Current Strategies for Management of Intervertebral Disc Disease.

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Objectives

• Pathophysiology/terminology
• Where the confusion about IVDD comes from
• Diagnostics
• Treatment options
• Prognosis
Pathophysiology/Terminology

• Type of Intervertebral Disc Disease
  ▪ Hansen Type I
  ▪ Hansen Type II
  ▪ Hansen Type III

• Myelopathy – spinal cord dysfunction

• Spinal Hyperesthesia – misused, but accepted
  ▪ abnormal pain on spinal palpation.
  ▪ Synonym - hyperpathia
IVDD

Hoerlein 447
Pathophysiology/Terminology

- Upper motor neuron deficits – spastic, normo- to hyperreflexive
- Lower motor neuron deficits – Decreased tone, normo- to hyporeflexive.
- Nociception – conscious perception of painful stimulus
- Withdrawal Reflex – Reflexive flexion of limb with noxious stimulus to toe
Spinal Reflex Arc

- Spinal cord segments L4, L5, and L6
- Dorsal root
- Grey matter
- Ventral root
- Alpha efferent
- 1A afferent
- Muscle spindle
- Gamma efferent
- Quadriceps muscle

Platt 21
Pathophysiology/Terminology

• Superficial pain perception
  ▪ Conscious response to mildly noxious stimulus
    • Light pinch, needle prick

• Deep pain perception
  ▪ Conscious response to bone crushing pain.

• Paresis – inability to move body part with appropriate strength
Pathophysiology/Terminology

• **Plegia** – paralysis – inability to move body part
• **Ataxia** – impaired coordination of movement
  ▪ Proprioceptive
  ▪ Vestibular
  ▪ Cerebellar
• **Proprioception** – knowledge of where body parts are in space w/o having to look at them.
Why is IVDD so confusing?

• Impossible to find all the information in one place
• Numerous persistent incorrect anecdotal practices - e.g.:
  ▪ Surgery not needed unless paralyzed
  ▪ Medical management should be tried/failed before surgery considered
  ▪ IVDD is not a systemic disease
  ▪ Length of medical management time is arbitrary
References – This is a partial list!

• Hoerlein, BF. Canine Neurology: Diagnosis and Treatment, 3rd Ed. W. B. Saunders Company. Philadelphia. 1978
Hansen Type I IVDD

- Herniation, rupture, extrusion of nucleus pulposus through a tear in the annulus pulposus
  - Abnormal/unhealthy nucleus pulposus material, unable to withstand normal forces
  - Mineralized nucleus pulposus
    - chondroid degeneration
Hansen Type I IVDD

• Pain and paresis/plegia caused by:
  ▪ compression of spinal cord and nerve roots
  ▪ Bruising type injury to spinal cord
  ▪ Pain from stretching/tearing of disc annulus and dorsal longitudinal ligament
Hansen Type III IVDD

- Catch all phrase
- Rupture of IVDD capsule under pressure (traumatic) with explosive extrusion of nucleus pulposus.
- Synonyms:
  - High Velocity Low Volume Extrusion, Missile Disc, Acute Non-compressive Nucleus Pulposus Extrusion (ANNPE),
  - Splatter disc (overlaps with Type I), Liquid Disc
Type I and III IVDD Signalment

- Peracute to gradual (days) onset
- Spinal hyperesthesia
- Abnormal posturing
- Muscle spasms
- Paresis/paralysis
- +/- abnormal reflexes
- +/- decreased or absent nociception
Type I and III IVDD Signalment

• Young (4-6y) chondrodystrophic dog breeds, and middle aged (6-8) non-chondrodystrophic.
  ▪ In young dachshunds – association between number of mineralized discs on radiographs and risk for future Type I IVDD
• Young to middle aged cats
• Type III associated with exercise/activity
  ▪ Similar presentation to FCE and/or spinal trauma
Neurolocalization

- UMN signs generally carry a better prognosis than LMN signs.
  - LMN signs - dysfunction of spinal cord intumescence (neuron cell bodies for the LMNs reside).
    - Damage to cell body results in permanent loss of that neuron.
  - UMN signs – axon damage/dysfunction.
    - Spinal cord/nerve axons have much greater ability to regenerate/resist injury.
- Hyperesthesia – if present, site of pain usually indicates location of IVDD.
- Cutaneous Trunci reflex – 1-2 segments caudal to site of injury
Schiff-Sherrington Posture

