allergic rhinitis in a general allergy clinic and found congestion in 91%, rhinitis in 87% and postnasal drip in 83%. Forty-nine percent met the ACR criteria for FMS, and the team concluded: "Rhinitis...is associated with fibromyalgia and may be an underdiagnosed but important causative factor."

One review shows how neurogenic mechanisms can complicate sinusitis (Baraniuk 2001). Stimulation of nasal sensory nerves leads to pain and congestion. Pain receptors cause release of substance P, stimulating mucosal defense mechanisms. Sympathetic dysfunction then can cause sinuses to fill and the mucosal lining to thicken. Fibromyalgia is associated with sympathetic hypersensitivity.

I have been working on a review of 200 patient interviews picked at random from over 1000 interviews done between 1992 through 1999. [This review will be posted on the website.] These patients had diagnosed or suspected FMS and/or CMP. The patient interviews were reviewed to identify and assess possible symptom clusters and patterns. Almost all of the patients had at least one myofascial trigger point (TrP), and most of the patients had either CMP or a combination of FMS and numerous TrPs. Of the 200 patients, only 11 patients did not have either FMS or CMP. The most common symptom listed was post-nasal drip. Of the 189 patients with either FMS and/or CMP, all 189 had post nasal drip. This result was unexpected. The post nasal drip was frequently accompanied by sinus congestion and runny nose.

Specific head and neck TrPs can cause drippy nose and congestion. Trigger points in the sternocleidomastoid muscles (SCM) alone can cause, among other things,

coordination problems, proprioceptor dysfunction, dizziness, imbalance, neck soreness, a swollen-glands feeling, runny nose, maxillary sinus congestion, tension headaches, eye problems (tearing, blurred or double vision, inability to raise the upper eyelid, dimming of perceived light intensity), spatial disorientation, postural dizziness, vertigo and nerve impingement (Simons, Travell, Simons 1999). Many of these symptoms mimic chronic sinusitis. A picture was developing.

Late in 2003 I had been given another piece of the puzzle, although I didn't know it at the time.

I met with Lawrence Funt, DDS, MSD, Director of the CranioFascial Pain Center in Bethesda, MD. During an afternoon together, we discussed Janet Travell and the founding of myofascial medicine. We also discussed the Funt-Symptom Index (Funt 1988). During the course of a long career in pain management, Dr. Funt had noticed patterns of symptoms that occurred in patients in sequence, according to age and length of pain history. His patients between the ages of 4 and 6 years experienced clenching of the jaw, stuffy ears and headaches. Symptoms progressed, and by age 21 to 30 there appeared, among other symptoms, maxillary sinus pain that became increasingly frequent. This later fit into the puzzle. We also spoke of biofilms.

Biofilms are becoming increasingly important in medicine, science and technology. Bacteria and other organisms have developed a successful survival strategy. They grow in a slimy mass, covering themselves with protective polysaccharides. These biofilms develop on the surfaces of medical devices, in air and water treatment systems, and in human bodies. Organisms in biofilms are remarkably resistant to anything you throw at them. Counter agents need to get through the slime and kill all the organisms, or the buggies just multiply themselves right back, often with a resistance to the first counteragent used against them. The July 4, 2003, edition of *Science* gave a good description of biofilms as a community, with a layer of slime covering "...the entire community, protecting it from attacks by the body's immune system." That piece fingered biofilms as the culprit in bladder infections. Osteomyelitis, Cystic Fibrosis, prostatitis, and middle ear infections are biofilm infections (Costerton 1998).

Organisms in biofilms are protected against antibacterial chemicals and environmental predators. Nutrient limitations and the build-up of toxic metabolites favor the formation of biofilms (Donlon, Costerton 2002). This occurs often in FMS (Starlanyl and Copeland 2001) and in the area of a myofascial TrP (Simons, Travell, Simons 1999). Biofilms are associated with increased fibronectin, coaggregation and adhesions, and the production of endotoxins. Biofilms can also be formed by mycobacteria (Hall-Stoodley, Keevil, Lappin-Scott 1999). Organisms forming biofilms are resistant critters. They could be perpetuating factors in a number of cases of FMS and CMP.

Some of my review patients with long-standing symptoms had mentioned childhood dental problems. Others recalled frequent bouts of sinusitis and earaches. Sternocleidomastoid TrPs are common and cause a lot of symptoms that can be mistaken for sinusitis. Trigger points in the longus colli muscle can cause sore throat, persistent tickle in throat, and a lump in throat. Deep anterior neck muscles can refer to the laryngeal area. Cricothyroid TrPs cause regional muscle pain on talking, and a sore throat. Other TrPs can be responsible for ear pain, stuffiness of the ear, and temporary hearing impairment. One study found that of 111 patients with suspected chronic maxillary sinusitis, only 56% had that diagnosis verified. In 61 % of the patients in whom it could not be verified, dental infections and/or myofascial pain were the most common cause (Lindahl, Lelen, Ekedahl 1982). Possible patterns were emerging.

Chronic sinusitis (or its symptoms) is frequently treated by antibiotics. Patients often reported frequent antibiotic use during periods of their lives. The use of antibiotics would enhance fungal problems, although they might reduce congestion if there were a secondary bacterial infection. The drippy nose and congestion would return, because the fungi and immune response would remain.

