Clinical Management of Spinal Cord Injury in a Horse

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Abstract
One gelding with traumatic history was presented for treatment. The horse was systematically screened under routine neurological protocol. Cranial nerve test and spinal reflexes tests were conducted. Radiograph was also made. The lesion was diagnosed as thoracolumbar spinal cord injury. The animal was treated for the spinal cord injury using Methy prednisolone sodium succinate epidurally within 12 hour interval and Streptopenicillin for 7 days.

Keywords: Dog sitting posture; spinal cord injury, thoracolumbar

Introduction
Trauma to the central nervous system is the most common causes of neurological diseases in horses. Among central nervous system diseases, trauma to the spinal cord is more prevalent than brain injury. Around 77 percent of horses presented for neurological examination were diagnosed with spinal cord injury. (Feige et al., 2000). Trauma to the spinal cord occurs due to incidents such as collision with an immovable object or falling down. Injury to the vertebral column or spinal cord can occur at all sites but cervical spinal affections are commonly reported (Reed, 1994). There are only few reports available about the thoracolumbar spinal affections in horses. Here we are presenting thoracolumbar spinal cord injury in a horse induced by trauma and its medical management.

History
A equine with history of trauma was screened for spinal injury. Examination included the life threatening conditions viz. airway obstruction, hemorrhage, cardiovascular collapse and pneumothorax from fractures in the rib cage. In addition further diagnosis of major long bone fractures was also carried out. Then a systemic neurological examination was carried out to localize the site of injury.

Neurological examination
The protocol for the neurological examination of horses (Blythe, 1987; Matthews and Andrews, 1990) was followed. Among 12 cases, one case (gelding, 350 kg weight) had the typical T3-L3 spinal segment affections symptom (dog sitting posture – Fig. 1) is described here. The lesion was localized by using the regular neurological protocol. The following were the signs helped to localize the T3-L3 spinal cord segment.

The behavior or mentation of the animal was active and alert and there was no head or neck tilt observed. Diffuse sweating was observed.

Posture assessment
The animal was not cooperative for fore limb lifting and mild base wide stance in the hind limbs was observed

Gait assessment
(Grade 0 - No deficits, Grade 1 - Just detected at normal gait, but worsened by backing, turning, loin pressure or neck extension, Grade 2 - Deficit easily detected at normal gait and exaggerated by backing, turning, swaying, loin pressure and neck extension, Grade 3 - Deficit very prominent on walking with a tendency to buckle or fall with backing, turning, loin pressure and neck

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extension, Grade 4 - Stumbling, tripping and falling spontaneously; Grade 5 - Horse recumbent)

The horse was graded 3 in the gait assessment with the deficit of paresis in both the hind limbs.

**Cranial nerve tests**
The menace test, pupillary light response test, facial sensation, facial symmetry, palpebral reflex, nystagmus, swallowing reflex and tongue tone test were performed. But no abnormalities could be detected.

**Spinal reflexes**
The biceps and triceps reflex were normal in both the limbs. The right patellar reflex was exaggerated but left patellar reflex was normal. Proprioceptive reflex of both the fore limbs were normal but in both the hind limbs diminished proprioceptive responses were observed. Clinical signs that helped to differentiate Upper and Lower Motor Neurons affections are LMN signs - diminished reflex responses, decreased muscle tone, profound muscle atrophy and UMN signs – exaggerated reflex responses, increased muscle tone, minimal muscle atrophy. Bladder evacuation, anal reflex and tail tone were normal. After the neurological examination, spinal cord lesion was localized by using UMN and LMN signs (Oliver et al., 1978) as thoracolumbar segment. (Table 1)

