

Hypertrophic Osteodystrophy

(HOD)

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Introduction

Hypertrophic Osteodystrophy (HOD) is a developmental disease in larger breed dogs (commonly, the Great Dane, Alaskan Malamute, Weimaraner and Irish Setter). This disease usually begins between the ages of 3 months to 5 months of age. Signs can vary in intensity, and several dogs from one litter may be affected, although at different times. The heritable predisposition of the disease has not yet been documented, and the Irish Setter Health Committee is supporting research into possible DNA HOD markers. The information in this paper is based upon current published literature on HOD, treating six Irish Setters personally and from telephone consultations in 32 cases of Irish Setter HOD.

Clinical Signs of HOD

HOD affected animals generally present with lameness or reluctance to walk. Early in the disease, the metaphyseal regions of the long bones (the area above the diaphysis (mid-shaft of long bones) and below the physis (the growth plate)) will be tender to digital palpation, slightly swollen and warm to the touch (using the inside of the wrist). The disease is usually bilateral, most commonly affecting the distal radial/ulnar metaphysis (above the wrist joints), although the metaphyses of all long bones are susceptible. More adversely affected animals may be systemically ill, exhibiting fevers of 104 – 105.8°F and anorexia (refusal to eat).

While symptoms may be episodic, without treatment the disease generally progresses, with HOD affected dogs continuing to experience high fevers, anorexia and rapid weight loss.

Physiological Changes

Initially, necrosis of the capillary loops that invade the cartilage model of the metaphyseal physis occurs in the primary spongiosa. The calcified cartilage lattice of the primary spongiosa becomes elongated, impacting the trabeculae. Necrosis, failure of osseous tissue deposition on the calcified lattice and trabecular microfractures are associated with acute suppurative inflammation in the intertrabecular areas. With progressing inflammatory changes, periosteal calluses form over the distal ulnar metaphysis. Radiographic changes reflect the underlying histologic changes, and include a radiolucent line (HOD line) parallel and immediately adjacent to the growth plate in the metaphyseal regions. (Troestel, Pool, McLaughlin). This line represents bone necrosis and reabsorption of some of the microspicules of bone. (Troestel, Pool, McLaughlin). With treatment and effective healing, the cuff of metaplastic cartilage and bone bridges to the cortex and moves toward mid-diaphysis (midbone).

These HOD related changes can disturb normal cartilage growth and development of the adjacent growth plate (physis). The resulting interference in the cartilage transformation into bone (endochondral ossification) at the growth plate may be visualized radiographically as finger-like projections extending into the metaphyseal marrow. Absent efficacious treatment, the growth plate disruption may result in shorter bone length or long bone curvature, especially at the wrist, e.g., radius curvus. In my experience, this is more common in the Great Dane than the Irish Setter. Additionally, dogs with sustained signs of high fevers, interstitial pneumonia, and bronchitis may have soft tissue calcification.

Causes and Predispositions

The cause of HOD remains unknown; however, there are many speculations. In Weimareiners, a hyper immune response to some trigger has been noted (Abels, Harrus, Angles; and Harrus, Waner, Aizenberg). The disease in Weimareiners sounds a lot like the disease in the Irish Setters (Angles). This is the rationale for anti-inflammatory prednisone. Stress may precipitate the disease, including a rapid dietary change over 1 to 6 days. Viral causes and vaccinations also have been implicated, although they too just might be one more kind of stress, e.g., 3 to 5 days after the third "combo" vaccine (modified live virus), after administration of Rabies vaccines in four- month old puppies (two cases), or a fourth (often unnecessary) vaccine at 16 to 18 weeks. Vitamin C deficiency also has been speculated as a possible cause; however, there is neither documentation nor scientific reason for this in the dog, and Vitamin C therapy has not met with scientific success.

An infectious origin has been proposed, and there are reports of hematogenous (blood borne) bacteria producing florid radiographic changes in the metaphyses similar to those of HOD. A good radiologist may be necessary to distinguish between possible hematogenous infection, osteomyelitis (bone infection) and HOD radiographic changes. This author is unaware of any published literature correlating blood culture results with HOD.

