A 49-year-old patient presented with bilateral lower extremity pain that began one week prior to presentation but was exacerbated two days prior to presentation. The pain was described as an intense bilateral radicular pain in both lower extremities. The patient was originally diagnosed with degenerative disk disease one-year prior, following a sports injury, and reports intermittent radicular leg pain since. He had been prescribed a steroid taper by a physician over the past several days. On a long flight one day prior to presentation, he experienced significant difficulty standing up or walking secondary to extreme pain and weakness. The patient reported new onset urinary frequency with dribbling of urine over the past 24 hours but no episodes of overt incontinence. He also described subjective bilateral lower extremity paresthesias in L4 and L5 dermatomal distributions.

Clinical examination revealed normal sensation to light touch and pin prick in all dermatomal distributions, with intact perianal sensation. There was noticeable decreased strength (3/5) in the extensor hallucis longus and (4/5) tibialis anterior as well as hyporeflexic patellar reflexes bilaterally. His past medical history was significant only for asthma.

MRI revealed large disk protrusion at L4-L5 with severe canal and foraminal stenosis. The patient was taken to the operating room for emergent laminectomy and decompression of L4-5 within six hours of presentation to the emergency department. Two weeks post-operatively, there was an overall improvement in motor function, with both tibialis anterior and extensor hallucis longus demonstrating 4+/5 strength. The patient's urinary symptoms resolved postoperatively, as well.

INTRODUCTION
The spinal cord terminates at the level of the intervertebral disc between the first and second lumbar vertebrae, forming the conus medullaris, below which is the filum terminale and a bundle of nerve roots constituting the cauda equina. Cauda equina syndrome is comprised of complex neurological disorders manifesting in a wide variety of symptoms, such as back pain, unilateral or bilateral leg pain, paresthesias and weakness, perineum or saddle anesthesia, and rectal and/or urinary incontinence or dysfunction. Frequently, it occurs following a large lower lumbar disc herniation, prolapse, or sequestration. Less common causes are epidural haematoma infections, primary and metastatic neoplasms, trauma, post surgical, prolapse after manipulation, after chemonucleolysis, after spinal anaesthesia, and it has been reported in patients with ankylosing spondylitis and gunshot wounds.

EPIDEMIOLOGY
Cauda equina syndrome is rare (both atraumatically as well as traumatically) and variable, depending on the etiology of the syndrome, and is estimated to account for less than one in 2000 patients with severe low back pain. In another report, the prevalence among patients with low back pain is approximately four in 10,000. Males and females are equally affected; it can occur at any age but primarily in adults. The prevalence among the general population has been estimated between 1:33,000 and 1:100,000. The most common cause of cauda equina syndrome is herniation of a lumbar intervertebral disc, which is reported by approximately one to 10 percent of patients with herniated lumbar disks.

ANATOMY
In a detailed anatomical review of the conus medullaris in the adult human, Malas et al. colleagues found that the conus had a variable location between T-12 and L-2. In the cross-section of cauda equina, the lower sacral (S2-S5) and coccygeal roots were located in the dorsal aspect of the thecal sac, whereas the lumbar and first sacral roots exhibited...
an oblique, layered pattern as they ascended. The motor bundle was situated anteromedial to its respective sensory bundle within each layer; invaginations of arachnoid held the nerve roots in a fixed relationship to one another.

PATHOGENESIS

The pathophysiological mechanisms of cauda equina syndrome are not completely understood. It may result from any lesion to cauda equina nerve roots, such as direct mechanical compression, inflammation, and venous congestion or ischemia. The cauda equina nerve roots have no Schwann cells covered, and their microvascular systems have a region of relative hypovascularity.

Table 1 shows some of the causes of cauda equina syndrome.

CLINICAL PRESENTATION

There are no broadly accepted definite diagnostic criteria of cauda equina syndrome up to now. In some patients, the diagnosis of cauda equina syndrome will be obvious, while others are more obscure.

Sensory nerves are smaller and more sensitive than motor ones. So the first sign of the Cauda equina syndrome is often sensory, but this is not true for all patients. Dinning et al., in their series of 39 cases, observed patients with sphincteric symptoms who had no saddle area sensory loss. Furthermore, symptoms can be unilateral or bilateral, acute or chronic, sudden or gradual in onset. Urinary retention is one of the important predictors of Cauda equina compression.

If one or more of the following is present in a patient with low back pain or lower extremity radicular pain, the cauda equina syndrome is likely: bladder and/or bowel dysfunction, reduced sensation in the saddle area, or sexual dysfunction, with possible neurologic deficit in the lower limb (motor/sensory loss, reflex change).

Some investigators have tried to classify patients according to their symptoms. (See Table 2)

DIAGNOSIS

Only with careful history taking and physical exam of the patient can cauda equina syndrome be diagnosed early and therefore treated early to avoid preventable life-long disability. Bladder function and perianal sensation should be assessed, as they are extremely important for the diagnosis as well as the prognosis.

Plain radiography is often helpless in detecting the cause of cauda equina syndrome but might be useful in searching of destructive changes, disk-space narrowing, or spondylolysis. MRI and CT scan are the gold standard tests for cauda equina syndrome diagnosis. By comparing with the surgically confirmed abnormality, MRI may be slightly more sensitive than CT scan and CT myelography, though they have roughly equivalent sensitivities and specificities.

