CAUDA EQUINA SYNDRONE: ABNORMAL POST-SURGICAL PRESENTATION

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Abstract

Cauda equina syndrome is an acute neurological emergency requiring immediate surgery to prevent long-term neurological complications.[1] The incidence is 1 case per 33000 to 1 case per 100000 people.[2] The diagnosis can be made clinically and should be combined with neuroimaging. The deficits that are present affect the L2-coccygeal nerves and their respective functions.[1,3] What happens when a patient wakes up from surgery experiencing upper limb dysfunction? This is what occurred in the following case. A 63-year-old female presented with symptoms of cauda equina syndrome. Upon waking from her L3-L5 decompression with fusion and allograft surgery she was confused and experiencing deficits in her upper limbs.

Introduction

Cauda equina syndrome is a rare acute neurological condition with many etiologies. It effects the cauda equina nerve roots which originates below the conus medullaris. The conus medullaris typically ends at the L1 segment. "The roots then course down through the intraspinal canal, forming the cauda equina, until they exit at their respective neural foramina".[3] Compression of the cauda equina (L2 – coccygeal nerves) can cause acute back pain, radicular pain, bowel and bladder dysfunction, sexual dysfunction, loss of anal tone, lower limb weakness, and paresthesia in the perianal or saddle region.[1] Emergent surgical intervention is needed to prevent irreversible damage. In order to perform the correct surgery, the underlaying cause must be known. The most common cause is lumbar disk herniation. Lumbar central canal stenosis can also lead to cauda equina syndrome, however, this is more rare.[1,4–6] "The spinal canal is bounded anteriorly by the vertebral body and/or vertebral disc and the posterior longitudinal ligament, laterally by the pedicles and facet joints, and posteriorly by the lamina and ligamenta flava. Pathology involving these structures is frequently responsible for producing lumbar central canal

stenosis".[3] When cauda equina syndrome is caused by lumbar central canal stenosis, the classic symptoms must be present on history and physical exam. Additionally, neuroimaging such as an MRI should be used to demonstrate spinal canal narrowing.[1,3] However, there is uncertainty in making this diagnosis radiographically as there is no set criteria for what is considered pathologically narrow.[3] A PubMed search for articles that contained the terms "cauda equina syndrome" AND "spinal stenosis" yielded two articles. These articles were related to cauda equina syndrome presenting as a post-op complication. Additionally, searching the database MEDLINE yielded 100 articles. Four of these articles were relevant to this case. Two articles were case reports in which spinal stenosis was the preceding event that caused the cauda equina syndrome.[4,6] One article was a retrospective study which found an annual incidence of spinal stenosis in Sweden to be 50 per million in the study time period. Of the 163 cases studied one case developed cauda equina syndrome.[5] The fourth article was a literature review of cauda equina syndrome. This article proposed several different mechanisms for spinal stenosis causing cauda equina syndrome.[7]

Case Presentation

A 63-year-old female presents to a rural hospital via ambulance where she is noted to have symptoms of cauda equina syndrome. The patient fell getting out of bed that morning as she did not have use of her legs. After several more attempts at getting up and falling she determined something must be wrong. Patient denied any bowel or bladder incontinence or retention. Patient denied sensation of numbness but was experiencing symptoms of weakness. Patient is transferred to a tertiary center where she is offered emergency surgery. It is determined her symptoms are caused by cauda equina syndrome from spinal stenosis. A L3-L5 decompression with fusion and allograft surgery was performed. Upon awakening, patient noted

that she experienced new numbness in both arms and that her cognitive abilities had declined. Most notedly were memory, inattention, expressive aphasia, and inability to follow commands. Due to these symptoms the patient was diagnosed with post-op delirium and Korsakoff's syndrome. It was suggested that her symptoms could be due to cervical spine pathology, therefore an MRI was ordered. The MRI of cervical spine and brain revealed no notable abnormalities besides the presence of a thyroid nodule that was determined to be benign. Over a three-month period, the patient's mental status slowly improved to the point where she was found to be eligible for rehab. She was then transferred to a rehab hospital in her region. This is where I first encountered the patient.

History

This patient's presentation is complicated by an extensive past medical history. The patient had a previous fall with fracture to L1 five years prior. She experienced extensive pain and was prescribed opioids and nabilone. Patient had to work modified duties and decreased hours at her job as an educational assistant. Three years post injury, the patient went on disability due to lumbar central canal stenosis. In addition, the patient also has bilateral total knee replacements (15 and 12 years prior), gastric by-pass surgery fourteen years prior from which she lost over 100 pounds, well controlled hypertension, asthma, seasonal depression, 20 pack-year smoking history, and alcoholism of a six-year duration with an average of a bottle of wine consumed per day. Prior to the fall she was experiencing infected mouth ulcerations thought to be caused by her alcoholism and complicated by her depression. The diagnosis of Korsakoff's syndrome was made on the suspicion that both her gastric by-pass surgery and alcohol consumption contributed to the possible thiamine deficiency.[8] It was not expected that the patient

would improve when first diagnosed. However, her mental status improved enough to be a rehab candidate.

Current Status

Patient has significantly improved both physically and mentally over the two-month period in rehab. Patient was discharged from rural rehab center during my time there. Her mood improved significantly since transferring hospitals as she felt there was hope. Patient uses verbal cues to help cope with remaining memory deficit. Patient improved from wheelchair bound on admission to ambulation with a walker upon discharge. She can perform all ADL's independently. Patient has ongoing issues with proprioception and still experiences back pain. Patient states that arms feel weak and numb while her legs feel the sensation of pins and needles. She used to get spasms in her right leg but no longer experiences them. Patient has not been satisfied with her diagnosis and she feels there is still something the doctors are missing. Additionally, patient feels that she should never have gotten the decompression with fusion and allograft surgery. This is due to being under a time pressure to make the decision and due to the numerous issues since the surgery.

