HYPERTROPHIC OSTEODYSTROPHY IN A WHITE SWISS SHEPHERD DOG TREATED WITH GLUCOCORTICOID

OSTEODISTROFIA HIPERTRÓFICA EM UM CÃO DA RAÇA PASTOR BRANCO SUÍÇO TRATADO COM GLICOCORTICÓIDE

A. C. CAMPLESI[1]; F. C. D. MOTA[2]; E. PORTUGAL[3]; R. V. CASALE[4]; H. C. BUENO[5]; R. G. NETO[5]; A. TREVISAN[4]; M. F. R. SOBREIRA[1]; C. P. BURGER[6]

SUMMARY

Hypertrophic osteodystrophy is a canine disease that primarily affects the metaphyses of long bones during growth stage. It is also known as Moller-Barlow disease, idiopathic osteoarthritis, hypo or avitaminosis C or metaphyseal osteopathy, among other less used. This is a case report of hypertrophic osteodystrophy treated with glucocorticoids. The disease was diagnosed through laboratory tests, especially radiographic examination, which showed radiolucent areas in the distal metaphysis of the radius, ulna and tibia, with a small periosteal bone formation in the ulnar metaphysis. The treatment based on prednisone, cephalexin, worked fast and on the 15th day limbs improvement was noticeable, with no further swelling. Based on these data it was concluded that glucocorticoids are the treatment of choice for bringing disease into remission quickly.

KEY-WORDS: Osteodystrophy. Canine. Corticosteroids. Prednisone.

RESUMO

A osteodistrofia hipertrófica é uma doença dos canídeos em crescimento que afeta primariamente as metáfises dos ossos longos. Por vezes também lhe são atribuídas outras denominações como: doença de Moller-Barlow, osteodistrofia idiopática, hipo ou avitaminose
C ou osteopatia metafisária, entre outras menos usadas. Este trabalho teve como objetivo relatar um caso de osteodistrofia hipertrófica tratado com glicocorticoide. O diagnóstico foi obtido através dos exames complementares, principalmente pelo exame radiográfico o qual apresentou zonas radioluscentes nas metáfises distais do rádio, ulna e tíbia, com pequena neoformação óssea periosteal na metáfise ulnar. O tratamento foi à base de prednisona, cefalexina, obtendo melhora rápida aos 15 dias onde já não se notava mais o aumento de volume nos membros. Baseado nesses dados concluiu-se que o tratamento com glicorticóides é uma boa opção para a afecção trazendo remissão dos sintomas de forma rápida.

PALAVRAS-CHAVE: Osteodistrofia. Canino. Corticoide. Prednisona.

1 Veterinarian. Prof. Dr. FCAV-UNESP-Jaboticabal/SP
[2] Veterinarian. Prof. Dr. UFU-Uberlândia/MG
[3] Veterinarian. Prof. M.Sc. UNICASTELO – Descalvado
[4] Veterinarian at Hospital Veterinário UNICASTELO – Descalvado.
[5] Veterinarian
[6] Post-graduate in Veterinary surgery at FCAV-UNESP-Jaboticabal/SP

INTRODUCTION

Dog musculoskeletal system presents high metabolic activity during the first 12 months of life (RICHARDSON & ZENTEK, 1998). During this period, this system is more susceptible to developmental abnormalities like osteodystrophy, a term that includes both osteopenia and osteomegalia.

Osteomegalia resultσ from a multifactorial process with genetic, nutritional and environmental etiology (DAMMRICH, 1991; ZENTEK & MEYER, 1991). Giant dog breeds are particularly susceptible to these conditions, including hypertrophic osteodystrophy, asynchronous growth of the radius and ulna, Panosteitis, Wobbler syndrome, coxofemoral dysplasia, elbow dysplasia and osteochondrosis (BRAWNER, 1998; HAZEWINKEL et al., 1998; RICHARDSON & ZENTEK, 1998; OWENS & BIERY, 1999; THRALL, 1998; COOK, 2001; FOSSUM, 2005). The most commonly affected breeds are Rottweiler, Bernese Mountain Dog, New Foundland, Great Dane, Boxer, German Shepherd and retrievers, as Golden and Labrador (DAMMRICH, 1991; MEYER & ZENTEK, 1991).

