TITLE: Acute hydrated non-compressive nucleus pulposus extrusion: what do we know so far?

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JOURNAL TITLE: Veterinary Record

PUBLISHER: BMJ Publishing Group

PUBLICATION DATE: 30 November 2017 (online)

DOI: 10.1136/vr.j5494.

ACUTE HYDRATED NON-COMPRESSIVE NUCLEUS PULPOSUS EXTRUSION, WHAT DO WE KNOW SO FAR?

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An acute extrusion of non-degenerated nucleus pulpous material through a tear of the annulus fibrosus can occur after sudden changes of intradiscal pressure and biomechanics (for example during a vigorous exercise, running, jumping). This type of intervertebral disc extrusion can be characterised as compressive or non-compressive and it is being more commonly recognised and studied as the cause of acute myelopathy in dogs and less frequently in cats.[1–6]

The term acute non-compressive nucleus pulposus extrusion was proposed to indicate when the extruded hydrated (as description of non-degenerated) nucleus pulposus contuses the spinal cord and dissipates within the epidural space without significant spinal cord compression.[2] Other terms have been used to describe this type of intervertebral disc extrusion including: high velocity low-volume disc extrusion, traumatic disc extrusion, dorsolateral intervertebral disc explosion, traumatic disc prolapse and erroneously also Hansen type III intervertebral disc disease.[1,2,4,7] From all the terms used, the one that ideally should be avoided is Hansen type III as Hansen described intervertebral disc degenerations (type I and type II) and this particular type of disc disease is non-degenerated and, moreover, it was never described by Hansen.[8]

A new study on this type of intervertebral disc extrusion (summarised in page 549 of this week's issue of *Veterinary Record*) proposed adding the word hydrated to differentiate it from the degenerated and potentially non-compressive/minimally compressive Hansen type I intervertebral disc extrusion, calling it then acute hydrated non-compressive nucleus pulposus extrusion (AHNCNPE).

The clinical presentation of dogs with AHNCNPE is characterised by peracute onset of often lateralised myelopathy that is non-progressive after the first 24 hours with some degree of physical activity at the time of the onset (Figure 1). Lateralisation of neurological deficits has been reported in around 60% of dogs (similar to recent study of this week's issue by *Ros and others*) however in one study the lateralisation of the clinical sings was reported in up to 90% of the affected dogs.[9] Discomfort or hyperalgesia during palpation of the affected area have been described in up to 57% of dogs with AHNCNPE at the time of the onset, however it is unlikely that this clinical sign sustained or become severe after 24 hours. Any canine breed and rarely cats can be affected. Male dogs seem to be affected more commonly than females. The age at diagnosis in dogs is usually around 6 years (range 2 to 12 years of age).[2,5,9,10] The T3-L3 spinal cord segments and in particular the T12-T13, T13-L1, and L1-L2 intervertebral disc spaces are most commonly affected.

It is important to emphasised that based on the signalment, history and neurological examination a high clinical index of suspicion for AHNCNPE can be reached. The clinical presentation can be very similar to ischaemic myelopathy (for instance in cases of fibrocartilaginous embolic myelopathy (FCEM)). A recent study from *Fenn and others* compared the clinical presentation in dogs with presumptive ischaemic myelopathy and dogs with AHNCNPE.[9] This study concluded that dogs with AHNCNPE were signicantly older at disease onset and were more likely to have a history of vocalization at onset of clinical signs and have hyperesthesia on palpation of the vertebral column during initial examination compared with dogs with ischemic myelopathy. A definitive diagnosis of AHNCNPE is only possible at post-mortem examination; however, the combination of clinical presentation and magnetic resonance imaging (MRI) findings provides the mainstay of presumptive antemortem diagnosis.[2,11]

The MRI features compatible with AHNCNPE (Figure 2) include evidence of reduced volume of the nucleus pulposus, focal hyperintensity within the overlying spinal cord on T2-weighted images, extraneous material or signal changes in the epidural space, and minimal to no spinal cord compression.[2,4,11]

On the other hand, some owners might have financial restriction or MRI might not be available in some institutions during out of hours. On these circumstances, other imaging modalities (such as myelogram or computed tomography) could be considered to exclude compressive myelopathies with an acute onset (for instance Hansen type I intervertebral disc extrusion) and therefore contribute to the presumptive diagnosis of AHNCNPE.

