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PRIMARYEVIDENCE

Postural orthostatic tachycardia syndrome (POTS) in a patient as a consequence of a concussion along with occipital headaches, dizziness and nausea: A case report

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Objective: To investigate the synergetic benefits of combined noninvasive chiropractic protocols for treatment of post-traumatic POTS.

Clinical Features: A 36-year-old female sought chiropractic care for POTS induced nausea/dizziness/palpitations, constant bilateral occipital headaches, neck and sacroiliac/sciatic pain, exercise intolerance and bloating. Patient history, physical examination, and active 10-minute stand test established that she met the criteria for POTS. Prior to her presentation at this office her symptoms had persisted for a year and were unresponsive to other interventions (medical care/over-the-counter-analgesics).

Intervention and Outcome: Craniocervical and spinal misalignments, believed possibly leading to cerebral vasculature and connective tissue deformation, were corrected according to the Sacro-Occipital Technique and Atlas Orthogonal upper cervical evaluation protocols. Specific POTS related therapeutic exercises, diet modifications, as well as ocular fixation exercises were also implemented. Orthostatic vital sign testing was regularly recorded to assess POTS patient's response to care. Following combined treatment and adjunct therapy, the patient no longer met the criteria for POTS, did not exhibit pain or orthostatic intolerance symptoms to postural change except for mild brief bouts of dizziness.

Conclusion: Further research is needed to determine what subsets of patients with POTS might also respond to this conservative chiropractic care approach.

Indexing terms: Chiropractic; Postural Orthostatic Tachycardia Syndrome (POTS); Atlas Orthogonal technique; sacro-occipital technique.

Introduction

Postural orthostatic tachycardia syndrome (POTS) is characterized by orthostatic intolerance and excessive increases in heart rate in the absence of hypotension during orthostatic stress. (1) It has been described as a heterogeneous illness, for it is a syndrome of several distinct synergetic pathomechanisms and presentations rather than one disorder. (2) Although the aetiology of POTS is incompletely understood, its main characteristic is tachycardia generated by increased sympathetic outflow without appropriate vascular response to posture change. (2, 3)

Recent literature defines POTS as being characterized by a sustained heart

'... a conservative approach as described in this report is able to achieve clinical benefits with noted patient improvement. The lesson is to have a range of clinical tools that can be tailored to the patient's presenting symptomatology ...'

rate (HR) increase of > 30 bpm within 10 minutes of a stand test without any orthostatic hypotension (>20 mmHg decrease in systolic blood pressure), along with orthostatic symptoms present for a minimum of 6 months, in the absence of medication or medical conditions inducing tachycardia. (1, 4, 5) Orthostatic symptoms include those resulting from posture induced tachycardia, and brain hypoperfusion such as palpitations, dizziness or headache, as well as those arising from diffuse autonomic nervous system dysregulation including nausea, bloating, bladder and bowel disorders. (5) Fatigue, pain and cognitive disturbances are common comorbidities. (4, 6). POTS constitutes one of the most common condition of the autonomic nervous system (ANS) and it is estimated that over 75% of the affected patients are female. (5, 6)

Concussion can be defined as a traumatic brain injury that causes a pathophysiologic process affecting brain function. (7) Head and neck trauma induces hyperextension, hyperflexion and translation at the craniocervical junction (CCJ). (8) The CCJ is composed of the base of the skull (occiput), caudal brainstem, first two cervical vertebrae (C1-2), upper cervical spinal cord, blood vessels to and from the brain, cerebrospinal fluid (CSF) system along with the muscles and connective tissue (including the dura mater) that serve to stabilize it. (8, 9) Such forced head and neck movements can cause instability/misalignments at the CCJ, potentially compromising brainstem, CSF system and other cranial vault tissue function, considering the ensuing deformations of structures such as the dura mater and blood vessels. (5, 8)

Several studies suggest that cardiac autonomic dysfunction can arise as a secondary consequence of concussion. (7, 10, 11) The ANS maintains body cardiovascular homeostasis, consists of central and peripheral autonomic pathways and is comprised of both the sympathetic (SNS) and parasympathetic nervous system (PNS). It originates in the hypothalamus, descends in the brainstem caudal pons and rostral medulla (ANS central network location) to reach the target organs in the periphery via the SNS and PNS. (5, 10) The SNS is thought to increase HR whereas the PNS results in decreased HR, and their combined action helps both regulate BP through different mechanisms and influence the function of many organs, including the heart. (11)

