Research

Chiropractic Manipulation, Functional Neurologic and Nutritional Management for the Reduction for Tourette Syndrome Symptoms in a 27 Year Old Woman.

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ABSTRACT

Objective: to report the treatment of a patient with Tourette syndrome (TS) utilizing nutritional supplements and dietary change along with chiropractic manipulative therapy (CMT), saccadic eye exercises, and audio therapy.

Case presentation: The patient is a 27-year-old female diagnosed with TS at age 23. Motor complaints centered on an eye blink. Vocal tics include throat clearing and sniffing. Obsessive compulsive components were described as touch-based and/or number-based.

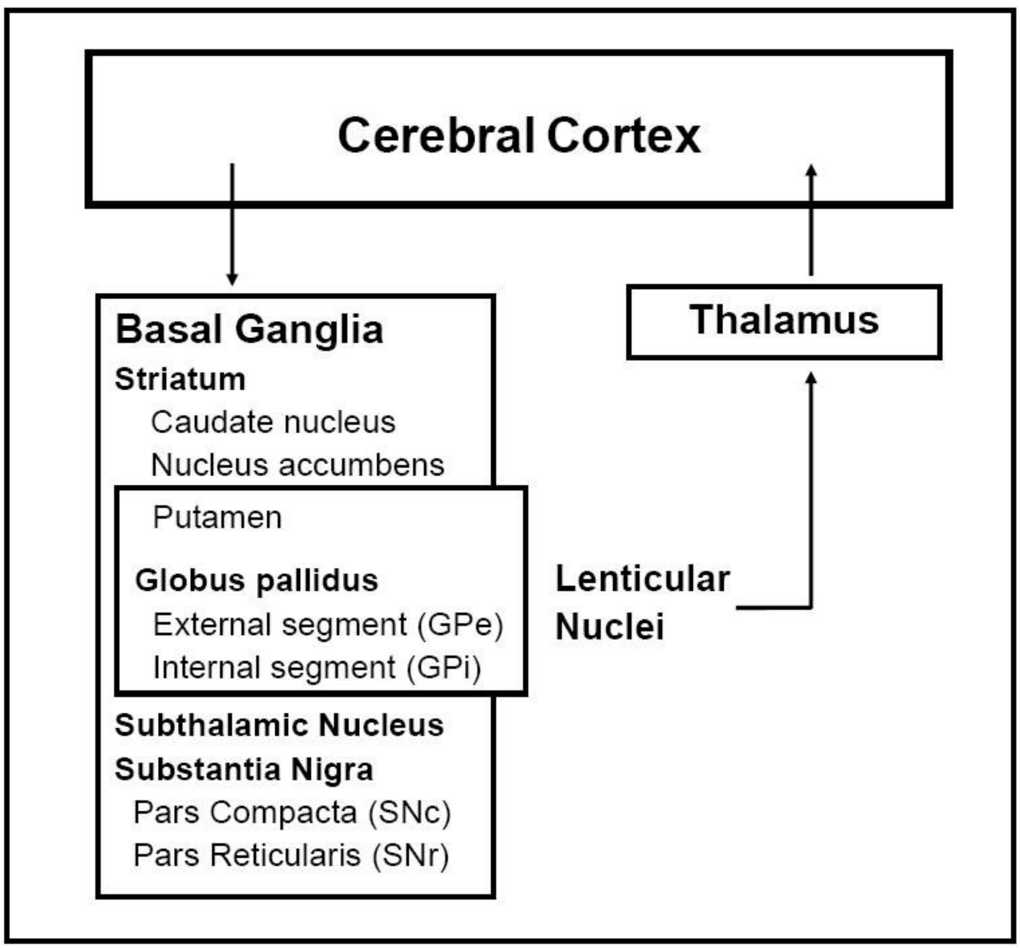
Intervention and Outcome: Primary measures included the Yale Global Tic Severity Scale (YGTSS), Yale-Brown Obsessive Compulsive Scale (Y-BCOS), and the Health Status Questionairre-12 (HSQ-12). Secondary measures included documentation of appendicular coordination, balance, gait, and saccadic eye movements. Treatment consisted of vertebral and extremity adjusting, saccadic eye exercises, audio therapy, and supplementation of magnesium, vitamin B6, and omega 3 fatty acid for 4 weeks. At baseline the YGTSS score was 52, Y-BOCS was 22 and the HSQ-12 was 53. At two weeks, the YGTSS: 22 and the Y-BOCS: 17. At completion the YGTSS: 14, the Y-BOCS: 12, and the HSQ-12: 82.1.