Diagnosis

Diagnosis is usually clinical, with subsequent radiographic confirmation. In the early stages there is point tenderness in the metaphysis, and radiographic changes, as discussed above, may be present as early as one week later. There must be an HOD line for a diagnosis of HOD! Metaphyseal regions may remain mildly affected throughout the course of the disease if well treated, or if poorly treated, may show early irregular widening with abnormal endochondral ossification and growth plate alterations. Severe alterations to the growth plate (most often occurring in the distal ulna), may produce lateral bowing deformities of the front legs. CBC will show neutrophilia, with bands of 3% or less. Bands present in quantities greater than 3% increases the suspicion for sepsis rather than HOD.

Treatment

In all cases of HOD, treatment is begun by immunosuppressive doses of Prednisone, covered by antibiotics. The dosing regimen is as follows: (1) place the dog initially on a 1.5 mg/kg/day dose of Prednisone for 4 to 5 days if symptoms show regression, and up to but not more than 7 days if signs are persisting, with half given in the a.m. and the other half in the p.m., (2) gradually wean down for 4-5 weeks, cutting the total daily dose by one-half each week, and (3) administer 5 mg of Prednisone every other day for an additional 1 to 2 weeks. In all cases, also use 3V Caps (or Derm Caps®) and either Glyco-Flex or Multi-source Glucosamine. Supportive care should be provided as needed. We add oral antibiotics: usually Clavamox, Amoxicillin or Clindamycin for 3 to 4 weeks. Also, administer antacids (Pepcid, Zantac) to counter acid secretions stimulated by the Prednisone. With Prednisone treatment, pain medications can be stopped sooner, thereby avoiding possible appetite suppression often associated with pain medications. HOD dogs should not be exposed to possible contagious disease, and owners should be advised not to take their dogs to dog shows, dogs parks, etc.

The prognosis for most cases is good if this protocol is instituted early. Even in severe cases this protocol has been effective. In our experience, mild cases are not difficult to treat, whereas the more severely affected animals require more aggressive care. Those animals that are not treated early may require IV fluids and electrolytes, nutritional support, and tremendous nursing care to arrive at a successful result. Nursing care is paramount in the successful management of the more severe cases. Mild cases which have been treated solely with Rimadyl® or Deramaxx® may respond incompletely, and often have a subsequent relapse.

In two cases of Great Dane HOD, mild puppy strangles (juvenile cellulitis/Staphylococcus plus toxins) were apparent. The use of Prednisone concomitant with antibiotics in cases of puppy strangles was critically important.

Conclusion

Early recognition and appropriate treatment of HOD will hopefully prevent your dog from reaching a critical state. I would hope that some of this information will assist in making the early diagnosis of HOD, and welcome your feed back.

Memo to Members (ISCA Memo) update:

Thanks to everyone who has shared information with me about Hypertrophic Osteodystrophy (HOD) cases. This is very informative and important information.

As a result, a few comments are in order:

1. Be sure sepsis or other infection has been ruled out before initiating glucocorticoid therapy (Prednisone, Prednisolone). Do a complete workup, including a CBC and radiographs. Remember, there must be an HOD line for a diagnosis of HOD!
2. The dosing regimen is as follows: (1) place the dog initially on a 1.5 mg/kg/day dose of Prednisone for 4 to 5 days if symptoms show regression, and up to but not more than 7 days if signs are persisting, with half given in the a.m. and the other half in the p.m., (2) gradually wean down for 4-5 weeks, cutting the total daily dose by one-half each week, and (3) administer 5 mg of Prednisone every other day for an additional 1 to 2 weeks.
3. Cover with Clavamox, Antirobe or even Amoxicillin for at least three weeks.
4. Administer antacids (Pepcid, Zantac) to counter acid secretions stimulated by the Prednisone.
5. Two HOD cases also evidenced sore mandibles before treatment. These cases showed more tenderness than that normally associated with normal puppy teeth eruptions.

Keep up the good work and the flow of information!

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