The selected minimal diagnostic approach utilizing patient history, thorough physical examination and orthostatic vital sign testing was deemed sufficient to screen for brainstem decreased function possibly leading to ANS dysregulation as a suggested mechanism of post-concussion POTS in this patient. (6, 12, 13)

Hence, since the tachycardia that characterizes POTS is thought to be the body's attempt to compensate for cerebral hypoperfusion, a suggested underlying cause for reduced venous return is impaired vascular sympathetic tone upon standing as a consequence of ANS dysregulation. (4, 6, 10, 12)

The chronic, nonspecific, heterogeneous, systemic nature of POTS symptoms and pathomechanisms, along with the absence of orthostatic hypotension, has probably led to this syndrome being often overlooked by health care providers. (1) This, along with the difficulty in obtaining medical help due to the limited current treatment options, outcome and understanding of this syndrome, can cause frustrations and reduced quality of life. (6)

This case report investigates the synergetic benefits of combined noninvasive chiropractic protocols for treatment of post-traumatic POTS.

Case Report

A 36-year-old woman sought care at this office on March 6th 2017. She reported falling on ice in March of 2016. The traumatic impact was both to the craniocervical junction and the left sacroiliac joint. She described experiencing immediate confusion, dizziness, nausea, bilateral occipital headaches, palpitations and transient amnesia. When assessed the next day at the emergency room, she was diagnosed with having suffered a moderate to severe concussion. (10)

After a few weeks, and up to the present, she reported experiencing constant bilateral occipital headaches, along with dizziness, nausea and palpitations upon fast head movements or any postural change to or from the upright position. Her condition was exacerbated by alcohol and fatigue. Concurrent symptoms included exercise intolerance, bloating, fatigue, constant neck pain as well as left sacroiliac and left sciatic pain extending down to the foot. At the time, the patient was a stay-at-home mom with pre-school children. She was able to maintain activities of daily living, but with great difficulty considering the pain, dizziness and intolerance to physical activity. The realization that her symptoms were not improving after one year prompted her to seek treatment.

Past history

This athletic patient's past medical history was unremarkable except for an episode of neck pain resulting from a mild head trauma in 2007 which was resolved after seeking low force upper cervical chiropractic care over the course of a few months. She had three pregnancies carried to term. The patient did not have a past or current history of iron deficiency, endocrine disorder (such as diabetes, thyroid, adrenal), sleep disturbance, hyperventilation, cardiac rhythm abnormalities, volume depletion, drug abuse, medication, neurological illness (such as syncope), depression, panic attack or general anxiety.

She had been under the care of her medical practitioner for about 4 months following her March 2016 accident. He had recommended over-the-counter analgesics for her pain which only provided temporary relief, but offered no treatment for her symptoms of exercise intolerance, bloating, fatigue, palpitations, dizziness and nausea upon postural change from or to the upright position. At the time of her initial visit, she was not taking any medication.

Physical Examination

The physical examination findings were unremarkable for upper and lower extremity sensory, motor and reflex exams. The cranial nerve exam was also unremarkable except for cranial nerve 3, 4 and 6 testing where hypermetria (13,14) was observed during left horizontal and downward left diagonal saccadic eye movements (rapid movements that move the eyes from one object to the next) as well as reduced smooth pursuit (predictive tracking to stabilize moving objects) velocity in tracking horizontally to the right. (Table 5) The finger tapping diadochokinetic diagnostic test (rapid tapping of tip of index finger against the tip of the thumb) showed slowed finger movement on the left. (14) (Table 5)