Conclusion: The patient's clinical picture indicated marked improvement in her symptoms. Although natural history cannot be disregarded, the clinical results documented suggest that a multimodal approach of diet, manipulation, and functional neurologic exercises may help in the management of Tourette syndrome symptoms. A controlled study is warranted.

INTRODUCTION

Tourette Syndrome (TS) is characterized by involuntary motor and phonic tics often accompanied by other psychiatric problems such as obsessive-compulsive disorder (OCD), attention deficit/hyperactivity disorder (ADHD), and anxiety and/or depression.[1] Disturbances in the maturation of the frontostriatal systems **(Figure 1)** in individuals with TS likely contribute to impairment in self-regulatory control processes that, in turn, contribute to the difficulty in suppressing tics. Neuroimaging studies have detected abnormal cortical volumes and differences in cross-sectional areas of the striatum and corpus callosum.[2-7] Studies of altered basal ganglia symmetries in subjects with TS suggest that anatomical and functional hemispheric asymmetries may be disrupted in persons with TS. [8;9] Persistence of symptoms into adulthood may reflect a failure of the frontostriatal circuitry to operate properly because of physiological limitations, attributable to either a failure of prefrontal plasticity or to disturbances in striatal functioning in individuals with TS.[10]

Figure 1



Conventional management of TS symptoms seeks to address the functional and neuro-physiological deficit within the frontostriatal circuitry. Pharmacotherapy has been the traditional method of care; however, many discontinue treatment due to unwanted side effects such as weight gain, lethargy, anxiety, and in some cases, dysphoria and dyskinesia.[11] Deep brain stimulation[12] has been advocated as an alternative for cases with severe uncontrolled tics; and transcranial magnetic [13] stimulation is a non-invasive alternative of inducing low frequency magnetic currents into premotor, prefrontal and motor cortices to inhibit activity in these areas. Non-pharmacological behavioral therapies such as habit reversal training teach participants to increase their awareness of premonitory urge and perform competing responses. This is thought to increase input from the prefrontal cortex which uncouples the discriminating stimulus (urge) from the performance of tics.[14]

In recent years, diet, nutrition, and use of complementary and alternative medicine (CAM), including chiropractic manipulative therapy (CMT), have increased for management of tics.[15;16] Dietary and nutritional studies have looked at the effect that foods and the nutrient content in foods may have on the neurochemistry in frontostriatal circuitry and consequently tic severity.[17-19] Neurological consideration for CMT is usually associated with peripheral radiculopathies or neuropathies; however, studies utilizing Somatosensory Evoked Potentials (SEP) have demonstrated the most attenuated SEP peaks to be in the frontal cortices after CMT of the cervical spine.[20;21] Functional neurology is a concept that emphasizes the nervous system as the modulator of human expression and experience. According to the American College of Functional Neurology (ACFN),[22] it is a clinical discipline that addresses the interaction of the nervous system with the environment. Practitioners utilize clinical history and physical examination observations along with current concepts of brain function to formulate neurophysiologic based rehabilitation programs. Leisman and Melillo et al have applied these principles in their studies of ADD/ADHD and autism.[23;24] TS and obsessive-compulsive disorder (OCD) are associated with neurological soft signs (NSS),[25;26] non-localizing deviant performances with motor and sensory tests without structural deficit in the central nervous system, which can be observed with the standard neurological examination. Conversely, functional neurologic practitioners seek to improve these deviant performances with brain-based rehabilitation. This manuscript reports a case of a patient with TS treated with a combined approach utilizing nutritional supplements and dietary change along with CMT, saccadic eye exercises, and audio therapy.