Hypertrophic osteodystrophy, also known as metaphyseal osteopathy, Moeller-Barlow or Barlow disease is an idiopathic disease that causes destruction of metaphyseal trabeculae in the long bones of young dogs that grow quickly. Clinical signs usually appear at about 3-4 months of age (JOHNSON & WATSON, 1997; FOSSUM, 2005); however, they can occur as early as two months and recurrences can appear up to 8 months (GRONDALEN, 1976; FOSSUM, 2005). Male dogs are affected more often than females. Canola and Andrade (1996) stated that HOD is a disease with low clinical manifestation.

The cause of HOD is unknown, thus classifying the disorder among idiopathic bone disorders of small animals. The pathogenesis is unclear, but an apparent disturbance of metaphyseal blood supply leads to changes in the physis and adjacent metaphyseal bone, resulting in delayed ossification of the physeal hypertrophic zone (FOSSUM, 2005). The acute phase of the disease lasts about 7-10 days. Affected puppies show metaphyseal swellings and pain, accompanied by depression, loss of appetite and variable pyrexia; they refuse to stand up and present generalized weight loss. These animals can become very ill and require intensive support therapy (JOHNSON & WATSON, 1997; FOSSUM, 2005). Some dogs recover in a few days, but others may experience one or more relapses before final recovery. However, when relapses, pain, weakness and cachexia are frequent, euthanasia is required (FABRIS, 2009).

The diagnosis can be confirmed by radiographic examination. The changes that occur mainly in the metaphyses of long bones are usually bilateral. In the initial stage, abnormal radiolucent lines or bands are present within the metaphysis, separated from the growth plate, which shows normal appearance of dense range (LENEHAN & FETTER, 1985; FOSSUM, 2005). The surrounding soft tissue can be inflamed. Subsequent radiographs may show metaphyseal growth with formation of irregular periosteal new bone, although not all affected dogs exhibit these changes (FOSSUM, 2005). Once the condition is no longer active, bone changes undergo remodeling and repair (JOHNSON & WATSON, 1997).

While there is no specific treatment for hypertrophic osteopathy, avoiding imbalances or nutritional excesses is advisable, and anti-inflammatories and analgesics should be administered as needed to minimize pain (FOSSUM, 2005). Lenehan and Fetter (1985) state that rest and analgesics are usually sufficient to prevent dehydration, malnutrition, and pressure and decubitus sores as well (JOHNSON & WATSON, 1997). There are even indications in the literature of treatment with antibiotics, vitamin C, dipyrone and correction of the diet (CANOLA & ANDRADE, 1996). But it is not proved that they can effectively shorten the cycle or severity of this disease.

Numerous treatments have been proposed; however, no consistent results have been reported. Most authors indicate rest, analgesics or nonsteroidal anti-inflammatory and corticosteroids in severe cases for fever and pain relief. This study reports a case of hypertrophic osteodystrophy diagnosed in a Swiss Shepherd dog, subjected to treatment with corticosteroids since there are few reports in the literature and wide divergence in relation to the recommended therapy and clinical course of the disease.

CASE REPORT

The dog was seen at the Veterinary Hospital of UNICASTELO, Descalvado, SP. The four-month old White Swiss Shepherd dog presented a history of persistent pyrexia, decreased appetite, paralysis of limbs, swelling of the radiocarpal and tibial-tarsal joints, lameness and soreness for one week. The animal was lethargic but had good body condition (score 5 in the 1-9 rating scale). The diet was based exclusively on premium food for puppies. The dog had been treated with ketoprofen, and dipyrone with no result.

Depletion, hyperthermia, increased volume at the long bones extremities in all four limbs, and pain sensitivity were all observed upon clinical examination. The additional blood tests showed leukocytosis with neutrophilia and monocytosis, while the X-ray displayed radiolucent zones in the distal metaphysis of the radius, ulna and tibia, with a small periosteal bone formation in the ulnar metaphysis. The clinical symptoms lead to the HOD diagnosis. The treatment prescribed consisted of: 2 mg/kg oral prednisone every 12 hours in the first week, gradually tapering to 50% of the dose every 5 days, and preventive antibiotic therapy, 30mg/Kg cephalexin each 12 hours for 15 days. Clinical improvement was significant on the second day of treatment, when the animal was already trying to walk. Bone swelling

started to decrease after one week of treatment, returning to normal after 15 days. The dog returned to eating well, became very active and was discharged after 30 days, with complete remission of clinical signs and no recurrence.