- Acute thoracolumbar junction spinal cord injury
  - Loss of upper motor neuron inhibition to forelimbs → Increased extensor tone in forelimbs
    - Forelimb proprioception, voluntary motor and reflexes normal to increased
  - Mistaken for Opisthhotonos
    - SSP – no central vestibular or RAAS signs.
  - Does not give information about prognosis.
Nerve Root Signature

Sharp 93
Myelomalacia

• “Spinal cord” – “Softening”
  ▪ Ischemic death of the spinal cord
  ▪ Can be focal, but often progresses to the “ascending-descending” form.
  ▪ Fatal due to progression cranially to the areas of the spinal cord controlling respiratory muscles

• Occurs in 10% of patients with acute paraplegia and loss of nociception
  ▪ May not manifest for up to 7-10 days (usually under 5)
  ▪ No treatment, including surgery, shown to prevent.
Myelomalacia

• Clinical signs
  ▪ Ascending cutaneous trunci reflex cutoff
    • This reflex should not decrease further after 24hrs post-op or post-disc extrusion.
    • Track daily by marking patient’s back with permanent marker
  ▪ Flaccid abdomen and pelvic limbs
  ▪ Patient appears restless/uncomfortable
  ▪ May be hyper- or hypothermic
  ▪ Progressive respiratory paralysis
Hansen Type II IVDD

- Thickening and protrusion of annulus fibrosus
- Fibrous metaplasia of nucleus pulposus
- May be associated with spondylosis deformans (ankylosing).
- Paresis caused by compression of spinal cord and/or nerve roots.
  - May result in spinal cord gliosis and nerve atrophy
- Pain caused by nerve root compression
Type II IVDD Signalment

- Older, generally large breed dogs or older cats
- Chronic slowly progressive paresis +/- spinal pain
  - Often reluctant to jump or use stairs
  - “getting old,” “slowing down.”
- Can acutely worsen
- +/- nerve root signature or paresthesia
Differential Diagnoses

• Other causes of pain and/or myelopathy
  ▪ FCE, spinal fracture, discospondylitis, neoplasia, myelitis (infectious/auto-immune), Deg. Myel., COMS.

• Other causes of LMN weakness
  ▪ Neuropathy, myopathy, junctionopathy (MG), electrolyte disturbances, diabetes mellitus, plexus avulsion.

• General causes of weakness
  ▪ Hypotension, hypoglycemia, hemoabdomen, etc.

• Orthopedic disease/joint pain
  ▪ Bilateral CCL disease, hip dysplasia, polyarthropathy
Diagnostics – Physical Exam

• General PE
  ▪ Back pain and abdominal pain can be hard to differentiate
  ▪ Particular attention to causes of weakness
    • Thorough auscultation, palpate pulses, ballotte abdomen, check MM color.
  ▪ Thorough orthopedic exam
Diagnostics - Neurological Examination

• Importance of performing neuro exam on normal patients.
  ▪ Make sure patient adequately supported to differentiate weakness from CP deficit.
• Should be mentally appropriate
• Cranial nerves – Possible Horner’s Syndrome if cervical/high thoracic. Otherwise normal.
• Nociception-if limbs move voluntarily, pain sensation should be present – no need to aggressively test.
  ▪ Reflex does not equal nociception!
• Remember tail/perineum!
Diagnostics- Neurological Examination

• Spinal palpation, neck range of motion
  ▪ If suspect animal is painful, do this step last!
  ▪ Neck pain and partial seizures can look similar
  ▪ Neck pain dogs will hunch their T/L spine too.

• Voluntary Motor assessment

• Assess patients in quiet room
  ▪ On the floor
  ▪ Good footing

Olby Fig.7-2, 3rd Ed
Diagnostics – Minimum Database

• Complete blood count (with manual diff)
• Chemistry profile (with CPK)
• Urinalysis (with sediment)
• +/- Tick testing
• Aspergillus antigen German Shepherds
• DM genetic test – Boxers, GSD, Corgi
• +/- Neoplasia screening if over 8yrs (3-view CXR/AUS)
• Viral testing for cats.
• ECG/BP if renal dz, arrhythmia, poor pulses, etc.
Diagnostics - Imaging

- Radiographs
  - Low yield for IVDD
  - Provides information about
    - Congenital malformations
    - Spinal fracture
    - Osteolytic disease – cancer, osteomyelitis, discospondylitis, etc.
  - V/D views less helpful, especially if not sedated
  - COLLIMATE – not a time for cat/dog-o-gram
Diagnostics - Myelography

