A Case Study on Cauda Equina Syndrome

Kilda.S¹ and Vasanth Kumar.P²

¹Dept of Medical Surgical Nursing, Coimbatore-641004, T.N, India,
   Email: kildakiruba1@gmail.com
²General Medicine, PSG Hospital, Coimbatore-641004, T.N, India
   Email: sparrowvasanth.cmc@gmail.com
http://doi.org/10.37794/IJSRR.2019.8406

ABSTRACT

A 23 year old lady got admitted in the emergency department with a history of severe back pain, she also felt numbness in her lower limbs, associated with retention of urine and inability to walk. Past history revealed tuberculosis of spine and on anti tuberculosis therapy. After thorough physical examination and from her history she was diagnosed as early stage of cauda equina syndrome. She was treated with corticosteroid and underwent physical therapy. No surgical intervention were done. After the treatment, there were reduction of pain and was able to perform her activities of daily living. This report shows the nature of cauda equina syndrome and it's impact on her functioning. It also explains the effectiveness of early diagnosis and treatment in better outcomes.

KEYWORDS: Cauda Equina Syndrome

* Corresponding author

Kilda S
M.Sc Nursing, PSG Hospital
Coimbatore- 641004, T.N, INDIA.
Email:kildakiruba1@gmail.com, Mob No-9843893830
INTRODUCTION

Cauda equina syndrome (CES) is a rare but serious neurological condition affecting the bundle of nerve roots at the lower end of the spinal cord. CES occurs as a consequence of compression of the cauda equina and can be caused by a number of pathologies. The prevalence among the general population has been estimated between 1:100,000 and 1:33,000. The most common cause of CES is herniation of a lumbar intervertebral disc and accounts for 2% of all herniated lumbar discs. It commonly affects the discs at the L4/5 and L5/S1 level. However, disc prolapse at any lumbar level can cause CES. Patients may be predisposed to CES if they have a congenitally narrow spinal canal or have acquired spinal stenosis. The prevalence among patients with low back pain is approximately four in 10,000.

The most common cause of CES is a prolapse of a lumbar disc but other conditions such as metastatic spinal cord compression can also cause CES. Cauda equina syndrome is caused by significant narrowing of the spinal canal that compresses the nerve roots below the level of the spinal cord. Numerous causes of cauda equina syndrome have been reported, including traumatic injury, disk herniation, spinal stenosis, spinal tumors (neoplasms), such as metastatic tumors, meningiomas, schwannomas, and ependymomas, inflammatory conditions, infectious conditions, and accidental causes by medical intervention (iatrogenic causes). Tuberculosis of spine can lead to neurologic abnormalities in 50% of cases (paraplegia, paresis, impaired sensation, nerve root pain, or cauda equina syndrome). Patients with spinal tuberculosis, 54/133 (40.6%) had neurologic deficits at admission and 17.3% presented with cauda equina syndrome. Compression of the Cauda equina is most often due to neoplasm such as ependymoma or neurofibroma, but nerves may be compressed by an associated lipoma in cases of spina bifida occulta, by a constructing fibrous band or by chronic arachnoiditis. Perineurial cysts on the posterior sacral roots are often asymptomatic but may cause sciatic pain.

The clinical presentation is variable, depending upon the site and extent of the source of compression. In many cases pain is the earliest symptoms of cauda equina tumor. Patient experiences dull aching pain in the lumbar or sacral region, which is exacerbated by jerky movements, coughing, or sneezing. Less often it is referred to one or both lower limbs in the distribution of the lower spinal nerves, or it may be referred to the bladder, rectum or testis. Motor symptoms consist of weakness and wasting and most often there is paralysis of the muscles below the knee and of the hamstrings and gluteal. In such cases, the ankle jerks are diminished or absent, and the plantar responses may be absent; but the knee jerks are
often preserved. The distribution of sensory loss also depends on the spinal nerves involved. Compression of the lower sacral roots gives a characteristic saddle-shaped area of anesthesia and analgesia, extending over the perineum, buttocks, and back of the thighs. Compression of the upper sacral and fifth lumbar nerves produces an area of sensory loss over the foot and over the posterior or outer aspect of the leg.

When the lower part of the sacral segments is involved, the external genitalias are anaesthetic, and the patient may be unaware of the passage of the catheter, some bladder sensation usually remains, so that patient is aware when it distends. Disturbance of sphincter control occurs in the later stage. Compression of the third and fourth ventral and dorsal sacral roots interrupts the reflex of evacuation of the bladder and rectum. This result is retention of urine and erectile dysfunction occurs in the male. When the lowest sacral nerves are compressed, the anal and bulbocavernous reflex are lost.

Superficial changes may occur in the lower limbs, which are often cold and cyanosed, and tend to become edematous. Slight injuries in analgesic areas can lead to sores which are slow to heal.

**CASE REPORT**

A 23-year-old female presented to the emergency department with history of severe back pain, numbness in lower limbs, urinary retention, and inability to walk. The patient had a past history of Tuberculosis spine, disseminated tuberculosis, miliary tuberculosis, tuberculosis vasculitis and tuberculosis arachnoiditis for which she was on anti tuberculosis Therapy. On physical examination straight leg raising test (SLRT) of 20 degrees on right side and 40 degrees on left side. In addition she has Paresthesia and numbness in perianal area was also present with decreased sphincter tone with a muscle power of 3/5 on manual testing. Regarding the range of motion test flexion, extension and hyperextension were absent in both lower limbs. She was not able walk due to weakness and numbness. A diagnosis of early Cauda Equina Syndrome was made.