Blood pressure (BP) was initially taken bilaterally and exhibited 100/60 mmHg on the right arm compared to 93/55 on the left. BP was consistently recorded on the right side for all the consecutive measurements. Upon performing the initial active 10-min stand test (following a 10 min supine baseline period, HR and BP were recorded before and after the patient stood for 30 sec, 2, 5 and 10 min) the patient reported strong dizziness, nausea and palpitations lasting up to 4 min .(1, 5, 15) (Table 5) However, the duration of the orthostatic symptoms were reduced, lasting only 2 minutes, when the patient went from supine to standing with ocular fixation on a target. (Table 5) While performing the active 10-min stand test, the patient experienced a maximum HR increase of 51 bpm within 10 minutes of standing without any orthostatic hypotension (<20 mmHg decrease in systolic pressure), along with orthostatic symptoms of dizziness, nausea and palpitations. (Tables 1 and 5) BP did not significantly fluctuate when tested upon postural change from the supine position to standing. (Table 1) Thus, through a complete patient history and comprehensive physical examination, as well as orthostatic sign testing including blood pressure (BP) and heart rate (HR) recording, it was established that this patient met all the criteria for POTS. (1, 4, 15) (Table 1)

Table 1. Orthostatic Testing and Active 10-min Stand Test at Initial Exam					
	After 10 min Supine	After 30 sec	After 2 min	After 5 min	After 10 min
Pre-testing HR supine bpm	63	-	-	-	-
HR supine to standing bpm	-	72	93	114	111
Pre-testing BP supine mmHg	100/60	-	-	-	-
BP supine to standing mmHg	-	105/60	105/60	102/62	100/65

Table 2. Stage 1: Orthostatic testing and Active 10-min Stand Test after 8 Weeks of SOT and AO Chiropractic Care					
	After 10 min Supine	After 30 sec	After 2 min	After 5 min	After 10 min
Pre-testing HR supine bpm	60	-	-	-	-
HR supine to standing bpm	-	75	93	90	90
Pre-testing BP supine mmHg	110/65	-	-	-	-
BP supine to standing mmHg	-	100/63	108/62	105/60	105/65

Chiropractic examination

Cervical rotation and lateral flexion range of motion (ROM) was initially reduced by 50% bilaterally and painful. Bilateral head rotation elicited dizziness and nausea of 2 minutes duration (Table 5). Lumbar vertebral flexion was limited with pain at the L5/S1 and left sacroiliac levels.

In order to assess if the CCJ bone structures (C1-2 vertebrae, occiput) were aligned in their best anatomical position, the patient was examined using the Atlas Orthogonal (AO) protocol which consists of a scanning palpation of the C1-2 nerve roots bilaterally, a supine leg-length differential as well as a weight-bearing postural cervical x-ray analysis.

CCJ palpation elicited moderate to severe pain at the C1 nerve root level bilaterally, indicating a positive result. Supine leg-length differential testing was also positive. The weight-bearing postural cervical x-rays that were taken according to the AO protocol revealed a C1-2 misalignment with respect to the occipital bone. (8, 9)

SOT protocol indicator tests were positive for:

1. category III including unilateral straight leg raiser test (on side of involvement), the 'step out toe out' (SOTO) test, unilateral psoas muscle contraction, sciatic nerve pain and prone leg-length differential;
2. cranial bone movement restrictions at the temporomandibular joint (TMJ) and zygomatic bone using the cranial bone range of motion (ROM) analysis; and

3. occipital fibers (3 and 7) and viscerosomatic/somatovisceral reflex distortions at the level of the 1st lumbar (L1) and 9th thoracic (T9) vertebrae (16,17) using the chiropractic manipulative reflex technique (a soft tissue and reflex balancing method utilizing occipital fibers, visceral reflex points and vertebral levels as indicators).

The *Dix-Hallpike* manoeuvre was negative for benign paroxysmal positional vertigo (BPPV) for the patient did not exhibit any nystagmus. However, it triggered strong dizziness and palpitations that lasted for over 30 minutes after completion of the test. (18)

Table 5. Physical Exams summary

	At Inital Exam (before 1st stage)	Re-Exam after 2nd Stage (3 months into treatment)	Final Re-Exam after 3rd Stage (4 months into treatment)
Diagonal saccadic eye movements up Rt/down Lt	Hypermetria down Lt	Hypermetria down Lt	Mild hypermetria down Lt
Horizontal saccadic eye movements to the Lt	Hypermetria	Unremarkable	Unremarkable
Horizontal smooth pursuits to the Rt	Reduced velocity	Unremarkable	Unremarkable
Finger Tapping test (diadochokinesia)	Lt side markedly slower	Lt side slower	Unremarkable
Bilateral head rotation	Strong dizziness/ nausea (2 min)	50% Improvement Dizziness/nausea (1 min)	Unremarkable
Stand Test from supine to standing	Strong dizziness/ nausea/palpitations (4min)	75% Improvement dizziness/nausea without palpitations (1min)	Mild dizziness, without nausea or palpitations (a few seconds)
Supine to standing with ocular fixation	Moderate dizziness/ nausea/palpitations (2min)	No dizziness/nausea/ palpitations	No dizziness/nausea/ palpitations

Intervention

The treatment was aimed at improving the patient's autonomic nervous system regulation and addressing co-occurring symptoms by correcting associated craniocervical junction, spinal and pelvic misalignments as well as implementing adjunctive therapies.

