

IMAGING DIAGNOSIS—AN ATYPICAL PRESENTATION OF DIFFUSE IDIOPATHIC SKELETAL HYPEROSTOSIS (DISH) IN A DOG

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A 10-year-old female spayed Dalmatian was evaluated for progressive cervical scoliosis and stiffness. This imaging report describes the imaging and postmortem findings for this patient. A diagnosis of an atypical manifestation of diffuse idiopathic skeletal hyperostosis (DISH) was made based on imaging and additional diagnostics. This report serves to increase awareness of DISH in the veterinary community, as well as to describe a unique presentation of the disease with atypical lesion distribution and severity. In addition, this report contrasts the pathophysiology and imaging characteristics of DISH with spondylosis deformans, which can appear comparable radiographically. © 2013 American College of Veterinary Radiology.

Key words: CT, extracortical ossification, hyperostosis, spondylosis deformans.

Signalment, History, and Clinical Findings

A 10-YEAR-OLD FEMALE spayed Dalmatian presented for a 1-year history of progressive cervical scoliosis and stiff gait. Mobility initially improved with oral corticosteroids, but 6 weeks later she fell without losing consciousness. At this time she was referred for further evaluation. Physical and neurological examination revealed a stiff, pacing gait in all four limbs without evidence of proprioceptive ataxia. Additionally there were postural reaction deficits in the right thoracic limb and the right and left pelvic limbs, moderate atrophy of left thoracic limb and epaxial musculature, and left-sided cervical scoliosis with limited cervical range of motion. Extensor tone was increased in the right thoracic limb (normal in the remaining limbs), withdrawal reflex was decreased in the right thoracic limb (normal in the remaining limbs), and the patellar reflex was increased in the right pelvic limb. Mild pain was elicited on palpation of the lumbosacral spine. These findings (right thoracic limb increased extensor tone with decreased withdrawal, right patellar hyperreflexia) indicated a lesion affecting the C₆ – C₈ spinal cord segments on the right side.

Imaging, Diagnosis, and Outcome

Lateral and ventrodorsal digital radiographs of the cervical and thoracolumbar spine were made at presen-

tation (Canon CXDI-50G imaging plate; eFilm Workstation, Merge Healthcare). Proliferative osseous lesions were present along the entire length of the spine with varying degrees of severity and distribution, depending on the segment (Fig. 1). In general, ossification adjacent to dorsal vertebral structures (vertebral arches, articular, and spinous processes) was more severe than ventral ossification, with the exception of the lumbosacral spine.

Pseudarthroses were present between the bases of adjacent spinous processes throughout the entire cervicothoracic spine and extended to the level of L₄. There was a small amount of ossification at the tips of several spinous processes (Fig. 1A and B). Ventral lesions in these segments were sparse, with mild ossification at the C₆–C₇ and T₁₀–T₁₁ intervertebral disc spaces. There was no evidence of disc degeneration in the cervical or thoracic spine with the exception of T₁₀–T₁₁, which had mild in situ mineralization and a narrowed disc space (Fig. 1B).

Lesions in the lumbosacral spine were predominantly ventral, with flowing ossification bridging the intervertebral disc spaces of L₂–S₁. The disc spaces in the lumbar spine did not display extensive signs of disc degeneration (i.e., disc space narrowing, endplate sclerosis, nuclear calcification) with the exception of the L₄–L₅ and L₅–L₆ disc spaces that appeared slightly narrowed (Fig. 1C).

Based on the radiographic findings, differential diagnoses included diffuse idiopathic skeletal hyperostosis (DISH), Hepatozoonosis, and hypervitaminosis A.

