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Chiropractic Management of a Paralytic Autoimmune Disorder

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ABSTRACT

The following report outlines the clinical observation of a patient presenting with a chronic severe paralytic autoimmune disease. Alleviation of signs and symptoms is noted subsequent to corrections of aberrant arthrokinematic function of the occipito-atlanto-axial complex. A relationship between biomechanical faults in the upper cervical spine and the manifestation of abnormal autonomic neurophysiology is suggested as the initiating and maintaining factor in the pathophysiologic process of this patient's autoimmune disease.

Key words: Autoimmune, Infrared Imaging, Upper Cervical Spine

INTRODUCTION

Approximately one in five people, or 50 million Americans, suffer from autoimmune diseases. The term "autoimmune disease" refers to a varied group of more than 80 serious, chronic illnesses that involve almost every organ system. It includes diseases of the nervous, gastrointestinal, and endocrine systems as well as the eyes, blood, blood vessels, skin and other connective tissues. In all of these diseases, the underlying problem is the same – the body's immune system becomes misdirected, attacking the very organs it was designed to protect. The greatest percentages of those affected with this disease are women, with autoimmune conditions representing the fourth-largest cause of disability among women in the United States. (1,2)

Research indicates that autoimmune diseases are linked to a dominant genetic trait that is very common (20 percent of the population), and that it may present in families as different autoimmune diseases within the same family. For example, a mother may have SLE; her daughter, diabetes; her grandmother, rheumatoid arthritis. These studies are very clear, however, that the genetic predisposition alone does not cause the development of autoimmune diseases. It seems that other factors need to be present in order to initiate the disease process. (1, 3-5)

CASE REPORT

A 33-year-old female was referred to our center with the chief complaints of stocking-glove numbness, bilateral upper and lower extremity weakness and twitching, and overall fatigue. Her symptoms began six years previously upon awakening with numbness in the left fifth finger. The symptoms progressed quickly over the next eight weeks resulting in two hospitalizations and finally complete quadriplegia. She became terrified by the severity of her condition and wondered if she was going to die. The patient was seen by multiple specialists and had a battery of tests including several MRIs, CTs, spinal taps, and lab exams all without conclusive results. A trial of prednisone was initiated with some improvement noted. This lead to a suspected inflammatory disease process. Intravenous immunoglobulin (IVIG) therapy was tried with subsequent cessation of all signs and symptoms. From the success of the treatment, a diagnosis of autoimmune disorder with associated peripheral neuropathy was made.

At the time of consultation, the patient had been experiencing progressive stocking-glove numbness, bilateral upper and lower extremity weakness and twitching, and overall fatigue over the past three months. Since the onset of her condition six years ago, the patient had been receiving IVIG treatments, followed with a 10-day regimen of prednisone to counteract the side effects, every three months in order to alleviate the progressive symptoms and weakness. After each IVIG treatment, her symptoms would progress to the point where if another IVIG treatment were not rendered complete paralysis would return. She noted that the aspect of having her condition return to its initial state was terrifying. At this time she was due for another round of IVIG; as her upper extremity dexterity and ambulation was quickly deteriorating. Since her occupational demands included sensitive manual dexterity, the patient was very concerned about whether she should wait to see what the chiropractic care would do or begin another round of IVIG and prednisone.

Upon examination, the patient presented with a mild limp and slight shuffling gait. Vital signs, ear, nose, and throat examinations were unremarkable. Orthopedic examination revealed significant palpatory hypertonicity of the paraspinal musculature from the occiput to C4 bilaterally with a marked increase on the left. Bilateral palpatory hypertonicity was also noted in the anterior cervical musculature with a significant increase on the left. The patient demonstrated a reduction in active and passive right cervical rotation and lateral flexion. Her thoracic and lumbosacral evaluation was unremarkable.