This POTS patient's presentation was multifactorial and two different chiropractic protocols were used in her evaluation and treatment in order to best improve or resolve the orthostatic intolerance and concurrent symptoms. In an attempt to better evaluate the patient's response to the different chiropractic protocols, orthostatic/ocular fixation exercises and lifestyle/diet modifications, the treatment was divided into three stages (Tables 2, 3, 4) and the patient reevaluated with an active 10-min stand test after every stage. The remarkable neurological findings obtained from the initial physical exam were also reevaluated before and at the end of the 3rd stage of treatment in order to reassess brainstem function. (Table 5)

The 3 stages consisted of:

a. interventions with AO and SOT chiropractic care for a period of two months;

b. a gluten-free diet, B vitamin supplementation, increased water intake and low intensity cardiovascular exercises were supplemented to the same chiropractic care for another month; and finally

c. a neurological examination retesting and daily postural challenge exercises with ocular fixation for one month were added to the aforementioned treatment and measures, followed by a final neurological examination testing.

The cervical spine misalignment at the CCJ was corrected using the low force AO percussion instrument according to the x-ray findings. Post-treatment cervical x-rays were then taken to confirm that the proper spinal alignment was restored. (8, 9) The frequency of the AO treatments was gradually reduced until CCJ stability was achieved. (8, 9)

Based on the sciatic pain symptoms and positive SOTO (step out toe out) test, SOT Category III procedures were performed. These included prone Category III blocking, unilateral SOTO manoeuvre, unilateral psoas muscle release and prone acetabular adjusting. (16, 19, 20, 21)

Chiropractic manipulative reflex technique (CMRT) was alternatively provided for the T9-associated adrenal gland and the L1-associated ileocecal valve. (22, 23, 24, 25) Restoring cranial sutural motion was achieved by releasing the unilateral movement restriction of the zygomatic bone and TMJ using SOT intra-oral (Malar Lift) and external *pterygoid* muscle release interventions, respectively. (23, 26, 27) The SOT care was maintained, with a gradual decrease in frequency, until the category III, cranial bones and CMRT indicators were no longer present. (26)

After 2 months of AO and SOT treatment, a gluten-free diet, daily B complex vitamins, increased water intake to 2-3 litres per day and decreasing caffeine to 1 cup of coffee per day, and low intensity cardiovascular exercises (2-2½ hour slight incline walking bi-weekly) were supplemented to the ongoing care for another 4 weeks. (2, 6, 12, 15, 28)

At this point, the patient no longer met all the criteria for POTS. However, since some dizziness and nausea were still present upon performing the active 10-min stand test and bilateral head rotation, daily home based postural challenge exercises with ocular fixation were added to the aforementioned treatment. These exercises were performed for the final month and included:

d. bilateral vestibulo-ocular reflex (VOR) cancellation (29) done seated on a rotating stool, spinning 90 degrees to one side, fixating on superposed thumbs, arms outstretched (10 repetitions left then right, 3x per day);

e. supine to standing with ocular fixation (3 repetitions, 3x per day).

The patient was instructed to lower the number of repetitions if symptoms of dizziness, nausea or palpitations where experienced while performing an exercise.

