28 Year Old Female - 5 years post concussive syndrome

Summary:

Taylor Reynolds presented to my office for evaluation on 12/2/2019 looking for relief of chronic muscle tightness and pain. Consultation shortly revealed a more involved history with complicating symptoms beginning 5 years prior with a concussive/ whiplash event after falling down the stairs. Her primary symptoms included (decreased drive; sense of apathy; daily headache; back pain; neck pain; head pressure; mental fog; severe fatigue; poor short term recall). After 5 years of unsuccessful therapies/ interventions both pharmacolological and non-pharmacological her frustration was high and she felt a full recovery was unattainable. A comprehensive neurological examination was administered uncovering several decompensated pathways and an inflammatory brain environment suspected to be contributing to her maintenance of symptoms. We diagnosed her with **post concussive syndrome complicated by a centrally mediated vestibulopathy and neurogenic inflammation**. A specific brain based rehabilitative program was initiated on-site at Pittsford Performance Care through various multi-modal sensory inputs (described beloiw) for the duration of 3 weeks. Her favorable recovery is detailed below.

Background:

I expect this case will stimulate interest in our readers for 2 primary reasons. 1. The symptomology for Taylor remained and worsened since her event 5 years prior despite several avenues of care. 2. A steep curve of improvement was achieved using a functional neurology intervention model for diagnosis and treatment. *Research has demonstrated that the occurance of traumatic brain based symptomology is 6x more prevelant than diabetes.* Functional deficits as a consequence of decompensated neurological pathways should be seen as valuable diagnostic information relevant in aiding the recovery of a traumatic brain injury.

Case Presentation:

Taylor suffered a concussive event falling down the stairs. Her initial impact was on the tailbone, described as a jarring throughout the entire spine followed by a whiplash of the head and an immediate feeling of head pressure and heaviness. Over several months after the event, Taylor developed extreme fatigue, mental fog, apathy, muscle tightness, and constant headache. The combination of symptoms 5 years post event is relevant because it highlights the negative impact of sustained neurogenic inflammation and the resultant neurologic decompensation.

At this point in the report an important distinction is imperative to understand the role of functional neurology. Taylor did not exhibit pathological neurological signs. Her CT scan post trauma was negative. At the time of her event her bedside neurological exam did not demonstrate hard positive findings. A common interpretation following the above exam is the patients condition is not compromised and any symptoms should resolve over time with rest.

A functional neurological perspective causes the practitioner to evaluate beyond pathology. *Functional brain deficits are a common result of trauma resulting in a decrease in the frequency of firing of neurons in a specific brain region or pathway.* This suppressed state can elicit neurologic symptoms without the presence of pathology. Common symptoms are those described in Taylor's case.

The next section will describe Taylor's full diagnostic detail to include her comprehensive neurological exam. Findings demonstrated soft neurological findings in the following regions/ pathways: Frontal lobe (Pre-frontal/ Orbitofrontal); Frontal Lobe (Supplementary); Frontal Lobe (Broca's Motor Speech Area); Medial Temporal Lobe and Hippocampus; Cerebellum (Spinocerebellum and Vestibulocerebellum); Autonomic Increased Sympathetic Activity. Her clear functional deficits during examination included moderate bradykinesia in right upper extremity; left sided Vestibular Ocular Reflex not intact and triggering severe head pressure with left geocentric vertigo symptoms; Parietal/Cerebellar testing with finger to nose accuracy demonstrated 40% accuracy on the left in comparison to 100% on the right. Because this pattern of decompensation is not following a common neuro-anatomical pathway, an assumption is made that an ongoing neurogenic inflammatory process triggered the cascade of decompensation. This influenced how we prioritized addressing her compromised brain environment. Additionally, the above findings necessitated an early re-examination to recognize the pattern of improvement. *The metabolic capacity of a group of neurons under the influence of inflammation exhibits suppression*. When the inflammation is removed and the metabolic capactiy can return, the specific brain based intervention may need course correction. This is the case for Taylor, as an initial left parietal intervention was replaced with a right intervention at the 1 week mark due to re-examination findings.

Investigations:

Comprehensive bedside neurological exam performed by C.Robert Luckey, Clinic Director assissted by Dr. James Gaffney, Associate Clinician. Initial examination performed on 12/11/2019.

RightEye performance eye diagnostic exam performed on 12/11/2019.

Cognitive exam utilizing Cambridge Brain Science platform performed on 12/11/2019.

Comprehensive bedside neurological re-examination performed by C. Robert Luckey, Clinic Director assisted by Dr. James Gaffney, Associate Clinician. Examination performed on 01/08/2020.

RightEye performance eye diagnostic exam performed on 01/08/2020.

Cognitive exam utilizing Cambridge Brain Science platform performed on 01/08/2020.

PITTSFORD PERFORMANCE CARE

Blood Pressure	Right	Left
Seated	119/78	115/80
Laying	109/69	104/69
Standing (immediate)	120/91	
Standing (post 2 min.)	110/82	
PulseOx		
Heart Rate		

Physical Examination	
Heart	Heart sounds good. Nice valsalva response.
Lungs	Lung sounds are clean.