DISCUSSION

The etiology of hypertrophic osteodystrophy is quite controversial and conflicting in the literature. The most common causes listed are hypovitaminosis C, excess calcium in the diet, puppies over nutrition and the presence of infectious agents, such as canine distemper virus, but the infectious etiology claim is not substantiated. Furthermore, according to Johnson & Watson (1997) the hypothesis that hypovitaminosis C is responsible for the pathogenesis of the disease, as defended by some authors, has not been substantiated. The attempts to identify causal infectious agent or transmission of the disease were all unsuccessful. In the case reported above, the animal was fed only dry dog food containing between 1.0 and 1.4% calcium, 30% crude protein and enriched with 70 mg of vitamin C per kg of feed. The animal presented no respiratory signs or other clinical signs that could lead to possible presence of canine distemper virus. There was no extra calcium supplemented in the diet of the animal, which did not exceed the optimum calcium level and there was no vitamin C deficiency. However, the animal was fed freely a good quality feed with high protein level. According to Carneiro et al. (2006) diets with high energy density, apparent digestibility of dry matter higher than 85% and protein source of high biological value and fixed source of ingredients may be responsible for the increased incidence of skeletal disorders in large breed dogs during the growing stage. These authors also stated that diet nutritional profile and appropriate feeding methods during the animal's life, especially during growth; decrease the risk of developing these disorders. Teare et al. (1979) suggest that animals that fed freely on highly palatable diets may develop skeletal changes possibly due to hypercalcitoninemia, resulting from a decrease in bone resorption induced by the calcium, protein and energy rich diet. Although the dog in the study had signs compatible with over nutrition, its good body condition (score 5) leads us to classify the disease as idiopathic.

The CBC results did not help the diagnosis, since it only indicates inflammatory process. Johnson & Watson (1997) stated that routine biochemical and hematological tests contribute little to the diagnosis, although neutrophilia, monocytosis and lymphopenia may occur during the active phase, indicating tension and inflammation. However, the complete blood count is important to exclude other differential diagnoses, such as septic arthritis and Panosteitis, in addition to the biochemical tests for renal and hepatic evaluation, which normally lie within the normal range in this disease. Radiographic examination is the most important tool for the diagnosis of hypertrophic osteodystrophy. According to Fossum (2005) the treatment of affected animals should consist of analgesics to control pain while corticosteroids, antibiotics and vitamin C may be also administered. However, they have not been proven effective to shorten the cycle or severity of the disease. Also, sometimes severely debilitated animals require water support. The dog in this case had already been treated with non-steroidal and analgesic anti-inflammatory, but the clinical symptoms had not improved. After being prescribed prednisone and having taken it for 2 days, the symptoms improved rapidly and the dog presented a satisfactory response regarding pain relief and general improvement. Anti-inflammatory corticosteroids decrease leukocyte migration, inhibit the synthesis and release of endogenous pyrogens. In the bones, they increase bone resorption, as well as the competence of osteoclasts, and decrease the generative activity of bone matrix by osteoclasts. Furthermore, they antagonize vitamin D and consequently reduce calcium absorption by the body (ANDRADE & JERICHO, 2002). According to the possible disease pathophysiology, the ways steroids act in the body show them to be very beneficial in the treatment of HOD.

CONCLUSION

Based on the clinical findings of this report, it is concluded that the animal had idiopathic hypertrophic osteodystrophy. It is suggested, given the remission of the disease in this study, that glucocorticoid therapy should be used to treat young dogs with HOD.

Figure 1 - Lord, the 4-month old dog showing swelling of the radio-carpal and tarsal tibial joints.

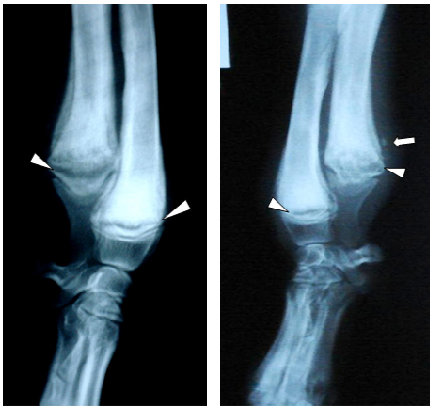

Figure 2 - X-rays of the left (MTE) and right (MTD) forelimb during HOD acute phase, showing radiolucent areas in the metaphysis of the radius and ulna (triangular arrows) and bone formation in the caudal portion of the ulnar metaphysis (long arrow).

