International Journal of Psychiatry Sciences
Online ISSN: 2664-925X, Print ISSN: 2664-9241
Received: 01-02-2019; Accepted: 02-03-2019; Published: 09-03-2019
www.psychiatryjournal.net
Volume 1; Issue 1; 2019; Page No. 20-22

Cauda equina syndrome in a patient injured in a car accident

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Abstract
Objective: Milder forms of the cauda equina syndrome remain often undiagnosed, particularly in persons injured in motor vehicle accidents (MVAs).
Method: Review of literature on cauda equina syndrome and the presentation of a case history.
Case History: A 50 year old lady injured in an MVA 18 months ago still reports numerous post-concussive symptoms as well as the following symptoms of the cauda equina syndrome: numbness in her left leg and in the "saddle area," severe urinary incontinence, instances of moderate loss of muscular control over her left leg, and severe tingling and some loss of feeling in her left leg.
Due to her residence in a very remote and medically underserviced area, a physician treated her symptoms with opiate based analgesics which partly obscured some of her symptoms and delayed diagnosis.
Discussion and Conclusions: More attention is needed by staff of emergency departments to warning signs of cauda equina syndrome in patients injured in MVAs. Rather than continuing to overmedicate patients with the addictive opiate based analgesics, scientific research is urgently needed on therapeutic use of relatively inexpensive, non-addictive, non-euphoric, and neuro-protective powerful anti-inflammatories such as cannabidiol for post-MVA pain and for non-surgical management of mild symptoms of the cauda equina syndrome.

Keywords: cauda equine syndrome, cauda equina, lumbosacral spine, car accidents

1. Introduction

The diagnosis of cauda equina syndrome is often missed by busy emergency physicians and it also remains undiagnosed by physicians in general practice, especially in elderly patients [1]. As reported in this article, one of the causes of cauda equina syndrome are injuries from car accidents.

Basic anatomical features of cauda equina are as follows. The numerous nerves branching out from the lower end of spinal cord are known as the cauda equina, due to their anatomical resemblance to a horse's tail. The neurological function of the collection of these nerves is, in the afferent direction, to receive messages from pelvic organs and from the lower limbs, or in the efferent direction, to send messages to pelvic organs and to the lower limbs. Briefly, the nerves in the cauda equina region allow the brain to receive messages from pelvic organs and lower limbs or to conduct messages from the brain to pelvic organs and lower limbs. Thus, the nerves within the cauda equina provide motor and sensory functions between the brain and the legs, the pelvis, and the urinary bladder.

From an anatomical perspective, the spinal cord ends at the upper portion of lumbar spine, i.e., within the lower back. Individual nerve roots that branch out from the end of the spinal cord continue along in the spinal canal. The cauda equina is the continuation of these nerve roots in the lumbar region. As already mentioned, their function is to provide motor and sensory function to the legs, the pelvis, and the bladder.

A frequent cause of cauda equina syndrome includes severely herniated discs in the lumbar region. This occurs as a result of age related degeneration, or of violent injuries such as from falls, car accidents, or gunshots. Predisposing factors are also spinal tumors [2], or spinal lesions, spinal infections or inflammations, birth abnormalities, or spinal hemorrhages. Other causes may include damage from postoperative complications of lumbar spine surgery. Spinal anesthesia can also be involved as the triggering factor for cauda equina.

Older persons are more vulnerable to the cauda equina syndrome because the spinal disc material degenerates with age and its ligaments weaken: a relatively minor strain or jolts or a twisting movement can cause the disc to rupture.

The symptoms subjectively reported by the patient usually include severe pain in the lower back, numbness over the gluteus area, between upper legs, and over lower abdomen (so called "saddle anesthesia"), recent onset of bladder dysfunction such as urinary incontinence or retention, or of bowel incontinence, sensory abnormalities in the bladder or rectum, sexual dysfunction, and symptoms in lower limbs or pelvis such as muscular weakness, numbness, pain, or loss of normal reflexes [3, 4, 5].

In mild or in initial stages, the patient may report being "asymptomatic at rest, but develops pain, weakness, heaviness or tiredness in the legs after standing erect or walking" [6].

2. Published Cases of Cauda Equina Syndrome Caused by Car Accidents

The most noteworthy report on cauda equina syndrome from a car accident so far was published by a group of Taiwanese neurosurgeons and neuroscientists headed by Muh-Shi Lin in 2013[7]. Their report includes two cases.

The first patient was a 27 year old male, a car driver involved in a vehicular accident, who presented with "dyspnea, abdominal and back pain, bilateral lower extremity weakness, saddle anesthesia, and acute urinary

retention." Computerized tomography (CT) scan and magnetic resonance imaging (MRI) showed "a Denis type A burst fracture involving the superior and inferior endplates at L5 with retropulsion of bone fragments into the spinal canal." The investigation showed that "the canal displacement of the retropulsed fragment was 60%, which was a result of intracanal fragment fracture and disruption of the posterior longitudinal ligament."

The case management was described as follows: "He underwent L4–S1 laminectomies

with short-segment transpedicle screws and rod system instrumentation between L4 and S1 25 hours after the injury. Retropulsed bone fragments were removed without impacting them to achieve adequate decompression."