- Technically challenging but inexpensive.
- General anesthesia, iodinated compound injected into epidural space.
  - Causes reactive meningitis x 2 weeks and small area of permanent focal damage to lumbar spinal cord where needle penetrates.
  - May cause seizures
- Highlights extradural compression
  - Does not always provide accurate side/site
  - Does not give information about health of spinal cord.
  - Helpful in evaluating Type II IVDD for dynamic compression
  - Does not differentiate neoplasia, etc. from IVDD.
Diagnostics – CT +/- myelography

- Rapidly identify acute, compressive, Type I, thoracolumbar IVDD in young dogs.
  - Site, side, extent
- Not as helpful for differentiating acute/chronic sites.
- Does not provide information about health of spinal cord.
- More expensive than myelography, less expensive than MRI
- Helpful in evaluating Type II lesions for dynamic compression with myelography.
- Does not always differentiate IVDD from neoplasia, etc.
  - IV contrast administration can help.
Hansen Type I IVDD
Hansen Type I IVDD
Diagnostics - MRI

• Best modality for evaluating IVD, spinal cord, and nerve roots
  ▪ Not as good bone detail as CT.
  ▪ Evaluates spinal cord for edema/gliosis.
    • More prognostic information than CT or myelography
  ▪ More readily differentiates acute vs. chronic sites.
  ▪ Can evaluate dynamic compression

• More time consuming and expensive than myelography or CT.
Hansen Type I IVDD
Hansen Type II IVDD
Treatment options – Medical

• Type I
  ▪ Rest, anti-inflammatory medication, pain management, bladder management
    • **6 weeks cage rest**, no jumping/stairs for life
    • NSAID OR anti-inflammatory dose of steroids
      – 10-14 days only.
    • Manage secondary damage – hydration, free radicals, etc.
    • +/- muscle relaxer, opioid, and/or gabapentin
    • +/- bladder management
      – U-cath, intermittent catheterization, manual expression.
      – Prazosin (1mg/15kg TID) +/- diazepam, +/- bethanechol
Treatment Options - Medical

- Type II
  - Physical therapy and/or intermittent cage rest.
  - Long term vs. pulse anti-inflammatory medications
  - Long term gabapentin and/or tramadol.
  - No jumping or stairs
  - +/- bladder management
Treatment Options - Surgical

- Hemilaminectomy
- Dorsal Laminectomy
- Lateral Corpectomy
- Ventral Slot
- Distraction/fusion
- Fenestration – Reduces risk of future Type I
Treatment – Post-OP

- 2-4 weeks strict rest, no jumping/stairs for life
- Gradual return to walking, then running, and playing.
- Anti-inflammatory therapy x 10-14 days
- Tramadol, gabapentin as needed
- Muscle relaxer – diazepam, methocarbamol, 5-7 days
Treatment Options - Complementary

- Acupuncture
- Laser Therapy – for muscle soreness.
- Physical therapy – when? How much?
- Massage
- Ice/Hot packing
**Prognosis Type I or III, acute**

<table>
<thead>
<tr>
<th>Neuro-localization</th>
<th>Pain + Medical</th>
<th>Pain + Sx</th>
<th>Pain - Medical</th>
<th>Pain - Sx</th>
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<td>C1-C5</td>
<td>50 %, 30-50% Relapse</td>
<td>90-95%</td>
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<tr>
<td>C6-T2</td>
<td>50%, 30-50% Relapse</td>
<td>70-95%</td>
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<tr>
<td>T3-L3</td>
<td>70%, 30% Relapse</td>
<td>90-95%</td>
<td>10%</td>
<td>50-55%, Timing???</td>
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<tr>
<td>L4-S1</td>
<td>50-70%, ~50% relapse</td>
<td>70-90%</td>
<td>Less than 10%</td>
<td>10-30%, Timing???</td>
</tr>
</tbody>
</table>

**with pain +, no change in prognosis for surgical success ~8wks from time of injury. However, if become pain -, prognosis decreases as above.**
Prognosis Type I, chronic, pain +

• Because of additional scar tissue, prognosis with surgery decreased by about 20-25% if surgery performed >8wks after herniation
Prognosis Type II

• Degree of spinal cord atrophy difficult to quantify.
  ▪ No improvement in neurological grade with surgery if due to atrophy
  ▪ Decreased functional reserve
    • More likely to be worse following surgery
      – Minor trauma during surgery = “last straw effect”

• Surgical vs. conservative management have similar outcomes
  ▪ Indications to try surgery
    • Chronic pain
    • Paralysis
    • Improved neurological grade with anti-inflammatories.
    • Owner goals – try to prevent further worsening knowing risk
References

Questions

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