Jane Doe – Medical Opinion

DOB: MM/DD/1940

DOI: MM/DD/2018

Case summary:

Ms. Jane Doe was then a YY-year-old female was involved in an accident on MM/DD/2018 at 3:00 P.M. When she bent over to hand over garment to the attendant through the window, she hit her head on the crossbar/iron pole of the opening desk. She was stunned and had to move back. She developed a knot/swelling, laceration on her forehead and had headaches. After 4 months, on MM/DD/2018, she presented to Ezekiel XX, M.D. (XXX Clinic) for headache.

***Missing records**: The records from MM/DD/2018 till MM/DD/2018, if any were unavailable.*

Past medical history: Gastroesophageal reflux disease, hiatal hernia, spondylolisthesis, and allergic rhinitis, diverticula of colon, abnormal mammogram, shoulder pain and brachial neuritis and Hypertension, knee replacement and gallstones.

Past surgical history: Bilateral knee replacement, Cataract removal in left eye, Gallbladder removal, Gland in right eye and Hysterectomy.

On MM/DD/2018, Ms. Doe presented to Ezekiel XX, M.D. (XXX Clinic) for headache. She was prescribed gabapentin 100mg. She was advised CT angio of brain and review in 4 weeks.

On MM/DD/2018, she consulted Mary XXX AGAACNP-BC (XX Health) for left shoulder, neck pain and headache. On evaluation, she was found to have restricted cervical spine range of motion associated with paraspinal and left upper trapezius spasm.

Ms. Doe was diagnosed to have

- Left shoulder pain
- Neck pain
- Headaches
- Status post personal injury
- Insomnia

She was advised physical therapy and medicines. She was advised X-ray cervical spine and CT head and to review in 3 weeks.

X-ray of cervical spine done on MM/DD/2018 demonstrated straightening of cervical spine, C5-C7 degenerative changes.

Ms. Doe was initiated in chiropractic therapy from MM/DD/2018 till MM/DD/2018.

MRI Lumbar spine done on MM/DD/2018 demonstrated grade 1 L3-L4 anterolisthesis associated with disc herniation L1-S1.

MRI left shoulder done on MM/DD/2018 demonstrated high grade supraspinatus tear associated with effusion and acromioclavicular arthritis.

CT brain done on MM/DD/2018 was normal.

MRI Cervical spine done on MM/DD/2018 demonstrated C5-C7 disc height loss associated with degenerative changes at C4-T1.

On MM/DD/2018, Ms. Doe consulted Nooruddin XX, M.D. (XXXXX) for neck and low back pain associated with left lower limb pain. She was recommended epidural steroid injection.

She underwent L5-S1 lumbar epidural steroid injection performed by Dr. XX (XXXXX) on MM/DD/2018.

Ms. Doe underwent C7-T1 Cervical epidural steroid injection performed by Dr. XX (XXXXX) on MM/DD/2018.

On MM/DD/2018, she reviewed with Dr. XX (XXXXX) for neck and low back pain.

Ms. Doe underwent L4-L5 lumbar epidural steroid injection performed by Dr. XX (XXXXX) on MM/DD/2018.

She underwent C7-T1 Cervical epidural steroid injection performed by Dr. XX (XXXXX) on MM/DD/2018.

On MM/DD/2018, Ms. Doe reviewed with Dr. XX (XXXXX) for neck and low back pain.

She underwent L2-L3 lumbar epidural steroid injection performed by Dr. XX (XXXXX) on MM/DD/2018.

On MM/DD/2018, Ms. Doe reviewed with Dr. XX (XXXXX) for neck and low back pain.

On MM/DD/2018, she consulted Huma XX, M.D. (XXX Institute) for head injury.

Ms. Doe was diagnosed to have

- Post traumatic headaches/migraines
- Traumatic brain injury with neuro-cognitive deficits
- Depressive disorder/generalized anxiety disorder
- Insomnia
- Post traumatic tinnitus, vertigo and vestibular ataxia
- Cervical and lumbar radiculopathy
- Intention tremor

On MM/DD/2019, she reviewed with Dr. XX (XXX Institute) for head injury.

Ms. Doe was diagnosed to have

- Moderate Neuro-cognitive Deficits due to Traumatic Brain Injury
- Depression
- Generalized Anxiety Disorder
- Post-Traumatic Headache
- Bilateral Motor Weakness of Hands
- Post-Traumatic Vestibular Dysfunction
- Insomnia

She now suffers from numerous deficits: Cognitive, psychiatric, psychological, emotional, and physical. She was advised medicines; ultrasound guided greater/lesser occipital nerve block, speech therapy, vestibular and neuro rehabilitation.

The test of pre-morbid functioning score done on MM/DD/2019 was average.

On MM/DD/2019, Ms. Doe reviewed with Dr. XX (XXX Institute) for traumatic brain injury.

On MM/DD/2019, she reviewed with Dr. XX (XXX Institute) for traumatic brain injury. She still complained of headaches, dizziness, ringing in ears, trouble focusing and left shoulder/neck pain. There was improvement in sleep, headache, attention and mood.

Opinion:

1. **Whether the persistent headache was due to the accident on MM/DD/2018?**

Yes, the persistent headache was due to the accident on MM/DD/2018.