Gross neurologic examination revealed a moderate loss of pain and light touch sensations in a stocking-glove distribution. Muscle strength deficits, with gradings of 4 to 4- of 5, were found in most of the muscles of the upper and lower extremities. A computerized paraspinal infrared analysis (neurologic imaging via a TyTron C-3000 Paraspinal Infrared Imaging System) was performed in accordance with thermographic protocol (6-8). A continuous paraspinal neurologic scan consisting of approximately 350

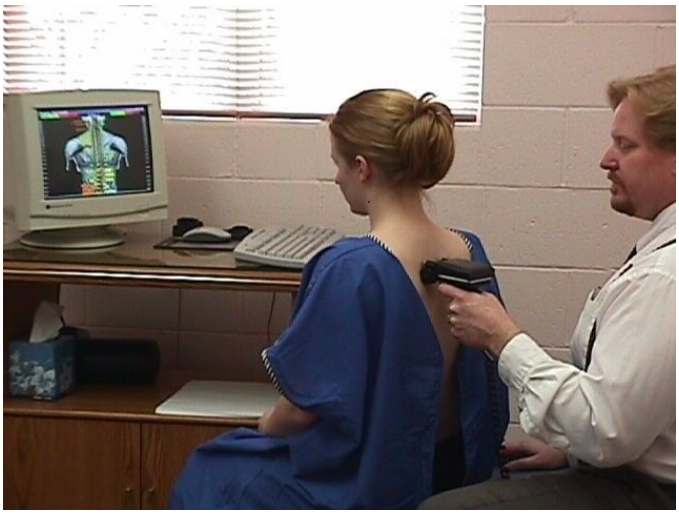

Figure 1

Figure 2

infrared samples was taken from the level of S1 to the occiput (Figure 1). The data was analyzed against established normal values and found to contain thermal asymmetries indicating abnormal autonomic regulation (9-12) (Figure 2). Since the cervical spine displayed abnormal thermal asymmetries, a focused neurologic scan was performed with approximately 85 infrared samples taken from T1 to the occiput (Figure 3). When interpreting these neurologic scans you will note the mild Delta-T findings (Figures 2 and 3 center graph field, or pre bar graph figures 4 and 5). In some patients with severe conditions, the degree of temperature differential is not excessive. As a minimum, a plotted graph denoting mild repeatable anatomic location differentials objectively indicates a lack of normal neurologic homeostasis. Since the neurologic scans in this patient showed objective findings of neuropathophysiology, existence of the vertebral subluxation complex was suspected.

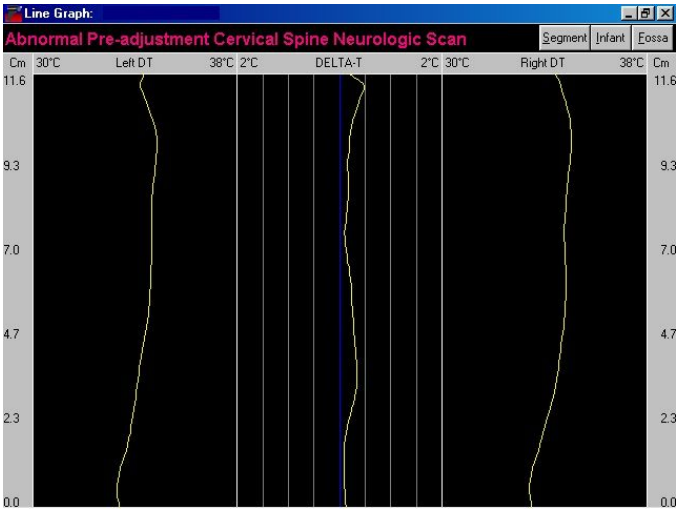

Figure 3

The information gained from the above examinations indicated a high probability of abnormal upper cervical arthrokinematics. Consequently, a precision radiographic series of the upper cervical spine was performed for an accurate analysis of specific segmental biomechanics (13). Neutral lateral, AP, APOM, and BP views were taken using an on-patient laser-optic alignment system in order to precisely align the patient to the central ray.