Her vitals were normal except her BP it was 90/60mm Hg and Saturation without O2 was 90% on room air.. She had neck muscles weakness, pooling of oral secretion and poor cough reflex present. Due to persistent hypoxia she was shifted to MICU on 10/09/2018 and put her on NIV for one day. Blood investigations showed hyponatremia (SIADH) which was corrected accordingly.

Lab finding revealed normal RBCs count (4.97×10.6/ul), Hb 13.9g/dl; Hematocrit 41.9%, WBC 6.1×10.3/UL and she had elevated ESR (1 hr) 34mm. Sr. Urea 16mg/dl, Sr. Creatinine 0.27 mg/dl.
Serum electrolytes such as Sodium 133 mEq/L, Potassium 4.28 mEq/L, Chloride 92 mEq/L, Bicarbonate 24.9 mEq/L and Ionised Calcium 1.035 mmol/L. Liver Function Test revealed normal finding total bilirubin 0.7 mg/dl, direct 0.2 mg/dl and indirect: 0.5 mg/dl; SGPT (ALT) 12 U/L, SGOT (AST) 23 U/L, alkaline Phosphatase 43 U/L. Glucose Random was 84 mg/dl.

A Pulmonology consultation was obtained and medications modified accordingly. Infectious disease specialist opinion was taken. Her LFT, RFT and CBC repeated before discharge showed normal results except mild anemia and raised GGT. She was advised to continue the medication regularly and to take adequate physiotherapy. Patient advised do to RFT and LFT after 1 week and report immediately if patient develops jaundice, profuse vomiting and breathing difficulty.

The patient A was managed with Tab. Livogen 1500 mg (OD), Tab. Dexa 8 mg (BD), Tab. Pantocid 40 mg, (OD), Syp. Racenerve 10 ml (BD) and Tab. Ethambutol 800 mg (OD). In addition, she was reviewed by a physiotherapist who initiated exercise to improve mobility and activities of daily living. She was educated on importance of fiber, protein rich diet, fluid and medications.

**DISCUSSION**

Although Cauda equine syndrome is not cause due to a direct spinal cord injury it still requires emergency neurological interventions. The clinical presentation is variable, depending upon the site and extent of the source of compression. In many cases pain is the earliest symptoms of cauda equine tumor. Patient experiences dull aching pain in the lumbar or sacral region, which is exacerbated by jerky movements, coughing, or sneezing. Less often it is referred to one or both lower limbs in the distribution of the lower spinal nerves, or it may be referred to the bladder, rectum or testis.

Motor symptoms consist of weakness and wasting and most often there is paralysis of the muscles, the ankle jerks are diminished or absent, the distribution of sensory loss also depends on the spinal nerves involved. Compression of the nerve roots interrupts the evacuation of the bladder and rectum. This result is retention of urine and erectile dysfunction.

Early diagnosis made after thorough physical examination and a complete neurological assessment to determine sensory loss, muscle weakness and reflex change. In case of suspected CES patient should undergo MRI/CT scan to confirm the diagnosis. Appropriate analgesia should be provided for a patient with cauda equina syndrome to provide adequate and rapid pain relief. Anti-
inflammatory medication may prevent worsening of injury, Methylprednisolone should be administered or Dexamethasone is commonly given intravenously at doses of 4 to 100 mg.

Anticoagulants provide prophylaxis against thrombotic complications. Use of antispasticity medications to reduce muscle tone is encouraged. Bisphosphonates may be beneficial in patients with bony lesions. Phosphodiesterase type 5 inhibitors treat erectile dysfunction (ED). The treatment must be started within 8 hours of injury. Administration of ganglioside GM1 sodium salt beginning within 72 hours of injury may be beneficial; the dose is 100 mg IV qd for 18-32 days. Emergency Surgical decompression is mandatory in acute compression of the conus medullaris or cauda equina. The goal is to relieve the pressure on the nerves of the cauda equina by removing the compressing agent and increasing the space in the spinal canal. It is an surgical emergency, it should be done within 48 hours after the onset of symptoms, and preferably performed within 6 h of injury. If herniated disk is the cause of cauda equina syndrome, a laminotomy or laminectomy to allow for decompression of the canal is recommended, followed by gentle retraction and discectomy.

Physiotherapy plays an vital role after the surgical intervention. It helps the patient to perform range of motion and strengthening exercises, sitting balance, transfer training, and tilt table as tolerated. Tilt table should start at 15 degrees, progressing by 10 degrees every 15 minutes up to about 80 degrees with the necessary precautions. Other therapies such as, wheelchair propulsion training, standing table exercises, functional electrical stimulation for increased muscle tone, lower extremity orthotic to aid balance and walking, ambulation exercises.

**PROGNOSIS**

The outcome of the treatment in Cauda Equina syndrome is based on the presentation of symptoms, if the patient present without problem in walking then they remains ambulatory and they continue to perform their ADLs after the treatment. If they were unable to walk during the admission after treatment they have to use assistive devices for ambulation. In case of urinary retention they have to continue to use catheter after their recovery. Patient who underwent surgery the recovery time ranges from months to years.
CONCLUSION

Cauda Equine Syndrome affects the individual’s life in all the areas, early identification of the problem and early intervention of low back pain after thorough evaluation possibly helps the patient to overcome the complication and facilitates recovery.

REFERENCES

8. Brain’s Diseases of the Nervous system, Eleventh edition, Michael Donaghy, 2001; 635