MRI also has the advantage of not using ionizing radiation or contrast injection and provides better resolution. Magnetic resonance imaging, therefore, should be the best initial procedure for patients with suspected cauda equina syndrome. Emergency MRI should be considered in all patients who present with new onset of urinary symptoms in the context of lumbar back pain or sciatica in order to avoid misdiagnosis or missed diagnosis of cauda equina syndrome.

As urinary retention is one of the important predictors of cauda equina compression, urinary retention of more than 500ml alone or in combination of with two or more specific clinical characteristics were the most important predictors of MRI confirmed cauda compressions.

TREATMENT

Cauda equina syndrome is an absolute indication for surgical decompression. There is no conservative care for this condition. Depending on the etiology and location, decompression may consist of posterior, anterior, or posterior lateral decompression. Discectomy and wide evacuations are done for intervertebral disc herniations and hematoma/abscess, respectively.
Fracture fragments and tumor excision would be done in those cases where compression exists from these conditions. With regard to the timing of surgery, some controversy does exist. Kostuik, et al.\(^\text{32}\) reported on 31 patients with CES and their treatment. The patients were divided into acute and chronic onset groups. Their conclusion was that decompression did not have to be done within six hours to obtain a satisfactory result, although the authors did recommend early intervention once the condition was diagnosed.

Delamarter\(^\text{33}\) studied the recovery of neurologic function in an animal study in which constriction of the cauda equina was performed on 30 canines. Timing of the release of the constriction varied from immediate release to one hour, six hours, twenty-four hours, and one week, respectively. The canines with immediate decompression generally recovered neurologic function within two to five days. The canines receiving one hour and six hour decompression recovered neurologic function within five to seven days, while those receiving twenty-four hour decompression remained paraparetic for five to seven days. There was no statistical difference in recovery of somatosensory evoked potentials among the groups. They concluded that was that there were no significant differences in neurologic recovery of the dogs with respect to immediate, one hour, six hours, 24 hours or one week.

Shapiro,\(^\text{34}\) on the other hand, performed a retrospective analysis of 44 patients with cauda equina syndrome secondary to lumbar disc herniation. All patients had bowel and bladder symptoms. In 20 patients, diagnosis and surgery were performed within 48 hours of the cauda equina syndrome onset, including 18 patients (90 percent) who underwent surgery within 24 hours. In 24 patients, surgery was performed more than 48 hours after onset. According to X2 analysis, a greater chance of persistent bladder/sphincter problem, persistent severe motor deficit, persistent pain and sexual dysfunction existed with delayed surgery. Data strongly supported the management of cauda equina syndrome from lumbar disc herniation as a diagnostic and surgical emergency.

A meta-analysis by Ahn, et al.\(^\text{35}\) studied the correlation of the timing of decompression and surgical outcomes of CES secondary to lumbar disc herniation. Forty two publications met the inclusion criteria. Three hundred twenty two patient outcomes were analyzed. There was significant advantage of performing decompression for CES before 48 hours compared to after 48 hours. Kohles, et al.\(^\text{36}\) reviewed the meta-analysis by Ahn, et al.

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### TABLE 1: CAUSES OF CAUDA EQUINA SYNDROME

| Non-Neoplastic Compression                      | • Herniated Lumbosacral Discs  
|                                                | • Spinal Stenosis              |
| Spinal neoplasm and compression of cauda equina | • Schwannomas                  
|                                                | • Lipoma and teratoma          
|                                                | • Arteriovenous Malformation    
|                                                | • Metastasis to Leptomeningies  |
| Non-Compressive etiologies of cauda equina syndrome | • Ischemic Insults  
|                                                | • Inflammatory conditions of cauda equina  
|                                                | • Spinal Arachnoiditis and infectious etiologies |

### TABLE 2: SUGGESTED CAUDA EQUINA SYNDROME PATIENTS CLASSIFICATION ACCORDING TO THEIR SYMPTOMS

| Tendon et al.\(^\text{23}\) Classification: | Group 1: Symptoms arose suddenly without previous History of backache.  
| Shephard et al.\(^\text{24}\) Classification: (Secondary to Central Disc lesion) | Group 2: Acute onset of bladder dysfunction following a long history of low back pain.  
| Gleave et al.\(^\text{25}\) Classification: | Group 3: Symptoms arose gradually from background of chronic low back pain and Sciatica.  
|                                             | Type 1: There were abrupt, more severe symptoms and signs and a slightly poorer prognosis after decompression; especially for the return of the bladder function.  
|                                             | Type 2: Slower onset, characterized by prior symptoms for varying time intervals before the more gradual onset.  
|                                             | • Incomplete (Impairment of bladder control): patients exhibited a spectrum of urinary difficulties but without urinary retention.  
|                                             | • Complete (Retention/loss of bladder control): patients had painless urinary retention with overflow incontinence.  

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stating the paper did not stress the importance of early decompression especially with reference to progressive nerve damage. They advocated early decompression (within 48 hours) and stated that poor outcomes increase proportionally with time.

CONCLUSION
Cauda equina syndrome can have a variable presentation, but the hallmarks are urinary and bowel dysfunction in association with sensory abnormalities. Saddle anesthesia and motor weakness may or may not be present but should be evaluated during the examination. Once the diagnosis is confirmed with the appropriate radiological studies then urgent surgery is recommended, as in our patient. The literature does confirm that best results occur with urgent decompression of the spine.

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