Case Presentation

The protocol for the study was approved by the Institutional Review Board of Parker College of Chiropractic, and written informed consent was obtained from the patient. The patient was a 27-year-old Caucasian female student at an outpatient teaching clinic who volunteered herself for this study. She was diagnosed with TS at age 23 while a military dependant under her father. She was examined by a military general physician who referred her to a military neurologist. Tests included blood work for Lupus (negative), and an MRI scan for any possible tumors that may have been restricting her motor/sensory nerves. Her official diagnosis for TS was concluded in April-May 2005.

Her motor tic complaints center on a noticeable eye blink that can progress to a neck and shoulder shrug and may include the whole upper extremity. Vocal tics include throat clearing, sniffing, and making a short shrill cry described as a squeak. She described her OCD component as touch-based and/or number-based: her socks must fit exactly; she must chew her gum a certain number of times.

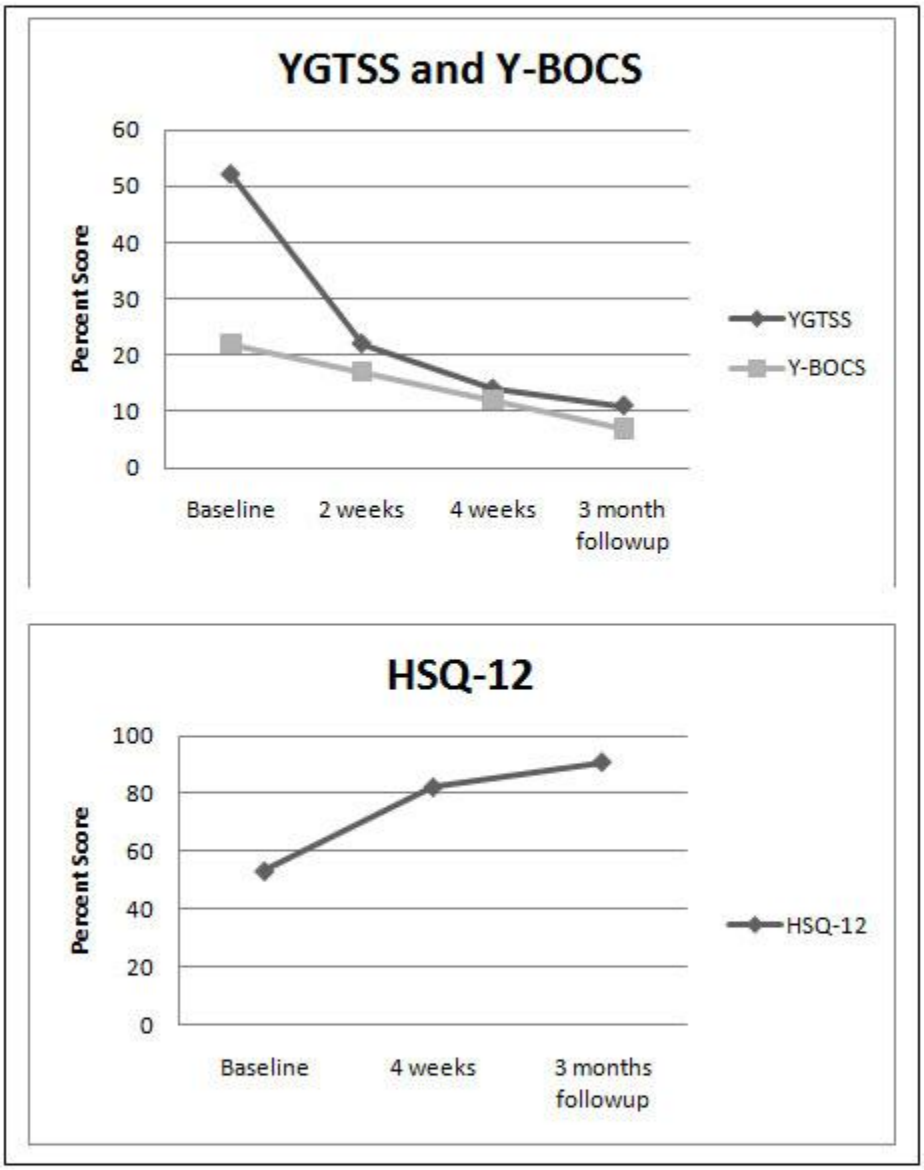
A comprehensive physical was performed. The neurological examination included testing appendicular coordination, balance, gait, and saccadic eye movements.[27] A left sway on Romberg testing with a correlating deviation to the left on Tandem Gait, incoordination of the left hand during diadochokinesia testing, and frontal eye field overcompensation to the left during saccade testing indicated NSS of the left cerebellar hemisphere. Throughout the physical examination, the patient exhibited tics of eye blinking that occasionally would include a neck musculature tic. She reported difficulty sleeping, and awakening several times during the night. She stated that her husband confided to her that she talks during sleep. Postural examination revealed a wide-based stance with her feet separated approximately 1.5 ft. Upon observation, she presented with a pronounced anterior head carriage with left rotation and left lateral flexion.