Complete blood count and biochemistry profile (unremarkable results) and PCR for *Hepatozoon americanum* (negative results) were performed in the initial visit. The dog was medically managed on oral meloxicam for approximately 2 years until her mobility became too severely

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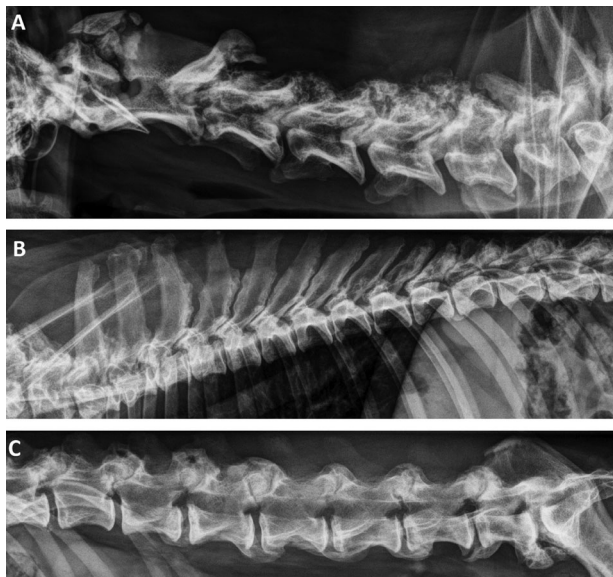


FIG. 1. Left lateral survey radiographs. (A) There is abundant irregular new bone formation surrounding articular and spinous processes, most severe in the caudal cervical spine. (B) Ossification in the thoracic spine has resulted in the formation of multiple pseudarthroses between the bases of adjacent spinous processes. (C) Ventral lesions predominate in the lumbar spine, sparing dorsal structures caudal to L_4 .

impaired and the owner elected euthanasia. Postmortem CT images of the vertebral column were acquired using a GE LightSpeed 8-multidetector helical CT scanner (General Electric, Fairfield, CT), followed by necropsy.

CT images revealed that the cortical margins and architecture of affected vertebral structures were maintained, and ossification appeared to be extracortical (Figs. 2 and 3). Moderate amounts of new bone surrounded vertebral arches and articular and spinous processes along the entire cervical and thoracic spine to the level of L_4 , causing variably narrowed intervertebral foramina between C_3 – C_7 (Fig. 2). There was mild peri-articular ossification at costovertebral joints and varying amounts of ossification along the ventral and lateral aspects of thoracic vertebral bodies, the most extensive bridging the T_{10} – T_{11} disc space.

Lumbosacral osseous lesions were distributed along the ventral and lateral aspects of the L_2 – S_1 vertebral bodies, forming bony bridges across intervertebral disc spaces. Dorsal structures caudal to L_4 were unaffected.

Cervical CT images also allowed evaluation of the proximal thoracic limbs. There was a large amount of extracortical ossification associated with both scapular spines and subscapular surfaces, and mild ossification at the lateral aspects of both humeral heads (Fig. 3).

Gross postmortem examination revealed severe bony proliferation that was especially pronounced over the dorsal aspects of multiple cervical vertebrae. Histopathology of the osseous lesions demonstrated chronic osseous proliferation without any evidence of neoplasia or osteomyeli-



FIG. 2. Transverse CT bone window image at the level of T_4 . The spinous process is surrounded by extracortical new bone growth and there is a small amount of ossification along the ventral surface of the vertebral body. Notice the preserved cortical margins of the spinous process (arrows).

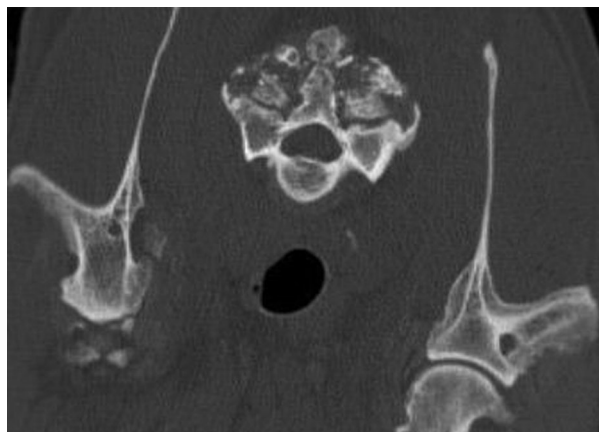


FIG. 3. Transverse CT bone window image at the level of C_6 . There is abundant irregular new bone formation within soft tissues adjacent to the spinous and articular processes and the dorsal lamina. There is also a large amount of extracortical ossification adjacent to both scapular spines and subscapular surfaces.

tis (Fig. 4). Histological findings in other organs included minor unrelated changes in the kidneys and lungs (membranoproliferative glomerulopathy, bronchiolar squamous metaplasia), but were otherwise unremarkable.

