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Conservative management of hip dysplasia in a dog

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Abstract
A 2 year old male Rottweiler dog was presented with the history of abnormal gait, intermittent lameness in its hind legs, reluctant to climb stairs, difficulty in getting up after a period of rest and exercise intolerance. The case was suspected for hip dysplasia on the basis of history and clinical signs. Radiographic examination revealed bilateral hip dysplasia. The case was clinically managed successfully by use of chondro-protective agents, non-steroidal anti-inflammatory drugs (NSAID), weight reduction and monitored exercise.

Keywords: Hip dysplasia, dogs, radiography

Introduction
Canine Hip dysplasia (CHD) is a commonly presented orthopaedic disease in dogs and is potentially debilitating. CHD is a complex developmental disorder which is characterized by joint laxity and osteoarthritis (OA) in one or both coxofemoral (hip) joints (Lust, 1997) [11]. The condition can affect dog of any breed but it appears to occur at a relatively high rate in large-bodied and brachycephalic dogs as well as those with high body length to height ratios (Roberts and McGreevy, 2010) [16]. Factors such as diet type, exercise and genetics together play a role in development of CHD. The diagnosis of the disease is based on history, clinical signs and radiography. Treatment of CHD is either by conservative management or surgical intervention. Conservative management can be used in dogs with mild or intermittent clinical signs and includes nutritional management and weight control, exercise modification, physical rehabilitation, pain management and disease-modifying agents(Harper TAM, 2017) [8]. The present article describes a clinical case of hip dysplasia and conservative management that has been followed.

Materials and Methods
Case history
A male Rottweiler dog, aged 2 years, weighing 33 kilograms was presented to Eden Pets Care, Pet clinic, Mogappair, Chennai with the history of abnormal gait, intermittent lameness in its hind legs, reluctant to climb up stairs, difficulty in getting up after a period of rest and exercise intolerance which was observed for a period of 2 weeks. The animal was dull on presentation, with intermittent limping. Hind limb muscles showed muscle atrophy and back legs were unnaturally close together with narrow stance.

Diagnosis
Radiography has long been the gold standard to assess and quantify joint changes associated with CHD joint remodelling (Risler *et al.*, 2009) [15]. Radiographic examination on ventro dorsal view revealed shallow acetabulum, osteoarthritic changes with osteophyte formation.

Results and Management
The radiographic findings (Fig. 1) were confirmative of hip dysplasia. It was decided to clinically manage the case by conservative method. Hence the dog was treated with tablet cosequin (chondroitin sulphate, Hyaluronic acid & Glucosamine) for two months and tablet carprofen at the dose of 4.4mg/Kg for a period of 14 days. Cosequin was administered at the dose 3 tabs daily for four weeks followed by 2 tablets for the next four weeks. The dog was provided with a veterinary prescription diet for obesity and owner was advised to avoid any

junk foods being provided to the pet. Better clinical improvement was seen with no lameness and improved activity in the dog. The importance of continuing the use of chondroprotective agents in a maintenance dose and maintenance of optimum weight was emphasized to the pet parent.