Results

After the 1st 8 weeks of combined AO and SOT care, the patient no longer exhibited bilateral occipital headaches, neck pain, left sacroiliac/sciatic pain or restricted cervical/lumbar ROM whereas the orthostatic symptoms of dizziness, nausea and palpitations upon postural change from supine to the upright position were improved 50% (2 minutes duration). She reported feeling less fatigue and improved tolerance to low intensity cardiovascular exercises while still experiencing bloating. The active 10-min stand test performed at that time revealed a maximum HR increase of 33 bpm compared to the initial 51 bpm within 10 minutes of standing without orthostatic hypotension. (Table 2)

The 'stand test', repeated 4 weeks after having added dietary restrictions and low intensity cardiovascular exercise to the treatment, showed further improvement with a maximum HR increase of 18 bpm within 10 minutes of standing, confirming that the patient therefore no longer met the criteria for POTS (Table 3). Orthostatic symptoms of dizziness and nausea (without

palpitations) were reduced 75% when compared to the initial stand test (1 minute duration), and the patient reported performing low intensity exercise without difficulty, while experiencing minimal fatigue and no more bloating. (Table 5) When re-evaluated, the initial neurological significant findings indicated normal left horizontal saccadic eye movement, no dizziness, nausea or palpitations while going from supine to standing with ocular fixation whereas dizziness/ nausea upon bilateral head rotation persisted. (Table 5) The downward left diagonal saccadic eye movements remained hypermetric while the horizontal saccadic eye movements to the left, and horizontal smooth pursuits to the right, were normal. (Table 5) The finger tapping diadochokinetic diagnostic test showed increased finger movement on the left however still slower than the right side. (Table 5)

After 4 weeks of implementing daily postural challenge exercises including ocular fixation, the last stand test remained negative for POTS and the patient reported experiencing mild dizziness (only for test duration) without nausea or palpitations while performing the test. (Tables 4, 5) The final neurological testing was unremarkable except for mild hypermetria upon diagonal downward left saccadic eye movement. (Table 5) The patient's blood pressure was the same on both sides. The adjunct therapy as well as both chiropractic protocols utilized in treating this post-traumatic POTS patient are considered conservative, noninvasive procedures. (9, 22)

The follow up *Dix-Hallpike* manoeuvre at this visit was negative, ruling out benign paroxysmal positional vertigo (BPPV) as a possible differential diagnosis considering the reported dizziness and nausea. (18) Of significance there was no triggering of dizziness and palpitations after completion of the test. The differential diagnosis of orthostatic hypotension was also ruled out since no significant blood pressure decrease was observed initially or at any time (>20 mmHg decrease in systolic blood pressure) within 10 minutes of the stand test. (1, 4, 5)

Table 3. Stage 2: Orthostatic Testing and Active 10-min Stand Test after 4 more Weeks of SOT/AO Chiropractic Care with Diet Change, B Vitamins, Increased Water intake and Cardio Exercises

	After 10 min Supine	After 30 sec	After 2 min	After 5 min	After 10 min
Pre-testing HR supine bpm	60	-	-	-	-
HR supine to standing bpm	-	63	78	78	66
Pre-testing BP supine mmHg	105/60	-	-	-	-
BP supine to standing mmHg	-	100/62	105/60	100/65	100/62

Table 4. Stage 3: Orthostatic Testing and Active 10-min Stand Test after 4 more Weeks of SOT/AO Chiropractic Care, Diet Change, Vitamins, Increased Water Intake, Cardio Exercises with Postural Challenge Ocular Exercises

	After 10 min Supine	After 30 sec Standing	After 2 min Standing	After 5 min Standing	After 10 min Standing
Pre-testing HR supine bpm	63	-	-	-	-
HR supine to standing bpm	-	72	75	72	66
Pre-testing BP supine mmHg	100/58	-	-	-	-
BP supine to standing mmHg	-	98/58	98/62	98/60	105/65

Discussion

Over the past two decades, there seems to have been a growing clinical interest for POTS. (15) Various underlying pathomechanisms have been described in literature for POTS, including brainstem dysregulation, hyper-adrenergic stimulation, distal neuropathy, impaired venous return, hypo-volemia, autoimmunity, neuro-inflammation, de-conditioning and psychological mechanisms, which are not mutually exclusive. (2, 3, 5, 6, 30) In order to keep a pragmatic focus, the description of these underlying pathomechanisms was omitted. The selected minimal diagnostic approach utilizing patient history and extensive physical examination suggested brainstem decreased function as a probable primary mechanism for ANS dysregulation in this post-concussion POTS patient. (5, 13) It is noteworthy that the patient did not have a past or current history of iron deficiency, endocrine disorder, sleep disturbance, hyperventilation, cardiac rhythm abnormalities, volume depletion, drug abuse, medication, neurological illness, depression, panic attack or general anxiety, all of which can affect the ANS leading to orthostatic intolerance. (2, 6) Nevertheless, if the patient's symptoms had not markedly improved or resolved, the necessity for more comprehensive ANS testing would have been considered. (6)

The treatment was aimed at improving both the patient's autonomic nervous system regulation and addressing co-occurring symptoms by restoring proper CSF flow, venous and arterial cerebral blood flow as well as the integrity and functional architecture of ligaments/dura mater collagen networks.