An analytical radiographic method consisting of mensuration combined with arthrokinematics was performed (13). Noticeable biomechanical abnormalities were found at the atlanto-occipital and atlanto-axial articulations.

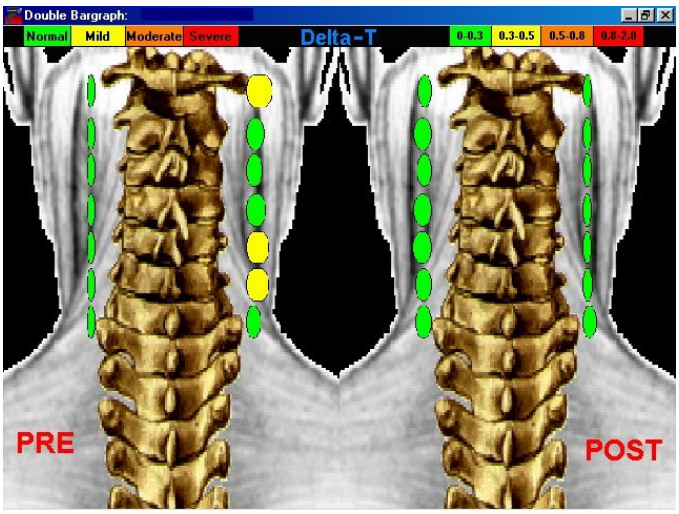

Figure 4

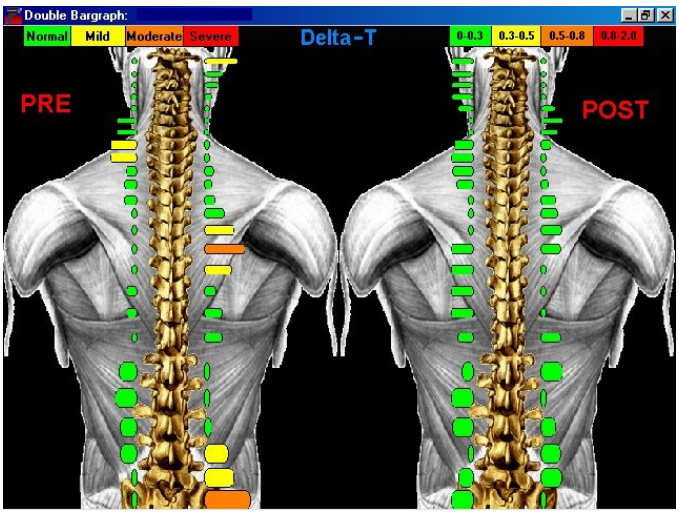

Figure 5

CHIROPRACTIC MANAGEMENT

From the accumulated degree of aberrant biomechanics noted in the upper cervical spine, the atlanto-occipital subluxation was chosen as the first to undergo adjustive correction. Before treatment was rendered, the patient was counseled that they might expect exacerbations in symptomatology as part of the normal response to care due to the global impact of neural reintegration.

To correct the subluxation, the patient was placed on a specially designed knee-chest table with the posterior arch of atlas as the contact point (Figure 6). An adjusting force was introduced using a specialized upper cervical adjusting procedure (14). The patient was then placed in a post-adjustment recuperation suite for 15 minutes as per thermographic protocol (6-8). Correction of the subluxation was determined from the post-adjustment neurologic image noting resolution of the patient's presenting neuropathophysiology (Figure 4). All subsequent office visits included an initial cervical neurologic scan, and if care was rendered another scan was performed to determine if normal neurophysiology was restored. Since the focus of the patient's care was in the upper cervical spine, neurologic scans were made only in this region during normal treatment visits with full spine scans performed at 30 day re-evaluation intervals.

The patient was adjusted once during the first three months of care. After the first adjustment the patient noted an increase in stocking-glove numbness for two days. By the end of the week she reported a significant decrease in symptoms and a mild

increase in muscle strength. She also noticed a mild improvement in her levels of fatigue. The patient mentioned that this change left her very hopeful, but that her condition was so severe and debilitating that she would have to see greater improvement to keep from her IVIG treatment.