Intervention and outcomes

The initial study period was four weeks, and the patient continued treatment for an additional three months. Each treatment session consisted of CMT of the axial and appendicular skeleton followed by saccadic eye exercises and audio therapy. Coupled motion adjusting technique was used for manipulation of the cervical spine, thoracic manipulation consisted of prone posterior to anterior thrusting, and lumbar manipulations utilized a side posture pull technique. Appendicular manipulation was rendered to the wrist, elbows, shoulder, and ankles. Wrists manipulations were conducted to include a fast stretch to forearm anterior and/or posterior compartment musculature. For example, when manipulating the scaphoid to include the anterior compartment musculature of the left forearm, a double thumb contact is made to the posterolateral wrist over the scaphoid while the index fingers support the anterior wrist at the base of the hand; the wrist is brought into extension and the whole upper extremity is tractioned inferiorly; the doctor then thrusts the wrist into extension while simultaneously pulling on the base of the hand. Saccadic eye movement exercises were performed with the patient's initial gaze directed to a target in the right frontal eye field and on command, shifting her gaze quickly to a target in the left field starting with one set/6 movements and ending with 2/8. Audio therapy consisted of sounding a 128 Hz. tuning fork to the left ear while the patient occluded the right ear and concentrated on the sound beginning with 1set/10 seconds and ending with 2 sets/10 seconds. A diet free of processed sugars and flours was recommended along with nutritional supplementation of magnesium lactate (210 mg), vitamin B6 (Betacol, 2 mg) and Tuna Omega-3 Oil (300 mg DHA, 60 Mg EPA). All supplements followed manufacturer's recommendations (Standard Process ®, Palmyra, WI) [28]. Primary outcome measures included the Yale Global Tic Severity Scale (YGTSS)[29] to monitor her tic disorder, the Yale-Brown Obsessive Compulsive Scale (Y-BCOS)[30] to monitor her OCD disorder, and Health Status Questionairre-12 (HSQ-12)[31] to measure her overall health status. The YGTSS and the Y-BOCS assign lower scores to greater resistance of tics as an indicator of health, while the HSQ-12 assigns higher scores to indicate better health status. Secondary measures included documentation of NSS of appendicular coordination, balance, gait, and saccadic eye movements.

At baseline her YGTSS score was 52, Y-BOCS was 22, and the HSQ-12 was 53. At two weeks, YGTSS score was 22 and the Y-BOCS score was 17. At completion her YGTSS score was 14, the Y-BOCS was 12, and the HSQ-12 was 82.1. At three months followup her YGTSS score was 11, Y-BOCS was 7, and the HSQ-12 was 90.83. **(Figure 2)**