Since the concussion-induced onset, one year prior, the patient had been suffering from constant occipital headaches. In view of the persistent presentation of these headaches, orthostatic intolerance was ruled out as a possible primary underlying mechanism. This hypothesis was further supported by the fact that the headaches reduced completely following a 2 month treatment period with AO and SOT, while, although significantly improved, some orthostatic intolerance symptoms persisted and the active 10-min stand test was still positive for POTS.

It has been reported that the prevalence of headache and neck pain after a concussion is 85.1% and 37.1% respectively, and prominent data suggests that altered cardiac autonomic function is found in patients with headaches, migraines or neck pain. (11) The present study suggests that these correlations can result from a trauma induced CCJ misalignment/instability. The mechanical strain suffered during head trauma can cause a rotary misalignment of the first two cervical vertebrae (C1-2) with respect to the occiput, possibly disrupting normal CSF flow and contributing to vertebrobasilar system blood flow compromise as a consequence of impingement of the vertebral artery as it passes through the inter-transverse foramina of C1-2. (5, 9, 8, 31) The vertebrobasilar system is essential in providing blood supply to posterior brain fossa structures including the cerebellum and brainstem. (8, 9, 31) The brainstem shares intimate neurological connections with the cerebellum and is directly implicated with vestibular and ANS regulation. (20, 25, 39) Equilibrating arterial and venous cerebral blood flow in addition to CSF flow can therefore directly impact brainstem function thus vestibular and autonomic nervous system regulation. (5, 8, 10)

Additionally, such a trauma induced craniocervical rotary misalignment creates a mechanical strain leading to the deformation of both the pain-sensitive dura mater, which is firmly attached to the foramen magnum, C1-2 vertebrae and pelvis, as well as the C1-2 innervated facet capsular ligaments that can, in turn, irritate the adjacent C2 nerve root. (8, 32, 33, 34, 35) These impaired dural tensions can possibly interfere with CSF flow, in turn potentially reducing brain perfusion and venous outflow. (8, 9, 16, 22, 36) Chronic ischemia to cerebral tissue (including the brainstem) and distortion of collagen network such as ligaments or dura mater has been associated with nociception in the head and neck, in addition to brainstem ANS reduced function.

(8, 32) Consequently, it is inferred that the craniocervical junction misalignment was a major contributing factor to the patient's constant headaches and neck pain. Moreover, it may have accounted for a significant portion of the orthostatic symptoms of dizziness, nausea and palpitation upon orthostatic challenge.

Furthermore, when present, these described trauma-induced cephalad dural tensions can be transmitted caudally to the spine and pelvis, given that the dura mater is continuous and has various attachments to the vertebral column connective tissue as well as the sacrum/coccyx. (33, 34, 37) Thus, it is suggested that releasing the tension on these pelvic and spinal dura mater attachments, using the SOT protocol, likely resolved the sacroiliac/sciatic pain symptoms, in addition to reducing some of the dural distortions involved in exacerbating the craniocervical junction misalignment/instability. (36, 38) For the same reason, correcting a CCJ misalignment could potentially improve or resolve more caudal symptoms. (39) Moreover, the CCJ also plays a central role in facilitating the cervico-collic and vestibulo-collic reflexes that seek to keep the eyes levelled (head vertical), over the center of gravity between the feet. (40) Hence, incorrect alignment of that region can result in further compensatory spinal or pelvic misalignments.

Following 8 weeks of SOT and AO treatment, the maximum HR increase recorded during the active 10-min stand test was substantially improved, exhibiting a 33 bpm maximum increase compared to the initial 51 bpm within 10 minutes of standing. These results suggest that the combination of these two treatment protocols improved the patient's condition in such a way that she only just met the criteria for POTS.