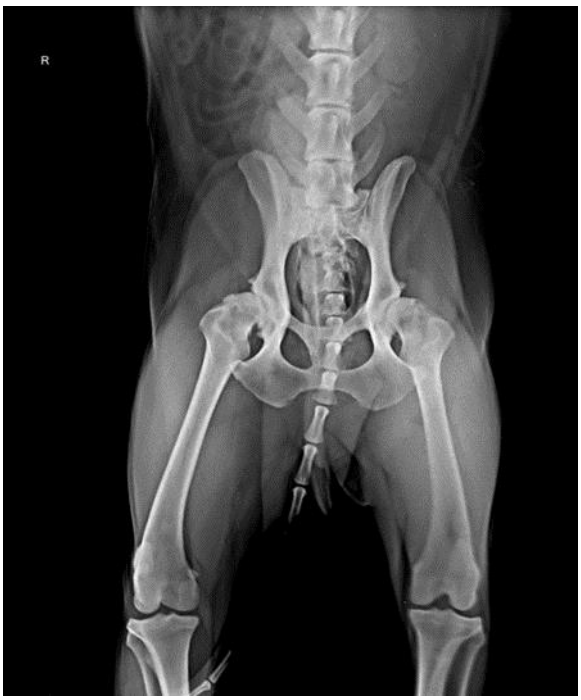

Fig 1: Radiography showing Hip Dysplasia

Discussion

CHD is characterised by the abnormal development of the coxo-femoral joint. CHD is characterized by subluxation or complete luxation of the femoral head in young animals and as a mild to severe degenerative joint diseases in older animals (Fossum, 2007 [5]; Slatter, 2003 [17]). The development of CHD has been associated with the abnormal and delayed endochondral ossification in the coxo-femoral joint which has been identified in 15-day-old dogs that developed CHD by the time they were 12 months old (Madsen *et al*., 1991) [12]. Dogs with dysplasia generally exhibit two general behaviours, lameness in young dogs under 1 year of age that increases with activity or trauma, and characterised by gait abnormalities and hind limb muscle atrophy in older dogs (Emma and Mandi, 2015) [3]. Hind limb lameness should be differentially diagnosed from pelvic, distal hind limb, and neurological pathologies, metabolic bone disease, ligament rupture, patellar luxation, and spine disorders (Fry and Clark, 1992) [7].

Several factors contribute together to the development of hip dysplasia in dogs, diet, exercise and genetic factors. Fries and Remedios (1995) [6] stated that when a dog has extra weight, it can lead to proliferation of the dorsal acetabular rim, atrophy of local muscles, stretching of ligaments in the femoral head, cartilage degeneration, and thickening of the joint capsule and femoral neck.

Conservative management of CHD generally consists of a combination of mechanisms to reduce progression of joint damage and alleviate discomfort (Kirkby and Lewis, 2012) [10]. The understanding of expression of certain genes which determine the development of hip joint shows the heritable relationship between parents and offspring. 85% of offspring have CHD when both parents have dysplastic hips, 52% of offspring if one parent has dysplastic hips, and 37.5% of offspring if both parents have normal hips (Fries and Remedios, 1995) [6]. This study has demonstrated the importance of genetic diversity in preventing the spread of hip dysplasia.

Three predominant genes, carbohydrate sulfotransferase 3 (CHST3), fibronectin 1, and fibrillin 2 (FBN2) have been found association in development of Hip dysplasia. In canines with hip dysplasia, a missense mutation in exon 3 of CHST3 causes deficiencies in the formation of joint cartilage leading to hip malformation (Tanteles *et al.*, 2013) [18].

Fibronectin 1 is an important glycoprotein that plays a role in the extracellular matrix of cartilage which is present in small amount in canines with normal hips, whereas it is present in large amounts in hip joint of dysplastic dogs. The accumulation of fibronectin in dysplastic hips may be due to the loss of extracellular matrix in the joint, which causes a build-up of this glycoprotein in the cartilage (Wurster and Lust 1982) [19].

Fibrillin 2 is a structural gene which plays a vital role in elastic tissue of extracellular matrix. This assembles to become a framework of elastic fibers and provides a place for growth factors to stimulate cellular growth. In canines with dysplastic hips there is a deletion of the fibrillin 2 gene on chromosome 11, which causes alterations in the connective tissue around the joint, leading to hip malformation (Zhu *et al.*, 2012) [20].

Conservative management of CHD is aimed to reduce progression of joint damage and alleviate discomfort. (Kirkby and Lewis, 2012) []. It is often employed to manage signs of CHD, with lifelong maintenance of body mass as one of the most promising methods (Panigrahi, 2014) [13]. This effectively reduces the signs associated with dysplasia and osteoarthritis (OA) (Impellizeri *et al.*, 2001) [8]. Kealy *et al.*, (2000) [9] stated that a lifelong dietary restriction of 25% delayed the appearance of OA as well as the intensity of clinical signs in Labrador Retrievers compared with feeding ad libitum.

Glucosamine hydrochloride (HCl) and chondroitin sulphate (CS) are commonly recommended natural health products for treating osteoarthritis in dogs (Bhathal *et al.*, 2017) [1]. Drugs such as non-steroidal anti-inflammatory drugs (NSAIDs) are commonly used for pain associated with severely affected arthritic joints in dogs (Pollmeier *et al.*, 2006) [14]. Surgical intervention is often employed to prevent joint changes or restore joint function Some surgical procedures designed to prevent onset of OA in hips identified as being predisposed to development of OA include double and triple pelvic osteotomy, acetabular shelf and excision arthroplasty, femoral osteotomy, and juvenile pubic symphysiodesis (Dueland *et al.*, 2017) [2]. Surgical procedure such as Total hip replacement is often applied in advanced cases of joint degeneration and is considered a salvage procedure (Fitzpatrick *et al.*, 2014) [4].

Conclusion

It is understood that though the genetic components also determines development of CHD, a control of environmental factors can help in effective management of dogs presented with dysplasia. It is also important for the dog owners to have the proper knowledge about their canine to ensure they are feeding them a proper diet and giving them the appropriate amount of exercise.

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