

Traumatic brain injury—missed diagnosis: a case report

Abstract

We present a now 51-year-old woman who sustained a head injury in a rear-end collision at the age of thirty five after the vehicle she was operating was struck by a moving vehicle while her vehicle and the rest of traffic was at a standstill. She was subjected to acceleration-deceleration, concussive, contusive and contrecoup-coup forces. Although significant traumatic brain injury (TBI)-related signs and symptoms developed and persisted, she had never been formally evaluated or treated for mild traumatic brain injury until 2016. We present this case report to raise outpatient provider awareness about atypical presentations of mild traumatic brain injuries and long-term sequelae; to raise the index of suspicion of providers; to trigger earlier and more efficacious diagnostic imaging and treatment in such patients.

Keywords: brain, closed, concussion, contusion, contrecoup, coup, diagnosis, head, injury, missed, traumatic

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Abbreviations: CTBI, closed traumatic brain injury; DAI, diffuse axonal imaging; TBI, traumatic brain injury; MOI, mechanism of injury; MVA, motor vehicle accident; MRI, magnetic resonance imaging

Introduction

Motor vehicle accidents are a common cause of traumatic brain injury, muscle, spine and nerve injury.^{1,2} Some injuries are the result of the skull or body impacting stationary objects via direct contact. Other injuries are the result of indirect contact, i.e., rapid acceleration and deceleration forces pitted against the head, neck and torso.³⁻⁵ Studies have shown that a substantial number of head injuries and traumatic brain injuries go unrecognized and untreated even after the patient reports to a primary care provider, urgent care center, accident center or emergency room.⁴⁻⁹ Signs and symptoms of an otherwise treatable condition can then continue to evolve and persist for months, years or even decades after the accident.^{8,10-13}

The patient was in a rear-end collision at the age of 35 when the two-door compact car she was driving was struck from behind, while at a standstill, by a 16-passenger van. She has experienced signs and symptoms of TBI since the accident. However, she was never assessed for or diagnosed with traumatic brain injury until February 2016 when she was finally evaluated for and diagnosed with Mild to Moderate Traumatic Brain Injury-Concussive Type with Acute Cognitive Deficits and Balance Deficits. The diagnosis was based on the presence of related signs and symptomatology and supported by the presence of a Virchow Robbins Space and Chiari Malformation (known correlates of CTBI) per a February 2016 magnetic resonance imaging study of her head and brain.¹⁴⁻²⁶ The February 2016 magnetic resonance imaging (MRI) was the first head diagnostic image taken since the accident and a 1999 pre-accident MRI of the cervical spine was negative for craniocervical junction abnormalities.

History of presenting illness

The moderate rate of speed at impact was 15-30mph and the attendant velocity created enough momentum to subject the patient's head, neck and torso to sudden rapid thrusting and snapping forward

and backward movements. While in rearward motion her head and torso each respectively came into forceful contact with the seat headrest, seat belt and seat back, two times each. That is to say, she was subjected to acceleration-deceleration, concussive, contusive and contrecoup-coup forces during the event. At impact and during the entire event the patient's head was turned to the left. Once the momentum subsided and her head, neck and torso came to a resting position she immediately felt dazed and dizzy. Additionally, she experienced pain in her head, neck, shoulder area and upper back areas. Over the next few weeks and months a myriad of other signs and symptoms subsequently manifested. Some were physical in nature while others were mental, psychological or emotional.