**Table 1:** Spinal cord lesion localization by UMN and LMN signs

<table>
<thead>
<tr>
<th>Spinal segments</th>
<th>Fore limb signs</th>
<th>Hind limb signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1-C5 cervical</td>
<td>Rostral</td>
<td>UMN</td>
</tr>
<tr>
<td>C6-T2 cervical</td>
<td>Cervical enlargement</td>
<td>LMN</td>
</tr>
<tr>
<td>T3-L3 thoracolumbar</td>
<td>Normal</td>
<td>UMN</td>
</tr>
<tr>
<td>L4-S3 lumbar sacral</td>
<td>Normal/ UMN/LMN</td>
<td>LMN</td>
</tr>
</tbody>
</table>

**Blood Parameters**
Blood collection was done before and after the treatment at weekly interval and the hematobiochemical parameters like hemoglobin, total erythrocyte count, packed cell volume, leucocyte count and total serum protein, BUN, creatinine, AST, creatinine kinase, LDH and ALP were recorded. Clinical parameters like heart rate and respiratory rate were also recorded at different intervals. (Table 2).

**Table 2:** Blood biochemical parameters and clinical parameters of the horse before and after treatment

<table>
<thead>
<tr>
<th>Blood Parameters</th>
<th>Before treatment</th>
<th>After 7 days of treatment</th>
<th>After one month treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate /min</td>
<td>58</td>
<td>53</td>
<td>51</td>
</tr>
<tr>
<td>Respiratory rate/min</td>
<td>21</td>
<td>17</td>
<td>15</td>
</tr>
<tr>
<td>Hemoglobin (mg/dL)</td>
<td>10.33</td>
<td>11.01</td>
<td>11.37</td>
</tr>
<tr>
<td>Total Erythrocyte count 10⁶/cmm</td>
<td>4.64</td>
<td>4.82</td>
<td>4.90</td>
</tr>
<tr>
<td>Packed cell volume</td>
<td>33</td>
<td>33</td>
<td>34</td>
</tr>
<tr>
<td>Total leukocyte count (10⁹/cmm)</td>
<td>8483</td>
<td>7716</td>
<td>5800</td>
</tr>
<tr>
<td>Total serum protein(g/dL)</td>
<td>7.43</td>
<td>6.75</td>
<td>6.50</td>
</tr>
<tr>
<td>BUN mg/dL</td>
<td>23.4</td>
<td>24.1</td>
<td>21.3</td>
</tr>
<tr>
<td>Creatinine mg/dL</td>
<td>2.4</td>
<td>2.3</td>
<td>2.6</td>
</tr>
<tr>
<td>AST U/L</td>
<td>44.7</td>
<td>24.3</td>
<td>22.6</td>
</tr>
<tr>
<td>Creatinine Kinase U/L</td>
<td>40.1</td>
<td>22.2</td>
<td>23.5</td>
</tr>
<tr>
<td>LDH U/L</td>
<td>524.6</td>
<td>521.6</td>
<td>531.6</td>
</tr>
<tr>
<td>ALP U/L</td>
<td>512.4</td>
<td>546.6</td>
<td>540.1</td>
</tr>
</tbody>
</table>

**Diagnosis**
The important clinical signs to confirm the T3-L3 segment localization were rear limb proprioceptive deficits, rear limb weakness and UMN signs, spasticity of pelvic limbs. The affected
animal showed the typical symptom of thoracolumbar affection, the dog sitting posture was also confirmed the diagnosis. The blood biochemical parameter observations were also indicating the trauma before treatment and their values came to normal after the treatment. Cerebrospinal fluid is one of the best indicators of spinal affections. But the owner was not willing to perform CSF collection. Plain radiograph of thoracolumbar region were made but they were of not interpretable quality.

Treatment
The horse was given complete stable rest for two months. It was treated with Methyl prednisolone sodium succinate 1gram (Solu-medrol®) epidurally within 12 hours after the trauma and apart from regular antibiotic coverage (Inj. Streptopenicillin 5gm i.m for 7 days) was carried out. First three days cold application applied at the thoracolumbar area and from fourth day onwards hot fomentation was applied for further two days. Ultrasound massage was done for a period of 15 days in the thoracolumbar region from the first day of treatment.