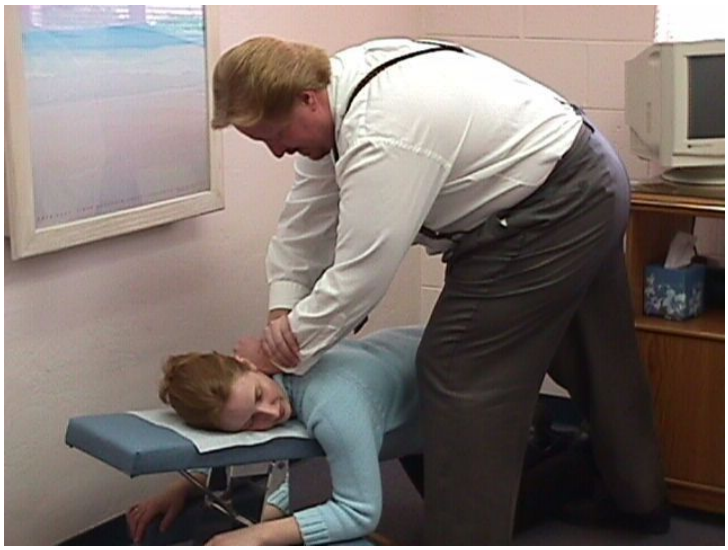

Figure 6

During the second week of care the patient reported continued improvement in both symptoms and strength. She noted having complete days without any stocking-glove numbness. Her upper extremity strength had improved so dramatically that she was able to take on a greater workload. The lower extremity weakness was also improving with a noticeable gait change, but the improvement was much less than in the upper extremities. The patient expressed disbelief in the amount of change in her condition and the speed at which it was occurring.

Between the third and fourth week of care the patient reported an increase in her stocking-glove numbness. Her upper extremity strength, however, had continued to improve. She noted that her lower extremity strength had been waxing and waning. Despite her increased workload, she continued to report an increase in energy. By the fourth week of care the patient had not received an IVIG treatment for four months. In the past six years she had never been able to avoid medical treatment for this long.

A re-evaluation was performed at this time. The examination revealed normal cervical muscle tone and ranges of motion, normal sensation findings in the upper and lower extremities, and a mild limp without shuffling. A full spine neurologic scan was performed noting total resolution of the patient's presenting autonomic neuropathophysiology (Figure 5).

Weeks five through twelve showed continued improvement. There were two mild increases in stocking-glove numbness that lasted for two to three days. The patient noted that she was now having days completely without symptoms or muscle weakness. She also reported that the lower extremity weakness was noticeable mostly with stair use only. A re-examination was performed at eight weeks with no remarkable findings.

Adjustments were made to the upper cervical spine at four and eleven months of care. At this time, the patient had not received any IVIG, prednisone, or other medical treatment for one year.

The patient has continued to improve with mild changes over time. She notes that some lower extremity weakness still shows up with stair use. Other than this, her lifestyle remains active and symptom free.

In consideration of the chronicity and severity of this autoimmune condition, permanent neurologic damage cannot be ruled out with regard to the residual weakness in the lower extremities. Complete resolution of this residual finding, however, may be possible over time considering the drastic improvements seen in this case.

NEUROBIOLOGIC MECHANISMS

Current autoimmune disease research clearly indicates that genetic predisposition alone does not cause the development of autoimmune diseases, but that other factors need to be present in order to initiate the disease process. There are two extensively studied neurophysiologic mechanisms that may explain the genesis of this patient's condition – CNS facilitation and cerebral penumbra. Both of these conditions are thought to arise from an initiating trauma (birth, falls, etc.), which causes entrapment of intra-articular meniscoids resulting in segmental hypomobility and compensatory hypermobility. As a result of this hypermobility, hyperexcitation of intra and periarticular neuroreceptors occurs causing afferent bombardment of the CNS. Over time this can result in facilitation, which is a state of neuronal conditioning where an exponential rise in afferent signals to the cord and/or brain occurs. This may cause a loss of central neural integration due to direct excitation, or a lack of normal inhibition, of pathways or nuclei at the level of the cord, brainstem, and/or higher brain centers. The upper cervical spine is uniquely suited to this condition, as it possesses inherently poor biomechanical stability along with the greatest concentration of spinal mechanoreceptors.