Laboratory Testing

In order to workup other possible causes for her symptoms, several tests were ordered. Since a nodule was noted on her thyroid TSH, free T3 and free T4 were ordered both immediately post-op and four months post-op. All tests were in the normal range. During her time in the rehab hospital bloodwork for vitamin B_{12} and vitamin D tested above the cutoff value. Electrolytes, urea, creatinine, and liver enzymes all tested within normal values. Patient

was noted to be mildly anemic with a hemoglobin of 101. Further investigations revealed an iron saturation of 10%. However, ferritin assay, iron, and TIBC were within the normal range.

Imaging

The MRI of L-spine on presentation to the tertiary center did not show significant nerve root compression.

Post-op MRI of cervical spine did not demonstrate any significant abnormalities that could explain the upper limb pathology. However, it did show moderate multilevel degenerative changes of the cervical spine.

Post-op MRI of brain did not demonstrate an acute intercranial abnormality.

A second brain MRI was ordered five months post-op to rule out chances of cerebellar stroke or other structural neurological deficits. It showed no changes from the MRI five months previous.

Discussion

This case was of particular value due to the abnormal presentation of upper limb disfunction following a L3-L5 decompression with fusion and allograft surgery. This presentation warranted further investigation but was hastily labeled a complication of alcohol misuse. Other explanations should have been explored.

Korsakoff's syndrome is difficult to diagnose. There is no consensus on criteria required to make a diagnosis.[8] It is a largely irreversible syndrome caused by severe thiamine deficiency caused by either alcohol misuse or nutrient deficiency. "It is characterized by an abnormal mental state in which episodic memory is affected out of all proportion to other cognitive

functions in an otherwise alert and responsive patient".[8] The literature also states that Korsakoff's syndrome will not improve over time. There is debate on whether an acute episode of Wernicke's encephalopathy is needed to proceed Korsakoff's syndrome.[8] Both of these statements would lead away from a diagnosis of Korsakoff's syndrome as the patient experienced significant improvement over four months and there was no documented case of Wernicke's encephalopathy.

The immediate post-op MRI of cervical spine was ordered as it was thought the upper limb dysfunction could be explained by cervical pathology and the confusion could be delirium. An MRI of the brain was also ordered as it might demonstrate the presence of a stroke to explain the upper limb dysfunction and cognitive change.

It was considered that the patient did not have cauda equina syndrome but instead had a missed case of Wernicke's encephalopathy. Wernicke's encephalopathy is often missed and it could help further explain the upper limb ataxia and confusion upon waking from surgery.[8,9] There is a clinical triad of symptoms used to diagnosis Wernicke's encephalopathy consisting of mental status change, ocular abnormalities, and ataxia. Not all or any of the symptoms must be present to make a diagnosis.[10] Due to not having access to the patients original file from the tertiary hospital it is unclear if she presented with these symptoms.

The incidence of cauda equina is low.[2] The most common etiology of cauda equina syndrome is lumbar disk herniation rather than spinal stenosis.[1] These facts would further suggest a diagnosis of Wernicke's encephalopathy and not cauda equina syndrome. Additionally, the MRI of the patients lumbar spine at time of presentation did not show any nerve compression in the cauda equina. The diagnosis of cauda equina syndrome was made clinically. More evidence in support of the diagnosis of Wernicke's encephalopathy is the presence of specific

periventricular white matter FLAIR hyperintensities on MRI. The radiologist determined these were most likely microangiopathic changes. However, "presence of bilateral symmetrical signal hyperintensities in the periventricular region of the third ventricle, periaqueductal area and mammillary bodies strongly supports clinical impressions of Wernicke's encephalopathy".[10] Therefore, this is a theory that warrants further investigation. However, when using PubMed to search for cases where Wernicke's encephalopathy was mistaken for cauda equina syndrome by using the search terms "Wernicke's encephalopathy" AND "cauda equina syndrome" it yielded no results. This leads this theory to being less likely. However, this case is complicated by the patient's extensive past medical history.

Recommendations

Due to Wernicke's encephalopathy being easily missed, clinicians need to be aware of and check for the triad of symptoms. High levels of suspicion are critical as there is a simple fix of intravenous thiamine.[8] It is important to rule out other etiologies of disease before making a final diagnosis of Korsakoff's syndrome due to alcohol misuse. Cauda equina syndrome requires a high index of suspicion due to the severe sequalae. Clinicians must be aware of the symptoms and any acute changes in status.[1] It is essential that cauda equina syndrome is diagnosed promptly, as 48 hours after symptom onset surgery outcomes are not optimal.[2] Complex presentations that can be given one overarching diagnosis to explain all symptoms is what medicine strives for. In this case the diagnosis of cauda equina syndrome did not explain the entire clinical picture. To compensate for the short comings of the original diagnosis Korsakoff's syndrome and post-op delirium were later diagnosed. While it is important to consider all diagnostic possibilities for a presentation, most often multiple diagnoses are not the best medical explanation. One resounding message from this case is that the patient is often their best

advocate. Since the patient was not satisfied with the diagnoses made, this prompted further investigation that might not have happened if the patient was afraid to speak up. Going forward this is a message that should be kept in mind with all patients.

Verbal consent was obtained from the patient on July 9, 2019

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