The patient's extensive historical medical record indicates that several medical providers documented numerous signs and symptoms but attributed them to various diagnoses such as fibromyalgia, chronic cervical strain, clinical depression secondary to chronic pain, sjogren's syndrome. It took between 1-16years to receive the correct assessments, tests, diagnoses and treatment (including the current TBI related diagnosis and treatment). The record indicates that within a few minutes of the accident the patient began to complain of dizziness, head neck, shoulder and back pain, which took three weeks to be identified as nerve damage by a chiropractor. Thus it can be inferred that various neuropathies began to develop instantaneously. However, it took nine years for a double boarded neurologist/physiatrist to perform an electromyogram (EMG) and find denervation of the left long thoracic, C-7 nerves and twelve years for another identically boarded physician to find left C-8 denervation. Scapular deformity and dysfunction manifested within 24hours and was first identified as such, two weeks later, by a nurse practitioner thru inspection and palpation. Yet it took one year for it to be identified as scapular winging by a physical therapist. Moreover, despite ongoing documentation of symptomatology it took an additional nine years for an orthopedist to identify it as scapular winging then orders the aforementioned EMG that revealed long thoracic and C-7 injury. Further, it took several days for trigger points to manifest and 2-3weeks for this finding to be identified as such by a nurse practitioner and chiropractor, respectively, via palpation and inspection but took one year for the patient to be diagnosed with chronic and persistent myofascial pain

per rheumatologist. Lastly, it took several days, weeks, months and years for post concussion signs and symptoms to manifest and be documented but sixteen years for them to be recognized as such and to be diagnosed with persistent mild traumatic brain injury.

Subjective and objective findings

The patient has experienced the following (TBI related signs and symptoms since the 1999 MVA):

Intermittent: exacerbations of overwhelming sadness, clinical depression, clinical anxiety; protracted periods of rumination and preoccupation with death and dying (abated in the last 1.5years), and visual problems.

Persistent: significant difficulties with memory retrieval, forgetfulness, immediate short– term memory, remote long– term memory (childhood memories are essentially lost or very delayed in retrieval); mental fogginess, feeling slowed down; significant moderate level difficulties maintaining attention, concentration, persistence, performing more than one task at a time; slow processing, poor time management and increased distractibility, disorganization, increased rumination and perseveration; fatigue, extreme sensitivity to light, noise and fumes/odors, drowsiness, sleep difficulties (vacillating severe insomnia–days at a time and hypersomnia–long hours); trouble with balance, abnormal Heel–toe walk test, abnormal Romberg test, depressed and anxious mood, emotional lability and affect (pseudobulbar–type affect i.e., crying during commercials, laughing during times of somberness, extreme irritability at times). Such signs and symptoms are strongly correlated with CTBI and Post–Concussive Syndrome.^{8,11–13,26–29} Chronic and Persistent Pain associated with diagnoses of chronic myofascial pain syndrome; primary sjogren’s syndrome; subacute cutaneous lupus erythematosus; inflammatory joint disease; scapula, shoulder, spine dyskinesia dysfunction.

Diagnostic imaging of head and brain

Medical records from December 1999 to December 2015 indicate that no brain or head diagnostic imaging was ever performed in that time frame following the accident. Said records do indicate that several cervical MRI studies were performed, however, none address or describe the diagnostic picture of the craniocervical junction. In February 2016, the patient received a brain MRI using a magnetic field strength of 3.0 Tesla. Axial and sagittal T1 weighted, axial T2 weighted, axial and coronal flair, axial diffusion images were obtained through the head. Thin section, fat–suppressed axial T2 weighted images through the posterior fossa were obtained. Susceptibility weighted images with maximal intensity projections were also obtained. The MRI report review revealed “findings of a prominent 4 mm perivascular space within the left parietal lobe (Virchow Robin Space), 5mm cerebellar tonsillar ectopia (Chiari Malformation) with tonsils demonstrating normal morphology and flow voids in the circle of willis were made.” As aforementioned the first two findings are strongly correlated with traumatic brain injury.^{15–26} The MRI was otherwise unremarkable.

Assessment and diagnosis

Chronic Mild to Moderate Traumatic Brain Injury–Concussive Type with Persistent Cognitive Deficits and Balance Deficits and MRI Correlates per patient’s 2016 medical records.

Plan

i. Referred to Neurologist secondary to associated diagnosis of

dizziness and associated balance deficits–positive Romberg test and positive Tandem Walk test.