Reflexes	Right	Left
Tricep	2+	2+
Bicep	2+	2+
Brachioradialis	2+	2+
Patellar	2+	2+
Achilles	2+	2+

Neuro Exam	Right	Left	Notes
Red Desaturation			No red desaturation
Pupillary Fatigue	5 seconds	2 seconds	
UE Extensor Weakness	X		Significant extensor weakness
UE Capillary Refill	3 seconds longer		
Finger to Nose	10/10	4/10	
Finger Tapping	3- speed & amplitude	0	Fatiguing on the right hand.
Rhythm Tapping			

PITTSFORD PERFORMANCE CARE

Neuro Exam	Right	Left	Notes
Pronation/Supination			
Heel to Shin	Normal	normal	
Babinski			
Hamstring Tone	Mild-moderate	Normal	
Anterior Flexor Tone	Mild	Mild	
LE Extensor Weakness			
Graphesthesia	5/5	4/5	
Digit Sense	2/5	1/5	Patient notes they were guessing a lot
Vibration Sense	Intact & appropriate	Intact & appropriate	
Temperature Sense	Intact & appropriate	Intact & appropriate	
Pinwheel Sensation			Pinwheel sensation intact bilaterally. No major differences left versus right globally.
Sound Localization			
Rombergs			
Unterbergers	60 degrees right rotation, no real anterior translation.		
VOR Head Thrust	Head thrust to the right created pain, head pressure, dizziness, and had a massive refixation saccade. Head thrust to the left did not create symptoms like the other direction, no comment can be made on refixation saccade		
Maddox Rod Test			
Eye Movements	*OPK:* in tact to the right, but did create some pressure. In tact to the left also, did not create symptoms (and decreased current symptoms). *Saccades:* refer to RightEye. *Antisaccades:* difficult for patient but scored 10/10.		

Canal Testing	Left	Right
Anterior	BEST- very obvious & clear	poor
Posterior		poor
Horizontal/Rotation		Poor

Differential Diagnosis:

Previous diagnosis included migraine headaches, tension headaches, anxiety and vertigo.

In a broad sense, treatment of the above can be pharmacological or non-pharmacological and she received both. Additionally, 4 years of intervention has led to progression rather than resolution. Furthermore, due to Taylor's wide symptomatic picture, it seemed necessary to search for a thread of connectivity rather than treating in isolation.

Stimulating her left anterior canal for a set of 6 repititions with eyes closed while standing reduced UPRDS score of 3 on the right to a 0. Identifying this diagnostic improvement post canal stimulation indicated a strong functional relationship. Because a central vestibular decompensation can be a cause of migraine headaches, anxiety and vertigo it seemed reasonable to diagnose Taylor with post-concussion syndrome complicated by a central vestibulopathy.

Treatment:

Treatment for Taylor included a comprehensive 3 week (12 hr) receptor driven multi-modal plan designed to have a central brain influence. The plan was authored by Dr. C. Robert Luckey and administered by Dr. James Gaffney. Taylor was recommended a supplementation strategy to reduce neurogenic inflammation and increase substrate to aid in rebuilding her cellular blood brain barrier density. Her rehabilitative program included: Ipsilateral upper extremity and lower extremity counter-clockwise passive figure 8's while supine; ipsilateral V1 stimulation of trigeminal branch; ipsilateral posterior canal stimulation stacked with ipsilateral active UE isometric contraction (eye-lids closed/ eyes moving up and same direction as canal); ipsilateral anterior canal movement with eyes fixated on target; gaze fixation during assissted head movement and without (utilizing Motion Guidance apparatus).

Patient was also given dietary recommendations to follow including removal of gluten and dairy as a precaution as no known allergies were provided.

Outcome/ Follow-up:

Comprehensive bedside neurological re-examination performed by C. Robert Luckey, Clinic Director assisted by Dr. James Gaffney, Associate Clinician. Examination performed on 01/08/2020.

RightEye performance eye diagnostic exam performed on 01/08/2020.

Cognitive exam utilizing Cambridge Brain Science platform performed on 01/08/2020.

Symptoms: **Taylor filled out a Brain Region Localization Form that illustrated her full symtomatic picture with severity before treatment and at the conclusion. They are available for review, but not attached to this PDF in an effort to stay concise. Her verbal description of primary symptoms with percentage improvement as asked on 01/08/2020 are as follows: decreased drive (100%); sense of apathy (100%); daily headache (100%); back pain (90%); neck pain (90%); head pressure (100%); mental fog (100%); severe fatigue (100%); poor short term recall (100%); mental fatigue (100%); body fatigue (100%); and muscle tremors (100%),**

PITTSFORD PERFORMANCE CARE

Blood Pressure	Right	Left
Seated	111/81	120/85
Laying	107/80	125/76
Standing (immediate)		
Standing (post 2 min.)		
PulseOx		
Heart Rate		

Physical Examination	
Heart	Heart sounds are good, no murmurs noted. Nice valsalva response.
Lungs	Lung sounds are clean. Rib expansion noted to be 5cm.