When performed one month later, after supplementing the described, combined chiropractic care with exercise, gluten-free diet, increased water and vitamin B complex intake, the active 10-min stand test was negative for POTS. It is difficult to infer whether this improvement is attributable to the life style/diet change, a longer period under chiropractic care or the combination of both.

It is noteworthy that low intensity exercise tolerance was only acquired after 8 weeks of the combined AO and SOT treatment. Exercise intolerance inevitably leads to de-conditioning, which decreases baroreceptor vasoconstriction reflex gain, compounding any pathological processes contributing to POTS. (2) Low intensity cardiovascular exercise, supported with increased water intake, is considered by most sources to represent the cornerstone of current mainstream management for POTS, regardless of the underlying patho-mechanism. (2, 5, 12) Water intake is a temporary compensatory mechanism that increases standing blood pressure, whereas mild to moderate exercise training is reported to increase orthostatic tolerance and blood volume over time. (2, 41) Since excessive caffeine absorption not only supports diuresis and hypo-volemia but also stimulates the SNS, the patient was instructed to keep coffee consumption down to a maximum of one cup per day. (2, 12).

POTS patients often present with GI complaints, and a possible correlation between gluten related disorders and POTS has been suggested in a recent study. (15) In support of this, another study revealed a higher prevalence of self-reported gluten sensitivity in POTS patients compared with the control group. (15) Interestingly, a proportion of these non-celiac gluten-sensitive patients were reported to exhibit serum antinuclear antibody positivity and antigliadin antibodies. (15) These serum findings could suggest inflammation (leading to malabsorption), resulting in impairment of intestinal mucosal barrier integrity, inducing an immune response. (15, 42)

Hadjivassiliou et al have demonstrated that non-celiac gluten-sensitive and celiac disease patients can both equally present with neurological dysfunction and be as responsive to a gluten-free diet. (43) Even though the underlying mechanisms by which neurological dysfunction may result from gluten sensitivity are still incompletely understood, leaky gut has been shown to

disrupt the blood brain barrier (BBB) via gastrointestinal-derived hormone secretion, small molecule/cofactor production or inflammatory mechanisms such as cytokine stress. (42) As BBB integrity is diminished, so is its capacity to selectively restrict the passage of elements including molecules, toxins, chemicals or pathogens from the blood to brain tissue. (40, 42) Furthermore, one hypothesis suggested that intestinal malabsorption can result in vitamin B deficiencies (thiamine, folic acid and cobalamin), vitamins that yield neuro-protective properties. (15) Hence, a gluten-free diet was implemented based on the persisting constant GI tract complaint on the premise that it might reduce bowel inflammation, a condition suggested to lead to CNS impairment as well as GI discomfort.

In addition, because of the potential inflammation-derived intestinal nutrient malabsorption, the patient was supplemented with daily vitamin B complex for its neuro-protective effects. Within the bounds of this study, it is not possible to objectively assess if the gluten-free diet or vitamin B supplementation had beneficial effects on the CNS. However, the active 10-min stand test being negative for POTS after one month of implementing adjunct therapy, including a B vitamin/gluten-free diet, raises questions. Nonetheless, there seems to have been a temporal correlation between the initiation of a novel diet and the cessation of the patient's bowel complaints.

Different eye movements result from the interaction of numerous systems, notably the eye muscles, brainstem, cerebellum, vestibular system and cerebral cortex. Oculomotor assessment has been utilized in the identification and diagnosis of mild traumatic brain injury by a growing body of research (44, 45, 46). It includes the measurement of saccadic eye movements, smooth pursuits, fixation and reaction time. Each of these various eye movements originate from different interacting brain areas, and a prior study on concussion has suggested that the severity of oculomotor dysregulation correlates with the severity of the cerebral neurological dysfunction. (45, 46) Regulation of saccadic eye movements is under the control of different brain areas including brainstem (burst neurons) and cerebral cortex. (44) The smooth pursuits system, although only partly understood, has been associated with the brainstem, cerebellum and cerebral cortex. (44) Initially, the patient's saccadic eye movements were found to be hypermetric and the smooth pursuits exhibited reduced velocity, whereas at the end of the 3rd stage of the treatment the oculomotor assessment was unremarkable except for mild hypermetria upon diagonal left saccadic eye movement. These post-treatment results suggest improved cerebral neurological function.