Using the diagnostic criteria in Table 1, the dog was determined to have an unusual manifestation of DISH. Fulfillment of four out of the first five criteria is sufficient to support this diagnosis.¹ Criterion 2 was not fulfilled, as there was evidence of mild intervertebral disc degeneration at T_{10} – T_{11} , L_4 – L_5 , and L_5 – L_6 . Criteria 6 and 7 could not be evaluated due to the absence of pelvic imaging. Criteria 1, 3, 4, and 5 were all fulfilled, thus a diagnosis of an atypical DISH was established.

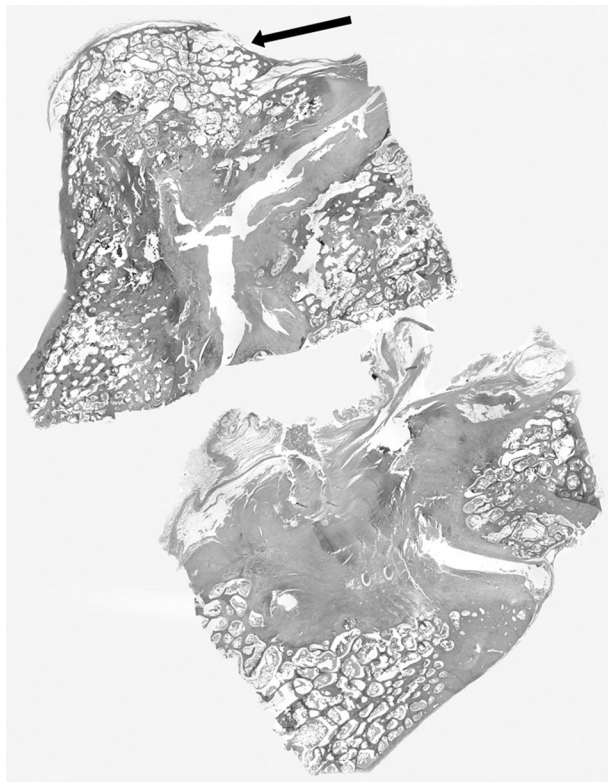


FIG. 4. Histopathology, sagittal section through C₅-C₆. There is severe proliferation of mature lamellar, trabecular bone bridging overlying the facet joint (top, arrow). The annulus fibrosus is fibrillated and degenerate (bottom).

Discussion

Diffuse idiopathic skeletal hyperostosis is a disease of unknown etiology affecting the axial and appendicular skeleton of humans, dogs, and other species of mammals. Metabolic, endocrinological, genetic, and biomechanical factors have been implicated in the pathogenesis, and associations with diabetes mellitus and obesity in humans have been made.^{2,3} Diffuse idiopathic skeletal hyperostosis often remains clinically silent but can lead to spinal pain, stiffness, and in advanced stages spinal cord or nerve root compression and neurologic deficits.³

In this report, we present a unique manifestation of DISH in a large breed dog. Diffuse idiopathic skeletal hy-

perostosis is relatively unknown in the veterinary community, gaining recognition only recently.^{3,4} The few reports that do exist in the veterinary literature mostly describe lesions similar to those found in humans, with flowing ossification and segmental bridging ankylosis along the ventral surface of vertebral bodies.^{3,4} In contrast, osseous lesions present in this patient predominantly affected dorsal vertebral structures, with relatively mild ventral lesions in the lumbar spine. All other differential diagnoses were ruled out based on negative serology for *H. americanum*, lack of historical vitamin A toxicosis (no known exposure to synthetic retinoids or diet with excessive vitamin A), as well as the character and histopathology of osseous lesions.