Figure 2

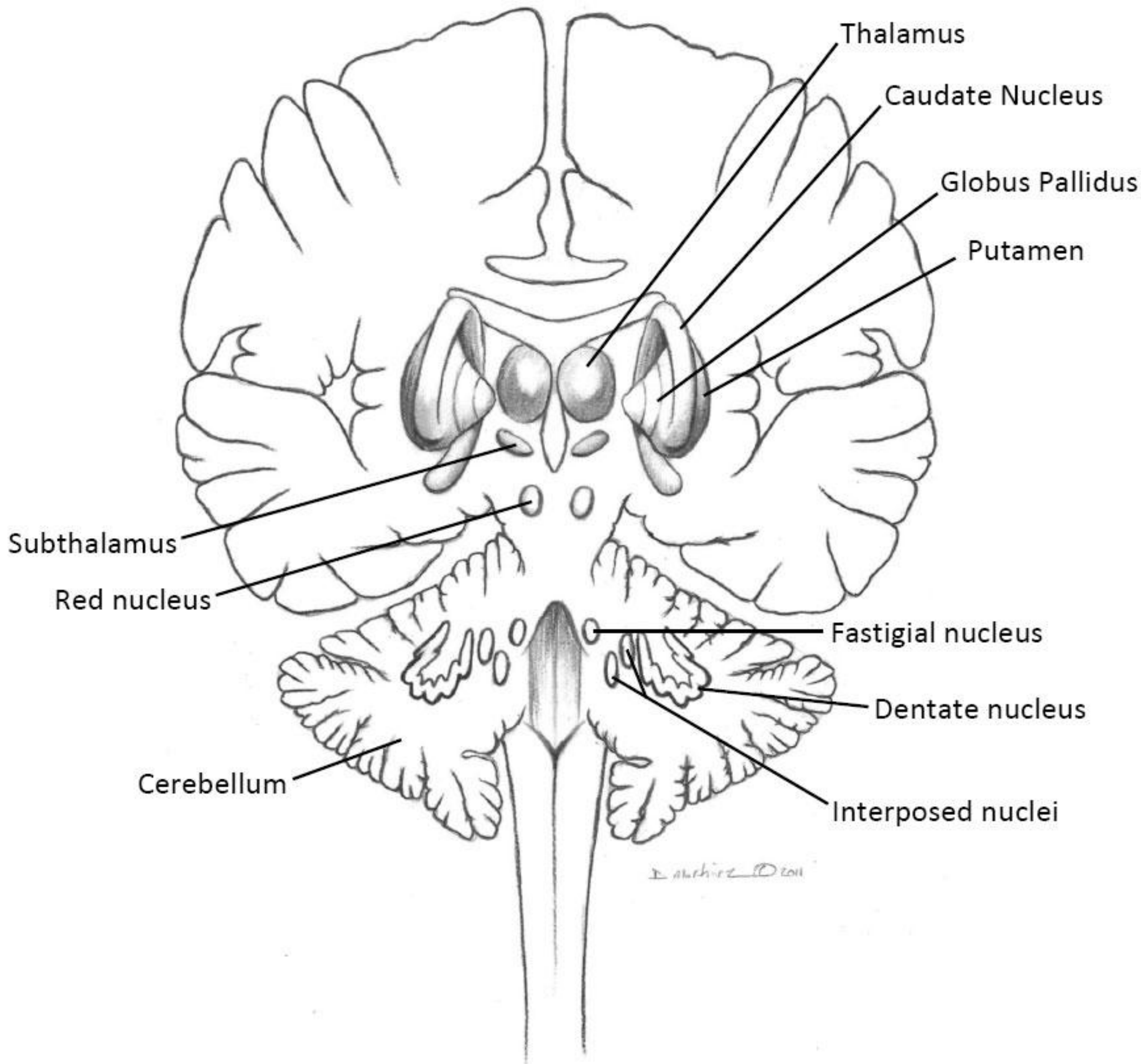


After the initial treatment, the patient stated she felt an immediate reduction in the intensity of her tics and slept uninterrupted for 8 hours that night, and that the reduction continued the following day. After two weeks, she reported a reduction in tic response to stressful situations and compulsions. She reported more restful sleep, and her husband also noted that she had not been talking in her sleep or awakening during the night. Her coordination with diadochokinesis testing appeared symmetrical at pre-treatment evaluations, and she was able to perform tandem gait for approximately 4 steps before a loss of balance. At four weeks, she continued to report restful sleep and a decrease in her compulsions. Her tic was not prominent during her final evaluation, and she stated that she was not aware of them during activities of daily living. Her stance had narrowed to approximately 1.5 inches in width, and she was able to perform tandem gait with no sway and increased speed with eyes closed. Upon observation, her coordination with respect to diadochokinesia appeared symmetrical in all positions and she no longer overcompensated to the left during saccade examination. Additionally, her head position was neutrally positioned with respect to her body.