Figure 3 - The 1-year old Lord, healthy, showing no signs of recurrence.

REFERENCES

ANDRADE, S. F.; JERICÓ, M. M. Antinflamatórios. In: Manual de Terapêutica Veterinária. 2.ed, Editora Roca, São Paulo, SP, cap.7, p.89-92, 2002.

BELLAH, J. R. Hypertrophic osteodystrophy. In Bojrab MJ, editor: Disease mechanisms in small animal surgery. 2.ed, Philadelphia, Lea & Fe 1993.

BRAWNER JR, W. R. et al. The role of. diagnostic imaging in assessment of canine skeletal development. In: REINHART G. A, CAREY, D. P. (Ed) Recent advances in canine and feline nutritional research: 2000 Iams nutrition symposium proceedings. Wilmington:Orange Frazer Press, v.2, p.13- 8, 1998.

CANOLA, J. C.; ANDRADE, A. L. Clinical and radiographic aspects of hypertrophic osteodystrophy in the dog: a retrospective study of sixteen cases. Ciência Rural: Santa Maria, vol.26, n.3, 1996.

CARNEIRO, S. C. M. C.; FERREIRA, R. P.; FIORAVANTI, M. C. S. et al. Superalimentação desenvolvimento do esqueleto de cães da raça Dogue Alemão: aspectos clínicos e radiográficos. Arq.Bras.Med.Vet.Zootec. vol.58, n.4, p.256-260, 2006.

COOK, J. L. Forelimb lameness in the young patient. Vet. Clin. North Am.: Small Anim. Pract., v.31, n.1, p.55-83, 2001.

DAMMRICH, K. Relationship between nutrition and bone growth in large and giant dogs. J. Nut., Bethesdo, v.121, p.114-121, 1991.

FABRIS, L. G.; Osteodistrofia em cães. Disponível em: http://www.redvet.com.br/ doencas/osteodis.htm. Acesso em 30 de outubro de 2013.

FOSSUM, T. W. Outras osteopatias e artropatias. In: Cirurgia de pequenos animais, 2.ed., Editora Roca:São Paulo, SP. Cap.37, p.1164-1165, 2005.

GRONDALEN, J. Metaphyseal osteopathy (hypertrophic osteodystrophy) in growing dogs. A clinical study. J Small Anim Pract 17:721-735, 1976.

HAZEWINKEL, H. A. W.; MEIJ, B. P.; THEYSE, L. F. H. Asynchronous growth of the radius and ulna in the dog. In: Clinical Nutrition Symposium, XXIII CONGRESS OF. THE WORLD SMALL ANIMAL VETERINARY ASSOCIATION, 1998. Buenos Aires – Argentina, v.6, p.05-14.

JOHNSON K. A., WATSON A. D. J.; PAGE R. L. Afecções esqueléticas. In: Tratado de medicina interna veterinária, Editora Manole Ltda, 4.ed., v.2, cap150, p.2883-2885, 1997.

LENEHAN, T. M, FETTER, A. W. Hypertrophic osteodystrophy. In: NEWTON, C. D., NUNAMAKER, D. M. (ed): Textbook of small animal orthopaedics. Philadelphia: Lippencott, p.597, 1985.

MEYER, H.; ZENTEK, J. Energy requirements of growing Great Danes. J. Nut., Bethesda, v.121, p.35-36, 1991.

OWENS, J. M.; BIERY, D. N. Principles of Radiographic Interpretation. In: OWENS, J. M.; BIERY, D. N. Radiographic interpretation for the small animal clinician 2ed. Williams & Wilkins, cap.2, p.9-11, 1999.

RICHARDSON, C. D.; ZENTEK, J. Nutrition and osteochondrosis. Vet. Clin. North Am.: Small An. Pract., Philadelphia, v.28, n.1, p.115-135, 1998.

TEARE, J. A.; KROOK, L.; KALLFELZ, F. A. Ascorbic acid deficiency and hypertrophic osteodystrophy in the dog: a rebuttal. Cornell Vet, v.69, p.384-401, 1979.

THRALL, D. E. Textbook of Veterinary Diagnostic Radiology, 3.ed., Philadelphia: Saunders Company, 663p, 1998.