The characteristic flowing ossification of DISH with segmental bridging ankylosis along the ventral surface of vertebral bodies can appear similar to the radiographic appearance of spondylosis deformans, a noninflammatory bony response to intervertebral disc degeneration.⁵ Differences in lesion distribution and pathophysiology indicate that these syndromes are two separate entities. Diffuse idiopathic skeletal hyperostosis is a systemic disorder characterized by fibrocartilagenous proliferation followed by endochondral ossification within soft tissues of the axial and appendicular skeleton. Ossification of DISH appears to affect an area rather than specific anatomic structures, as it develops not only at entheses but also along the surfaces of ligaments, short fibers and in neighboring connective tissue, suggesting a possible biomechanical component to its poorly understood etiopathogenesis.^{2,6} Spondylosis deformans develops in an effort to stabilize the disc space following degeneration of the annulus fibrosus, with focal new bone formation in association with vertebral disc articulations. Lesions of spondylosis typically spare at least part of the ventral surfaces of adjacent vertebral bodies.^{4,5,7}

Disc degeneration and spondylosis are not thought to contribute to the development of DISH, however, these conditions all have higher prevalence with increasing age and may coexist.^{3,4} Potential coexistence of disc degeneration, spondylosis deformans and DISH in older dogs can confound the radiographic differentiation of the disorders. Close inspection is necessary to distinguish the conditions and recognize their coexistence, if present.^{3,4}

TABLE 1. Diagnostic Criteria*, Morgan and Stavenborn (1991)

1. Flowing calcification and ossification along ventral and lateral aspects of three contiguous vertebral bodies leading to segmental bony ankylosis.
2. Relative preservation of disc width within involved areas and absence of extensive radiographic changes of degenerative disc disease such as end-plate sclerosis, nuclear calcification, or localized spondylosis deformans.
3. Peri-articular osteophytes surrounding true vertebral joints.
4. Formation of pseudoarthrosis between the bases of the spinous processes.
5. Peri-articular osteophytes and calcification and ossification of soft tissue attachments (enthesiophytes) in both axial and peripheral skeleton.
6. Peri-articular osteophytes, sclerosis, and ankylosis of sacroiliac joints.
7. Bony ankylosis of the symphysis pubis.

*Presence of 4 of the first 5 criteria is sufficient to support a diagnosis.

TABLE 2. Diagnostic Criteria, Resnick and Niwayama (1976)

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1. Presence of flowing calcification and ossification along the anterolateral (ventrolateral) aspect of at least four contiguous vertebral bodies with or without associated localized pointed excrescences at the intervening vertebral body–intervertebral disc junctions.
 2. Relative preservation of intervertebral disc height (width) in the involved vertebral segment and the absence of extensive radiographic changes of “degenerative” disc disease, including vacuum phenomena and vertebral body marginal sclerosis.
 3. Absence of apophyseal (articular process) joint bony ankylosis and sacroiliac joint erosion, sclerosis, or intra-articular osseous fusion.
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The criteria in Table 1 (also known as the “Morgan criteria”) have been accepted by many as the gold standard for the diagnosis of DISH in dogs, however, dispute over which diagnostic paradigm should be used in veterinary medicine still exists.^{1,7,9} The criteria in Table 2 (also known as the “Resnick criteria”) have long served as the mainstay of diagnosing DISH in humans, where lesions are typically most extensive in the anterior (ventral) spine. However, two past reports in dogs describe prominent dorsal lesions that deviate from the “classic” appearance, prompting the proposal of the Morgan criteria. Some state that the Morgan criteria are too strict, calling for a return to the Resnick criteria for the radiographic diagnosis of DISH in dogs in order to enhance our identification of the disease and support the “one medicine” concept.⁷

Given the significant variation that may occur between humans and dogs with DISH in both lesion severity and distribution, it seems that both diagnostic paradigms have limited reliability when confronted with an atypical man-

ifestation of the disease. The Resnick criteria emphasize “classic” ventral lesions, excluding potential extra-articular involvement of dorsal vertebral structures. The Morgan criteria offer wider lesion distribution, including dorsal vertebral and appendicular involvement; however, rigid inclusion requirement for four out of the first five criteria may exclude cases that are in fact DISH.

Ultimately the Resnick criteria appear to be reliable for diagnosing most cases of DISH in dogs, however in atypical cases (such as the one herein reported), the character and distribution of ossification may facilitate a diagnosis. Diffuse idiopathic skeletal hyperostosis should be suspected in any case of widespread extracortical, extra-articular ossification affecting entheses and soft tissues of the axial and/or appendicular skeleton.

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