This same mechanism is the genesis of cerebral penumbra. Hyperafferent activation of the central regulating center for sympathetic function in the brain may cause differing levels of cerebral ischemia. A second route via the superior cervical sympathetic ganglia may also cause higher center ischemia. When a certain threshold of cerebral ischemia is reached, a neuronal state of hibernation occurs; the cells remain alive, but cease to perform their designated purpose. Entire functional areas of the cerebral cortex or cerebellum may be affected.

Propagation of these mostly non-adapting signals to the CNS may have systemic autonomic ramifications. Local, and long tract, autonomic manifestations are readily seen with neurologic imaging, as the scans are a direct reflection of sympathetic nervous system function (Figure 5). It is suggested that one of the secondary affects of upper cervical hyperafferency may be global autonomic pathophysiology, specifically

the sympathetic division. Sympathetic dysregulation would be capable of maintaining the autoimmune disease process through direct affects on the immune response.

The role that the sympathetic nervous system plays in the regulation of immune function is paramount to understanding the possible relationship between aberrant upper cervical biomechanics and autoimmune disease. Direct sympathetic innervation of the thymus, spleen, pineal gland, Peyer's patches, lymph nodes, lymphocytes, and bone marrow is well understood in the regulation of immune responses (27-29). It has been discovered that biochemical messengers in the form of cytokines and neurokines, the signal molecules of the immune and nervous system respectively, are expressed and perceived by both systems. Since both systems are capable of acting on terminal immune response tissues, and receiving feedback from the same, is there any difference between the two? The sharp delineation of the two systems has become blurred as research has uncovered their homeostatic interrelationship. Historically viewed as separate, the two systems are now considered as a single integrated mechanism – the neuroimmune system (27-29). Consequently, sympathetically mediated immune dysfunction may be implicated in the maintenance of autoimmune diseases. Correction of pathologic central sympathetic regulation, secondary to aberrant upper cervical biomechanics, may lead to a return of normal immune function. Considering this mechanism, the potential for the improvement of any autoimmune disease is tremendous.

CONCLUSION

Autoimmune diseases remain among the most poorly understood and poorly recognized of any category of illnesses. A myriad of chronic and severe disease states can result from the dysfunction of the body's immune system. As it can be seen, the controlling ability of the nervous system on the immune response cannot be ignored in the pathophysiology of autoimmune diseases.

The most important factor in this case was our ability to objectively monitor the adjustment's affects on the patient's autonomic neurophysiology. Many different types of pre and post-adjustment tests are used in our profession such as leg length, cervical challenge, motion and static palpation, and others. However, these tests lack objectivity, posses inherent errors, and have no literature confirmation of their ability to monitor neurophysiology (30-33). Thermal neurologic imaging, however, has been researched for over 30 years compiling almost 9,000 peer-reviewed and indexed studies confirming its use as an objective measure of neurophysiology. By using this technology, our center has been able to consistently determine the correct adjustive procedures that produce reproducible and dramatic neurophysiologic improvements in our patients.

To what magnitude the upper cervical spine is involved in the genesis of autoimmune disease remains to be seen. In an atmosphere where much of the public see our profession as useful for neck and back pain treatment at most, patients with complex disorders are left unaware of the possible benefits of care. The body of

literature detailing a possible upper cervical etiology, or at least contribution, to organic disorders is substantial. Further research into this area of the spine, combined with objective monitoring of neurophysiology, may reveal that chiropractic does indeed offer consistent conservative management of autoimmune disorders.

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