- ii. Referred to Physical and Occupational Therapy for Vestibular Dysfunction Therapy secondary to associated diagnoses of persistent dizziness and balance deficits including a positive Romberg test and positive Tandem Walk test.
- iii. Referred to Neuropsychologist for Cognitive Testing secondary to associated diagnosis of TBI–Concussion and numerous persistent related signs and symptoms of impaired cognition.
- iv. Referred to Integrative Cognitive Rehabilitation Program secondary to associated diagnosis of TBI–Concussion and persistent related signs and symptoms of impaired cognition.

Discussion

TBI can be classified as a neurological disorder or an organic mental disorder.³⁰ In the former case neurological impairments such as decreased levels of consciousness, epilepsy, cerebrovascular accidents, disorganization of motor function, and vestibular dysfunction can manifest. Whereas in the latter case psychological or behavioral abnormalities associated with a dysfunction of the actual brain manifest, such as any or all of the ones that have been experienced by our patient. History and physical examination and/or diagnostic studies and/or laboratory tests will typically demonstrate the presence of a specific organic factor etiologically related to the abnormal mental state and loss of previously acquired functional abilities. This patient’s symptoms are classified as both. A working hypothesis for why this diagnosis was delayed for so long is that 16years ago concussions were historically and widely considered to be a separate entity from traumatic brain injuries. Much of the literature and practice protocols held that concussions were “briefly disabling transient disorder of the brain without long–term sequelae”.^{27,29,31} Further, a loss of consciousness was required in order for a head injury to be labeled a concussion or for said concussion to be considered significant.^{32–33} Thus they were often ignored or dismissed as minor if an overt loss of consciousness had not occurred or overt physical signs of trauma and dysfunction were not present. Conversely, current evidence based literature and research establish that a concussion, by definition, is properly classified as a TBI and is the most common type of CTBI.^{27,32,34} Moreover, the lack of loss of consciousness does not necessarily correlate with the severity of a TBI or the chronicity of attendant symptomatology.^{5,35,36} Thus evaluation and follow– up of such an event is warranted under every circumstance where there is direct and indirect trauma involving the brain.^{35,37,38} Since records do not indicate the type of MRI and size of slices, this introduces diagnostic ambiguity. Standard MRI’s have 6mm unseen slices due to standard MRI cervical protocol which includes a section that is skipped to save time. Therefore, one must order thin 2mm stacked no–skip to get best read in the cervical region. Since the Chiari was 5mm this could be why it was missed. Thus, it is suggestive to suggest that all MVA cases be handled with smaller slices in the MRI.

Another possible reason for the missed diagnosis, in this case, is that healthcare students and providers have experienced a learning gap in the identification and management of traumatic brain injury. Confusing or overlapping language furthers this gap e.g. concussion is a type of TBI yet it is often referred to as if it is a separate and less serious entity.^{27,32} Lack of knowledge, experience or training in the arena of TBI leads to an inability to recognize related or atypical signs and symptoms and improper evaluation and assessment. Fear of the unknown and unwillingness to step out of one’s professional comfort zone or scope of practice also contributes to gaps in care e.g. failure

to investigate signs and symptoms, order diagnostic tests for all head injury patients or make appropriate referrals. Such language confusion, learning gaps, and personal or professional considerations lead to misconceptions and biases in the diagnostic process. For example, in many cases concussion and post concussion syndrome are treated with a light touch instead of as the serious life impacting conditions they are. A watch and wait approach is taken and the patient is told that the symptoms are nothing to worry about and usually resolve on their own within a few days or months.²⁷ In turn, this produces ambiguous science, poor clinical guidelines and confused policies e.g. only ordering a CT or MRI if the patient experience loss of consciousness instead of on all head injury patients. Overall, these factors typically force patients with invisible, hidden, confounding, rare or atypical signs, symptoms and concurrent conditions to take the long way home to wholeness from symptom onset to diagnosis and treatment.