To date, little data have been published on the myelographic appearance of AHNCNPE.[5] McKee and other reported radiographic and myelographic features in 48 dogs with presumptive AHNCNPE, however none of the affected dogs had MRI and therefore some minimally compressive Hansen Type I intervertebral disc extrusions could have been included. The recent study by *Ros and others* describes the myelographic appearance of 21 dogs with suspected AHNCNPE (diagnosed on MRI findings): all dogs had intramedullary patterns (attenuation of both contrast columns), 57% of the dogs showed extradural pattern and the affected intervertebral disc was narrowed in all the dogs. The length of spinal cord swelling measured on myelogram is a controversial indicator for prognosis and this was supported by the study of *Ros and others*, where spinal cord swelling (measured by myelogram) did not associate with neurological grade or outcome.

When evaluating MRI of affected dogs with AHNCNPE, it is obvious the reduced volume of the extruded nucleus pulposus on transverse planes (Figure 3) and sagittal planes of T2W images, however this finding has not been previously evaluated or associated to the clinical presentation. *Ros and others* measured the volume of the affected nucleus pulposus, compared that volume with the mean volume of the two-adjacent intervertebral discs and concluded that the extruded volume of the nucleus pulposus was significantly associated with the neurological grade at presentation.

Outcome of AHNCNPE is successful in the majority of the dogs. A recent large study by *Mari and others*[10] reported successful outcome (ambulatory without assistance and complete urinary and faecal continence) in 73% of the dogs. This study also found that dogs with AHNCNPE were five times more likely to develop faecal incontinence (23% of the affected dogs) than dogs with suspected ischaemic myelopathy, enhancing the benefits of diagnosing these conditions by MRI to further evaluate possible outcomes. [2,9–11]

Despite the reported myelographic findings by *Ros and others*, MRI should still be considered the gold start imaging modality for the diagnosis of ANNPE. Myelography can provide useful information in emergency situations to assess the degree of spinal cord compression and therefore the need to bring that patient to surgery if indicated.

Clinical importance for practitioners

- Dogs with acute hydrated non-compressive nucleus pulposus extrusion (AHNCNPE) present with characteristic clinical signs
- The signalment, history and neurological examination provide a high clinical index of suspicion for AHNCNPE
- The mainstay of presumptive antemortem diagnosis can be reached with the combination of clinical presentation and MRI findings

- Myelography could help to rule out surgical conditions when there are financial restrictions or advanced imaging is not available
- AHNCNPE has a good outcome in the majority of the dogs
- Dogs with AHNCNPE are at risk of developing faecal incontinence

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Figure Legends

Figure 1 - 6 years old Staffordshire Terrier Crossed, female spayed with peracute onset of ambulatory paraparesis with spontaneous knuckling on the left pelvic limb after playing in the park. She was diagnosed with an acute hydrated non-compressive nucleus pulposus extrusion at the level of L1-L2 intervertebral disc.

Figure 2 - (A) Sagittal T2-weigthed image of the thoracolumbar spine of an 11yo dog with peracute onset of left pelvic limb monoplegia. There is focal hyperintensity within the spinal cord overlying the IVD L2-L3 with reduction in volume of the nucleus pulposus (black arrow head). (B) Transverse T2-weighted image; (C) transverse T1-weighted FSE image. There is a focal area of hyperintesitiy on T2W within the spinal cord parenchyma lateralised to the left (asterisk) and signal change within the epidural fat (black arrow) on the same side (B, C) with minimal spinal cord compression.

Figure 3- Transverse T2 weighted images at the level of L1-L2 intervertebral disc (A), at the level of L2-L3 (B, same case as Figure 2, AHNCNPE at L2-L3) and at the level of L3-L4 (C). The volume of the affected nucleus pulposus is reduced (B) compared with the cranial and caudal adjacent disc (A,C).