This could be proved by the following points:
Ms. Jane Doe was then a YY-year-old female was involved in an accident on MM/DD/2018 at 3:00 P.M. When she bent over to hand over garment to the attendant through the window, she hit her head on the crossbar/iron pole of the opening desk. She was stunned and had to move back. She developed a knot/swelling, laceration on her forehead and had headaches (Ref1)
On MM/DD/2018, she consulted Huma XX, M.D. (XXX Institute) and was diagnosed to have posttraumatic headache/migraine (Ref2)
She was diagnosed to have Post traumatic headaches/migraines, traumatic brain injury with neuro-cognitive deficits, Depressive disorder/generalized anxiety disorder, Insomnia, Post traumatic tinnitus, vertigo and vestibular ataxia, Cervical and lumbar radiculopathy and Intention tremor

Reference:

Ref1: https://www.uptodate.com/contents/acute-mild-traumatic-brain-injury-concussion-in-adults?search=traumatic%20brain%20injury&topicRef=4825&source=see_link

Acute symptoms and signs: The hallmark symptoms of concussion are confusion and amnesia, sometimes with, but often without, preceding loss of consciousness [1,44]. These symptoms may be apparent immediately after the head injury or may appear several minutes later [45]. It is important to emphasize that the alteration in mental status characteristic of concussion can occur without loss of consciousness. In fact, the majority of concussions in sports occur without loss of consciousness and are often unrecognized [46].

The amnesia almost always involves loss of memory for the traumatic event and frequently includes loss of recall for events immediately before (retrograde amnesia) and after (anterograde amnesia) the head trauma. An athlete with amnesia may be unable to recall details about recent plays in the game or details of current events. Amnesia also may be evidenced by the patient repeatedly asking a question that has already been answered.

Other early symptoms of concussion include headache, dizziness (vertigo or imbalance), lack of awareness of surroundings, and nausea and vomiting; these may immediately follow the head trauma or evolve gradually over several minutes to hours [45]. Over the next hours and days, patients may also complain of mood and cognitive disturbances, sensitivity to light and noise, and sleep disturbances [47].

While many concussions occur without observed findings [44], signs observed in someone with a concussion may include [45]:

Grossly observable incoordination (stumbling, inability to walk tandem/straight line)

As well as neuropsychiatric impairments, including:

- Vacant stare (befuddled facial expression)
- Delayed verbal expression (slower to answer questions or follow instructions)
- Inability to focus attention (easily distracted and unable to follow through with normal activities)
- Disorientation (walking in the wrong direction, unaware of time, date, place)
- Slurred or incoherent speech (making disjointed or incomprehensible statements)
- Emotionality out of proportion to circumstances (appearing distraught, crying for no apparent reason)
- Memory deficits (exhibited by patient repeatedly asking the same question that has already been answered or inability to recall three of three words after five minutes)

Occasionally, associated transient neurologic deficits, such as global amnesia or cortical blindness, can occur. The pathogenesis underlying these symptoms is not well understood; it is speculated that vascular hyperreactivity and trauma-induced, migraine-equivalent phenomena may play a role [48-50].

Less common are cranial nerve deficits such as extraocular muscle weakness, vertigo, and nystagmus. (See "Sequelae of mild traumatic brain injury", section

on 'Other cranial nerve injuries' and "Sequelae of mild traumatic brain injury", section on 'Post-traumatic vertigo and dizziness'.)

Clinical findings **NOT** consistent with mild, uncomplicated TBI include focal neurologic findings such as limb weakness or hemiparesis, visual field deficit, pupillary abnormality, or Horner syndrome. These should be evaluated independently. A stroke syndrome, in particular, raises suspicion for traumatic vascular injury, while paraparesis or paraplegia suggests spinal cord injury. These presentations and their evaluation and management are discussed separately in individual topic reviews.

Seizures: Early post-traumatic seizures are those that occur within the first week after head injury. These seizures are considered to be acute symptomatic events and not epilepsy. Post-traumatic seizures occur in less than 5 percent of mild or moderate TBI, and they are more common with more severe TBI, especially if complicated by intracranial hematoma [51,52].

Approximately half occur within the first 24 hours of the injury; one-quarter occur within the first hour [52,53]. The earlier a seizure begins, the more likely it will be generalized in onset; after the first hour more than half are either simple partial (pure motor) seizures or focal with secondary generalization [51,52].

Early post-traumatic seizures increase the risk of post-traumatic epilepsy by fourfold, to more than 25 percent [52]. While antiseizure drugs may be used in the treatment of early seizures, they are not helpful in the prevention of post-traumatic epilepsy.

Ref2: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5237624/

There are pathophysiological alterations in migraine headache that may underlie a physiological susceptibility to concussion and post-concussion symptoms. This is supported by previous research suggesting a common molecular pathophysiological cause of migraine and mild traumatic brain injury (mTBI).[10-12] For example, extracellular potassium and intracellular sodium, calcium, and chloride are all increased with respect to their usual concentrations in the brain after mTBI as well as in migraine.[13-15] Furthermore, both mTBI and migraine are associated with an excess release of excitatory amino acids such as glutamate, in addition to beta-endorphins.[10,14] Mild head trauma may also stimulate trigeminal sensory afferents through cervicogenic mechanisms similar to those seen in migraine.[16] Following forced flexion and extension of the cervical spine, upper cervical sensory nerve roots converge on the trigeminal nucleus caudalis, resulting in the sequential activation of second and third order neurons in the nociceptive pathways of the brain stem, hypothalamus, and thalamus. This in turn can lead to cortical spreading depression. [16] If damage to cervical structures also occurs during the mTBI, nociceptive input from cervical segments arriving to the trigeminal nucleus may also serve as a source of referred pain, ultimately resulting in mechanical hyperalgesia.