DISCUSSION

Many adults are aware of their premonitory urges and can be taught to inhibit tics or change the tic through utilizing competing motor responses made in response to the premonitory urge. Increasing the input from the prefrontal cortex may be possible to alter the functional association of premonitory urge and tic expression.[14] The basal ganglia and the cerebellum **(Figure 3)** are the major subcortical nuclei that control multiple aspects of behavior through interactions with each other and with the cerebral cortex.[32] Recent studies have indicated that their output fibers reach cortical areas involved in higher executive function. Their interconnection means that dysfunction in one structure can be propagated to the other.[33] Input to the cerebellum includes most areas of the cerebral cortex, sensory modalities, including the vestibular, visual, auditory, and somatosensory systems, brainstem nuclei, and the spinal cord.[27] The cerebellum is also endowed with a high degree of plasticity. Cellular blockade and ablation studies show that the cerebellar circuitry has a remarkably structural plastic potential not only following damage, but also to maintain its normal architecture under the influence of activity.[34] The high degree of cerebellar baseline activity keeps the firing rates of their neurons in the middle of their dynamic range because these rates can be increased by synaptic excitation and decreased by synaptic inhibition.[35] Since plasticity is synaptic-specific,[36] the premise for the treatment protocol was to provide input to the frontal cortices directly and indirectly through the cerebellum by somatosensory, visual, and auditory stimulation. Axial manipulation was performed to stimulate the midline cerebellar nuclei responsible for balance and gait, and is plausible for the narrowing of the patient's wide-based stance. Appendicular manipulation was performed to stimulate the lateral cerebellar nuclei, which was manifested in improved coordination with diadochokinesia testing. Saccadic eye movements are shifts in the direction of gaze that rapidly and accurately aim the fovea at the target of interest. They supply the brain with a series of still pictures until the desired object is located and are under voluntary control. Adaptation of saccadic amplitude or direction occurs gradually and is thought to represent a true motor plasticity.[37] Although no objective measurements for saccadic overcompensation were performed, subjective improvement was observed. Auditory attention can be directed selectively to a variety of features including spatial location, auditory pitch, frequency or intensity.[38] The 128 Hz tuning fork was chosen out of convenience; however, the focus of the therapy was to place attention on the pitch in order to create a competing stimulus to rival premonitory urge.

Figure 3



Diets free of low-nutrient, high-energy dense foods have been advocated as primary prevention of cardiovascular disease and mental illness.[18] Deficiencies in magnesium and vitamin B6 have been specifically implicated in Tourette's patients. Magnesium deficiency produces neuronal hyperexcitability; there may be heightened anxiety and orofacial dyskinesia, an increased release of dopamine, and greater modulation of serotonin receptors. Magnesium deficiency also reduces the activity of vitamin B6, which has been related to spasmodic movements and hyperirritability.[39] Docosahexaenoic Acid (DHA), along with Arachidonic Acid (AA) can be broken down to play an important role in neural function including sleep induction, long term potentiation, spatial learning and synaptic plasticity, resolution of inflammation and anti-inflammatory and neuroprotective activity. [40]

Natural history cannot be disregarded in this case. Spontaneous remission of symptoms is common among TS patients, such that there are bouts followed by periods of tic-free behavior.[1] It may be the case that treatment was begun at the end of a waxing period whereby the patient's condition would have improved without the intervention. Many TS patients are able to suppress tics temporarily, but often at the expense of concentration and exhaustion.[41] A final consideration is the Hawthorn or placebo effect.[42] The positive expectancy of treatment effect could be considered high for this patient. Yet, this may seem to explain only the short term results as much of the attention had diminished after the initial study period.

CONCLUSION

The patient's clinical picture indicated marked improvement in her symptoms. The clinical results documented in this report suggest that a multimodal approach of diet, manipulation, and functional neurologic exercises may help in the management of Tourette syndrome symptoms, although natural history cannot be disregarded. Limitations include a single subject and outcome measures that are predicated on the skill of the clinician and the accuracy in detail of the patient. A controlled study that includes a naïve population with an appropriate sample size, a controlled environment, and technology that can document objective changes in neurological function is warranted.

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