The animal showed mild improvement in the weight bearing on 7th day in both the hind limbs and proprioceptive reflex was still not normal. No deficit was observed when the animal was standing. But deficits were identifiable when the animal made to trot. Condition improved on every week observations. On day 30th complete weight bearing was observed in both the hind limbs and proprioceptive reflex became almost normal and there was deficit observable neither on posture nor on gait.

Discussion
As soon as animal comes for treatment met with trauma should be ruled out shock. As the shock occurs, animal’s first response is to hold its breath, as this occurs a signal is sent from the heart to the adrenal glands to release large doses of adrenaline to stimulate the cells in the body to fight or flight response. This response activates an increased respiratory rate, blood shunting from the digestive tract to the muscles, dilated pupils, sharpened awareness, quickened response of nerve impulses, reduced pain perception and the animal would be in exited condition and the neurological symptoms might not be marked. So, before starting treatment we have to stabilize the animal by giving fluids and or other analgesics if warranted then proceed to the next step.

Many Veterinarians feel that evaluation of systematic neurological examination is a complicated and difficult procedure. But most of the time diagnosis of the spinal cord disease is a challenging one without the systematic neurological approach in Indian conditions. Because we are not equipped with x-ray machines capable of making radiographs of vertebral columns in large animals in an interpretable manner. Moreover advanced diagnostic aids like CT scan and MRI scan are not available for the veterinary use.

In horses, lesions in the C₁-T₃ region are common and result in various degrees of tetraparesis to recumbency. Sacral cord damage can result in fecal and urinary incontinence, loss of use of tail and anus and deficit of pelvic limb function. Saccrococcygeal spinal cord injury can produce hypalgesia, hypotonia and hyporeflexia of the perineum, tail and anus or total analgesia and paralysis of those structures. Thoracolumbar spinal cord injury can result in paraparesis to recumbancy and the horses may dog-sit (Reed, 1994). Observations of Reed (1994) correlates with our case study. Diffuse sweating observed in our case might be due to result of loss of supraspinal input to the preganglionic cell bodies of the sympathetic system in the thoracolumbar intermediate grey.

Blunt injuries to the spinal cord occur under various conditions, including flexion, extension, axial load, rotation and distraction. In this primary injury blood vessels are broken, axons are disrupted and neuron and glial cell membranes are damaged. The initial hemorrhage, edema and hypo perfusion of the grey matter extends centripetally within minutes to hours and results in central necrosis, white matter edema and eventually demyelination of axons through secondary injury process (Kwon et al., 2004; Tator
and Fehlings, 1991). The symptoms observed in our case might have occurred due to the above pathophysiology.

The blood biochemical values were suggestive of muscle related injury. The elevated AST and creatine kinase value obtained is suggestive of this, which returned to normal values upon treatment. This is similar to those observed in human beings (Bloom et al., 1989). The elevated values of ALP may be correlated with bone remodeling during treatment (Rohde et al., 2000).

In horses, corticosteroid is the most commonly used drug for Central Nervous system affections. But in our study we didn’t use corticosteroids since the complication of laminitis on usage of steriods. The next choice to counteract the spinal inflammation is flunixin and phenylbutazone. Both the flunixin meglumine and phenylbutazone act by inhibiting cyclooxygenase which converts arachidonic acid to inflammatory mediators. Both the drugs help in maintaining normal rectal temperature. But until now, only Methyl Prednisolone Sodium Succinate (MPSS) was shown to be efficacious in both animals and humans in the treatment of spinal cord injury (Bracken, et al., 1997). Action of MPSS is inhibition of lipid peroxidation, eiconosanoid formation, maintenance of tissue blood flow, elimination of intracellular calcium accumulation and improved neuronal exitability and synaptic transmission. So, we were using MPSS through epidural route and the results were encouraging. Our study once again proved that MPSS was good in spinal cord injuries when used immediately after trauma.

References