In order to prevent a missed diagnosis of traumatic brain injury, facilitate appropriate and efficacious treatment sooner and speed patients along the road to recovery several hurdles must be overcome. The first hurdle that must be overcome is knowledge deficiency. Providers will benefit by recognizing that:

- i. TBI is interchangeably a noun, a verb and a medical diagnosis.
- ii. TBI is defined as transient or long lasting disruption of brain structure and or function caused by an external mechanical force.^{36,39}
- iii. Closed TBI's are caused by direct contact of the brain with the skull after an external source has directly or indirectly impacted or caused movement of the skull, i.e., blasts, blows, falls, jarring, shaking, etcetera.
- iv. These causative factors encompass several different mechanisms of injury including acceleration, acceleration–deceleration, concussion, contusion, contrecoup, coup, contrecoup– coup, coup–contrecoup and deceleration.
- v. More than one mechanism of injury can simultaneously occur during the event.^{3,5}
- vi. The various mechanisms of injury must be well understood, ascertained during history taking and evaluated in order to properly assess the level of damage to the patient.
- vii. Concussions are not a separate diagnostic entity but are actually a type of TBI and can result from and/or cause any of the other mechanisms and types of TBI.³⁴
- viii. Any type of TBI can range in severity, symptomatology and persistence with or without loss of consciousness.
- ix. Neuropsychological impairments are often expressed in the form of increased mental foginess, short and long term memory impairment, delayed problem solving, slowed reaction time, slow response to questions and decreased awareness of problems, concentration, attention span, awareness of one's surroundings, organization, planning, follow through on decisions, judgement, insight, reasoning.
- x. Atypical presentations are often expressed in the form of persistent anxiety, depression, fatigue, impulsivity and personality changes and continued ability to perform previous educational, household or occupational tasks (but at a decreased level of concentration, pace or persistence).

A clear basic understanding of the mechanisms of injury is the second hurdle that must be overcome. Providers must understand that

the amount of damage to a vehicle does not correlate with the amount of damage to occupant(s) and head position and the headrest position can minimize or significantly contribute to injury. In this case the patient's left turned head contributed to the severity of her injuries as evidenced by the location of the findings per MRI and her associated signs and symptomatology.^{40–45} Further, that: 1) the brain is widely considered to be the most fragile organ in the body and the most susceptible to injury with even the slightest provocation. 2) The brain is lipid dense, has a gelatinous consistency, and is subject to neuronal shearing with trauma and liquefactive necrosis with infarction or fungal/bacterial infection, unlike almost all other tissues/organs. 3) CTBI can cause an insult to the brain and central nervous system at the moment of impact (primary injury) or several minutes, hours, days, weeks or months later (secondary injury). 4) The aforementioned mechanisms of injury can cause direct or indirect trauma to the brain by deformation and/or movement. Finally, in general the basic mechanical action of any of the MOI or TBI classifications is most graphically and accurately compared to shaking, tapping or throwing an egg. When an egg is being shaken, tapped or thrown, the yolk, like the brain inside a skull, moves against the inside of the shell, shifts around as it is contained inside the shell and changes its shape, position and consistency. The protective shell surrounding the egg yolk and whites does not prevent either from moving around inside any more than the skull prevents the brain from moving around inside. As described the force of direct or indirect impact can still affect the yolk contained inside the eggshell just as it does with the brain inside the skull.

Conclusion

Fortunately, with regard to the now established diagnosis of persistent TBI– Concussion, the patient now has a new treatment plan. Our purpose in presenting this case–study has been to raise outpatient provider awareness about atypical presentations of mild traumatic brain injuries e.g. anxiety, depression, personality changes, psychiatric disorders and vestibular dysfunction; overlooked signs and symptoms e.g. persistent clumsiness, increased irritability, preoccupation with death and ruminations; long term sequelae e.g. persistent dizziness, headache, imbalance, neck ache, decreased ability to perform previous functions at the same level or at all, slowed ability to perform a timed cognitive task and clumsiness;^{11,13} to raise the index of suspicion of providers; and to trigger earlier and more efficacious diagnostic imaging and treatment in such patients.

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Conflict of interest

The author declares no conflict of interest.

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