Furthermore, the patient's blood pressure was initially slightly higher on one side but was the same bilaterally when rechecked during the final neurological exam. Blood pressure is partly dependent on peripheral resistance that can be different on either side due to asymmetrical control of vasomotor tone. (40) Increased vasomotor tone can occur because of overstimulation of excitatory vestibulo-sympathetic reflexes. (13, 28) Brainstem regions that regulate ANS function as well as receive input from the vestibular system are mostly found in the pons and medulla. (13) It is common knowledge that the rostral ventrolateral medulla is directly involved in SNS tone regulation, but studies have shown that the vestibular nucleus also plays a key role in ANS regulation during movement and posture change based on the observation that it lead to cardiovascular deficits during orthostatic stress when lesioned. (28, 40) Moreover, vestibular activation on the SNS (through the vestibulo-sympathetic reflexes) has excitatory and inhibitory phases that help protect the body from the effects of orthostatic stress. (13) The vestibulo-sympathetic pathways are thought to be independent from the baroreceptor reflex in that they can be elicited before blood distribution change occurs upon postural alterations. (28)

The present study suggests that the initial oculomotor findings, compounded with the initially higher blood pressure recording on one side, may have originated from brainstem reduced

function contributing to ANS dysregulation. Dysdiadochokinesia is often present with cerebellar dysfunction through different cerebral connections, including the vestibulo-cerebellar and vestibulo-spinal (cerebellar oculomotor) system. (31, 47) Steady reduction of the dysdiadochokinesia was recorded throughout treatment and ultimately resolved. Brainstem and cerebellum structures are intricately connected. (31) Hence, it is suggested that improved brainstem function, as revealed by the final oculomotor and orthostatic stress tolerance assessments, could have ameliorated cerebellar coordination.

After performing the postural challenge exercises including ocular fixation three times daily, for one month, the patient's diagonal saccadic eye movements and tolerance to head/body position variation further improved notably. It is conceivable that these remaining symptoms could have completely resolved with continuation of care. Nonetheless, it is postulated that the theories of habituation, improved gaze stability through retinal slip or a combination of both may have been at play in the mechanism that led to this amelioration.

One hypothesis suggested that habituation can be used to rebalance the vestibular system. (48) Habituation is a learned decreased response to a stimulus acquired from repeated exposure. (48) Initially, the patient experienced nausea/dizziness upon fast horizontal and vertical head movements, such as performed during the bilateral head rotation and stand test, respectively. The repetitive (3x per day) horizontal head movements used in VOR cancellations, as well as the vertical head movements resulting from the supine to standing position change exercises, reproduced those symptom-triggering movements, possibly causing vestibular system habituation, thus improving her tolerance to orthostatic stress.

Another study has shown that the repetition of horizontal and vertical head movement exercises while maintaining visual fixation causes retinal slip that subsequently sends an error signal to the brain, eventually inducing vestibular adaptation. (48) Moreover, the patient initially reported orthostatic symptoms when going from the supine position to standing, as opposed to significant symptom reduction when doing the same thing with ocular fixation. This empirical finding, with the two previously proposed mechanisms in mind, became the basis upon which a home-based exercise program incorporating ocular fixation during orthostatic stress and vestibular activation was devised.

This case study, based on combining noninvasive chiropractic protocols and adjunct therapy, was intended to describe an approach to management of post-concussion POTS that has proven successful with this patient. As with all case reports it is difficult to extrapolate the findings from this patient's presentation and response to care to the general population. Without controls and comparative sham interventions it is not possible to rule out confounders such as placebo/ideomotor effect and regression to the mean so the patient's results need to be appreciated with caution.

Conclusion

As previously stated it is important to note that the chronic, nonspecific, heterogeneous, systemic nature of POTS symptoms and pathomechanisms, along with the absence of orthostatic hypotension, has confounded many health care practitioners. This case report discusses a novel integrative care of a patient suffering from POTS believed to be part of a post-concussion syndrome symptomatology.

Pre and post assessments of the patient appeared to support that a combined therapy of upper cervical AO chiropractic and SOT, postural challenge ocular exercises, and dietary/nutritional modifications had a positive effect on this patients presenting symptomatology. Further research is needed to determine what subsets of patients with POTS might also respond to this conservative chiropractic care approach.

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