As a result of the excellent and timely surgical intervention by Muh-Shi Lin's team, the "urinary retention improved and the Foley catheter was removed 2 months later." Their patient "gradually regained full muscle power in both lower extremities within 2 weeks. CT scan at 18 months showed good consolidation of the L5 burst vertebral body, and the percentage of spinal canal compromise was 0%, a result of posterior decompressive laminectomies."

The second case was a 25 year old female involved in the same car accident as a passenger. She presented with severe lumbosacral pain and with leg numbness. The CT and MRI of lumbar spine showed that "canal displacement of the retropulsed fragment was 55%" and it also indicated "fracture of the superior endplate; therefore, the fracture was a Denis type B burst fracture."

The patient experienced urinary retention within 6 hours after the injury: Foley catheter was inserted. Surgical management of this patient was described by Muh-Shi Lin's team as follows: "She underwent L4–S1 laminectomies with short-segment transpedicle screws and rod system instrumentation between L4 and S1 8 hours after the injury. Retropulsed bone fragments were removed without impacting them. The spinous processes were harvested for autograft posterolateral bone fusions."

As a result of early surgical intervention and medical management, this female patient's urinary retention gradually improved and the Foley catheter was removed within 15 days after surgery."

In both patients, the surgical intervention was performed within 48 hours of their car accident.

3. Case History of a Canadian Patient Injured in a Motor Vehicle Accident

Our patient is a 50 year old female involved in a motor vehicle accident (MVA) 18 months ago. She wore her seatbelt. While she proceeded on the green traffic signal through an intersection, a young male driver from the street on the right ran the red traffic light, drove into the intersection, and collided into our patient's car. She was jolted by the impact and thrown to the side. She is retrospectively unable to determine whether or not she partly or fully lost consciousness for a brief time span, however, she recalls that she subsequently felt dazed, stunned, confused, dizzy, and disoriented: these are presumptive signs of cerebral concussion as determined via the Immediate Concussion Symptoms (ICS) Scale [8]. After the MVA, she realized that she had a bruise on her head: she hit her head on something in the impact, but her attention was either exclusively focused on other immediate issues at the time of the external head injury or she has amnesia for the brief time span in which her head was injured.

After becoming more clearly aware of her situation, our patient felt shocked, panicked, and worried. She felt unable to react. Our patient experienced pain and was briefly assessed in the emergency department of a hospital. Within the first 5 hours of the MVA, she suffered from headaches and pain in her neck, back, shoulders, arms, knees, feet, and pelvis. Over the next 2 to 3 weeks and until the present, she has suffered from headaches and pain in her neck, back, shoulders, arms, feet, and pelvis. With respect to cauda equina, her post-MVA symptoms include numbness in her left leg and in the "saddle area," severe urinary incontinence, instances of moderate loss of muscular control over her left leg, and severe tingling and some loss of feeling in her left leg. The responses of this patient to the Post-MVA Neurological Symptoms (PMNS) Scale [9] determined that her other post-MVA neurological symptoms included moderate bilateral hand tremor, moderately impaired balance, instances of a moderate loss of muscle control over her hands and arms, severe tingling, numbness, and loss of feeling in her hands, bouts of moderate tinnitus in the form of a high-pitched ringing or humming sound, moderate difficulty articulating words, moderate form of the syndrome of word finding difficulty, and a mild stutter.

Her responses to Rivermead scale of post-concussive symptoms[10] indicated the following residual current post-concussion symptoms: moderate dizziness, moderate nausea, moderate problems with blurred and double vision, moderate irritability, severe headaches, severe oversensitivity to bright lights and loud noise, severely impaired memory, severe problems with slow speed of thinking, severely impaired concentration, severe fatigue, severe restlessness, and severe impatience.

Unfortunately, this patient resides in a geographically too remote, isolated, and medically underserviced area of Canada and has been managed by her physician only with high dose of opiate based analgesics such as Percocet which partly concealed some of her diagnostically important symptoms. Her cauda equina syndrome has remained undiagnosed and medically unmanaged until the present.

4. Discussion and Conclusions

Pain is an important warning signal for the patient to avoid strenuous activities. If placed on opiate analgesics, the patient might reinjure while performing arduous physical chores. Furthermore, the opiate analgesics obscure important diagnostic cues and could delay the diagnosis and therapeutic management as in the present patient's case.

Hopefully, extensive scientific research would soon follow on the use of relatively inexpensive, non-addictive, non-euphoric, and neuro-protective very powerful anti-inflammatories such as cannabidiol (see Camposa's 2016 review[11]) in non-surgical management of prodromal or mild symptoms of the cauda equina syndrome. The disadvantage of cannabidiol is its very slow rate of gradually decreasing the pain levels in injured tissues (weeks or even months), but its advantage is that it does not obscure important warning signals such as pain when the patient attempts to engage in arduous physical activity: this helps to prevent the repeated reinjury that is common if the patient is managed via opiates. Furthermore, the neuroprotective properties of cannabidiol seem of much importance for the patient's recovery, in addition to the neurosurgical management, as needed.

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