Reflexes	Right	Left
Tricep	2+	2+
Bicep	2+	2+
Brachioradialis	2+	2+
Patellar	2+	2+
Achilles	2+	2+

Neuro Exam	Right	Left	Notes
Red Desaturation			No red desaturation
Pupillary Fatigue	8 seconds	12 seconds	
UE Extensor Weakness			No notable extensor weakness.
UE Capillary Refill	5 seconds	6 seconds	
Finger to Nose	10/10	9/10	

PITTSFORD PERFORMANCE CARE

Neuro Exam	Right	Left	Notes
Finger Tapping	0	0	
Rhythm Tapping	5/5		
Pronation/Supination	5/5 no bradykinesia/dysmetria		
Heel to Shin	normal	Normal	
Babinski			
Hamstring Tone	normal	normal	
Anterior Flexor Tone	normal	normal	
LE Extensor Weakness			
Graphesthesia	5/5	4/5	
Digit Sense	5/5	5/5	
Vibration Sense			
Temperature Sense			
Pinwheel Sensation			Intact, appropriate and symmetrical globally.
Sound Localization			
Rombergs	Negative, no sway		
Unterbergers	50 degrees right rotation. After 5 reps VOR fast phase right slow phase neutral X1 fixation there was no notable rotation.		
VOR Head Thrust	No refixation saccade, really accurate bilaterally.		
Maddox Rod Test			
Eye Movements	*OPK:* intact & accurate both directions. *Saccades:* refer to RightEye *Antisaccades:* 10/10 not as fatiguing		

Canal Testing	Left	Right
Anterior		
Posterior		
Rotation		

Discussion:

I suppose a case of this nature may generate more questions than answers due to the fact that is resides in a space where delineated clinical guidelines do not exist. Current clinical policy as of 2013 dictates a clear clinical path regarding acute mild traumatic brain injury (mTBI), however, there is not a common understanding of post acute mTBI guidelines.

In an article published in 2013 in the Journal of NeuroTrauma by the title, 'Current Recommendations for the Diagnosis and Treatment of Concussion in Sport: A Comparison of Three New Guidelines', an effort was made to summarize a common understanding of definition, risk factor, symptoms, diagnosis, imaging studies, balance testing, neuropsychological testing, return to play/ school, long-term effects, prevention and management. Three groups to include the American Medical Society for Sports Medicine, The American Academy of Neurology, and the Zurich Consensus working group set a goal to clearly define the best practices as it relates to the above, to include the management of post-acute mTBI.

Statements where concensus was reached were published addressing all of the above with the exception of post-acute mTBI management. This is not suprising, but rather ironic as the greatest need lives in the millions of individuals suffering from the neurological cascade post trauma that deliver ongoing symptomology. In short, once the life threatening sequela are ruled out in the hospital setting and the patient is released, where do they go? I believe a debate for best practices will continue for decades, however, I also believe that practitioners who are skillfully trained in the practice of Functional Neurology have a unique examination/ perspective and treatment platform that can service the post-acute mTBI with specificity and efficacy.

Learning Points/ Take Home Message:

Post concussion syndrome is common. The symptoms that exist in the presence of this syndrome can be mild to debilitating in severity. Post-acute treatment for mTBI is not agreed upon providing poor patient resource relative to diagnosis, guidance and treatment. A functional neurological approach to this condition can provide positive clinical improvement.

Relative to Taylor, the key to her profoundly positive swing is symptomology is theorized to be a combination of the following: reducing her neurogenic inflammation and activating cortical and sub-cortical pathways that exhibited suppression.

The neurological impact of a concussive event can and often will heal on its own. However, a brain that experiences multiple insults or a severe insult can lead to a cascade of neurophysiological, neurochemical, and neuropsychological events that cannot be self-regulated.

References: Include only relevant references (New England Journal of Medicine Style)

Patient's Perspective:

My experience at Pittsford Performance Care with Dr. Luckey and Dr. Gaffney was incredible. Prior to visiting PPC, I had already seen a multitude of doctors, gone through a series of MRI and CAT scans, tried multiple medications including injections. Nothing worked for me. I was suffering with severe tension headaches, back pain, neck pain, fatigue and muscle tremors for a period of 5 years. You could say that I had lost hope until my first appointment with Dr. Luckey a few months ago. I always knew exactly what to expect with my treatment and always felt like I was in the best hands possible. Although I started to feel a positive change after my first treatment, Dr. Luckey and Dr. Gaffney were always very cautious with me and paid close attention to how I reacted to certain treatments. I very much appreciated this because I felt like I really understood what the treatment consisted of. I can easily say that Pittsford Performance Care gave me my quality of life back and I am so incredibly thankful for the experience. - *Taylor Reynolds*