Patients often mentioned chronic yeast infections. Women especially reported this, although some men and boys also mentioned gastrointestinal yeast problems or thrush. Many patients also had symptoms of reactive hypoglycemia or insulin resistance. Insulin resistant states provide a fertile home for fungi and yeasts. A subset of patients also reported mold sensitivity, although this was not one of the parameters of the review. Several patients also reported treatment with antifungals Nystatin and/or Diflucan. Some required Nystatin to be administered concurrently with any antibiotic therapy to avoid further yeast infection. Sensations of CNS swelling were reported, often linked with cognitive deficits. Some mentioned these worsened when sweets or other heavy carbohydrate meals were eaten, and some described easing of these symptoms with use of diuretics and/or higher protein diets.

Nystatin works in the gastrointestinal system to destroy yeast there. Diflucan works systemically, but what about the blood-brain barrier? This protective barrier prevents molecules from crossing over to the CNS. It is also a pesky obstacle to effective therapy of the CNS, as many medications can't cross it. What if an immune response to fungi, possibly in biofilm, were a common instigating or perpetuating factor of central sensitization? Why would the Amphotericin B nasal spray used by Dr. Sheris and his team work better than Diflucan? Enter the last piece of the puzzle — I needed to try an experiment on my own.

Dr. Gunter Oberdörster and his team have conducted a study to see if an inhaled ultrafine particle could cross along the olfactory nerve into the olfactory bulb (Oberdörster, Sharp, Atudorie, et al 2004). They found that a particle could move into the CNS from the nasopharyngeal area. The study is not yet published but is in press and is available on the web. It was done on ultrafine particulates, but it

indicates to me that a nasal spray might have a better chance of reaching the CNS and any fungi within.

The puzzle, while by no means complete, gave me enough for action. I was still hesitant, because Amphotericin B is not a medication to be used lightly. I had no experience with it as a nasal spray. I called my compounding pharmacist, George Roentsch, at The Apothecary in Keene, NH. He told me that compounded Amphotericin B nasal spray was generally used at 20 mcg/ml, required refrigeration and had a short shelf life, but his experience was that the spray used 5 times a day in each nostril for two weeks was sufficient to bring relief of symptoms without side effects. I spoke with my primary care physician and my allergist as well as my local myofascial trigger point doctor. With my history of severe FMS and CMP, plus Metabolic Syndrome, sleep apnea, a long history of mold and yeast allergies, immune therapy for multiple fungi, and frequent interstitial swelling, they agreed that I seemed like a good candidate for this therapy. I had other allergies and knew that this would do nothing for them, but I hoped that any CNS mold component and associated immune response might be brought under control.

I first went on a course of Diflucan therapy, with no change in symptoms. After allowing my body recovery time from the Diflucan, I went on the Ampho B nasal spray for 2 weeks. The deep congestion that I hadn't been able to relieve since they took PPA (original Contac formula) off the market went away. The fluids in my body tissues are rearranging themselves. The TrPs are becoming more available to treatment, although I noted a phenomenon that others have reported.

As the central sensitization lessened, the TrP symptoms became more noticeable. The increase in myofascial pain was considerable at first, but I have TrPs in almost every layer of every muscle. My myofascia is unwinding, satellite and secondary TrPs are being eliminated, and bones are shifting back into a more normal position. This is not a comfortable process, but it is a necessary one to resume a higher standard of health. My pain level is down with less medication. I believe that one perpetuating factor in my life has been found and brought under control, at least somewhat. I have multiple allergies and the symptoms were further aggravating several of my medical conditions. After further research, I decided to go for retesting and resumed allergy shots."

My blood levels are regularly monitored by Dr. Lynne August at Health Equations. We had been unable to get the cholesterol and triglycerides down with diet alone, and my health team agreed that the cholesterol could well be protecting me from something and I did not wish to return to cholesterol medications. After the Ampho B nasal spray therapy, my triglycerides dropped from 261 to 155, my cholesterol dropped from 350 to 287, my cholesterol/HDL ratio normalized, and the toxin load dropped. This is only one test, but the only thing that changed was the nasal spray therapy. Time and later blood testing will tell if the Metabolic Syndrome can be taken off my co-existing conditions (and perpetuating factors). This is all very recent, and I don't know where my health level will stabilize. I

know that there are other hidden perpetuating factors. The good thing about that is when a perpetuating factor is found, something can often be done. It just takes a little detective work and the right pieces of the puzzle.

This therapy is not a cure for FMS or CMP. Chronic sinusitis caused by an immune reaction to fungi *may* be part of the central sensitization process in *some* cases of FMS. It may be a perpetuating factor in CMP. We still don't know how safe this therapy is.

This is all very new. It will take time and money for researchers to provide these answers. If patients have an indicative history, such as frequent yeast infections, reactive hypoglycemia or insulin resistance, mold sensitivity, resistant congestion and post nasal drip, I believe that this is a therapy that is worth considering.

Update: Current information indicates that intestinal permeability and insulin resistance are often some of the causes contributing to chronic illness. (*Textbook of Functional Medicine*, Jones DS, Quinn S, editors, 2005-6; see "functional medicine.org" on the web). More about this on a handout "Healing Dysfunctional Gut" on this website. In many cases, excess mucus production contributing to post nasal drip may be the body's attempt to protect against gastric reflux (GERD). Many cases of GERD may be silent, without obvious heartburn symptoms but causing excess mucus, chronic cough, and disruptive sleep. If you have unrestorative sleep, GERD, silent or active, may be part of this, and a sleep study that includes gastric monitoring may be what you need. Talk to your doctor about this possibility.

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