J. Vet. Malaysia (1999) 11(1): 1-5

CANINE HIP DYSPLASIA: PATHOGENESIS AND DIAGNOSIS

H.G. Heng

Faculty of Veterinary Medicine
Universiti Putra Malaysia
43400 Serdang, Selangor, Malaysia

SUMMARY

Canine hip dysplasia (CHD) is a hereditary disease that affects large-breed dogs. Affected young animals usually exhibit clinical signs associated with subluxation of the femoral head, while older dogs is associated with secondary degenerative joint disease (DJD). There is poor correlation between the radiographic changes and the clinical signs. Thus, diagnosis of CHD should not be based either on physical examination or radiography alone, but a combination of both.

Keywords: Canine, hip dysplasia, pathogenesis, diagnosis

INTRODUCTION

Hip dysplasia literally means abnormal development of the hip. The word dysplasia is from Greek words "dys" meaning abnormal and "plassein" meaning to form (Morgan and Stephens, 1985). Canine hip dysplasia (CHD) is a common developmental problem affecting canine population, first described by Schnelle (1935). It is characterised by varying degrees of hip joint laxity, subluxation and subsequent degenerative joint disease but remained as a complex disease with many unanswered questions and numerous misconceptions among the general public (Fries and Remedios, 1995).

The Orthopaedic Foundation for Animals has identified more than 42 canine breeds affected by hip dysplasia (Bloomberg, 1990). The large and giant breeds are at greatest risk and have a greater incidence compared to the general population (Corley and Hogan, 1985). Canine hip dysplasia accounted for up to 30% of orthopaedic cases with hind limb lameness at a major university veterinary teaching hospital (Richardson, 1992) with males and females are affected with equal frequency (Fries and Remedios, 1995). It usually occurs bilaterally although it can also occur unilaterally (Bloomberg, 1990).

PATHOGENESIS

The aetiology of CHD is multi-factorial involving both genetic and environmental factors. For canines that are genetically predisposed to hip dysplasia, changes begin to occur within the first 60 days after birth (Alexander, 1992). The genotype determines the genetic blueprint for shape, size, anatomical relationships, musculature, innervation and the growth and remodelling programmes of the hip. Nutritional and other environmental factors probably influence the expression of genes in determining the phenotype for CHD (Fries and Remedios, 1995; Tomlinson and McLaughlin, 1996). However, the exact cause of CHD is unknown and the progression of the diseased hip joint is probably similar regardless of the aetiology (Morgan and Stephens, 1985).

The critical period for the development of the hip joint is from birth to 2 months of age. At this stage, the bones have not fully formed from their cartilage models, the muscles and nerves have not fully developed while the soft, plastic tissues of the hip can be strained beyond their elastic limit (Alexander, 1992). Thus, subluxation of the hip joint, one of the early pathological changes in CHD (Morgan, 1987) can be detected radiographically as early as 4 to 8 weeks of age (Henry, 1992).

The age at which hip joint instability first occurs will determine the pattern of changes within the acetabulum and the femoral head. The greater the amount of soft cartilage present at the time of joint laxity, the greater changes in the acetabulum and the femoral head will occur. This will lead to a more important consequence, which is the degree of hip joint luxation. The more extensive the subluxation, the less contact the femoral head has with the acetabular rim and consequently, less remodelling occurs (Morgan and Stephens, 1985).

As dogs with CHD grow, the disease progresses to secondary degenerative joint disease (DJD) due to the joint incongruity and instability. Degenerative joint disease is a condition characterised by alterations in joint architecture due to cartilage degeneration leading to misdirected bone and cartilage growths (Alexander, 1992). The basic pathological lesion in DJD is a progressive disintegration of articular cartilage of the hip joint. The primary causes of DJD are poorly understood and may be related to ageing, wear and tear and heredity. Secondary DJD may occur following

trauma, overload, joint instability, acute inflammatory joint disease, infection or some other primary bone disease such as osteochondritis dissecans or osteomalacia. Once triggered, it continues in a cyclic manner unless arrested by the ability of chondrocytes to synthesise sufficient matrix components (Clyne, 1987).

Damage to the chondrocytes often leads to acute synovitis and capsulitis. As a result, leukocytes, prostaglandins, lysosomal enzymes and hyaluronidase enter the synovial fluid causing the synovial fluid to become less viscous while the transit path for cartilage nutrition is disrupted. Proteoglycans in the cartilage matrix undergo a variety of changes, probably due to the chondrocyte damages caused by lysosomal enzymes or due to the damages of collagen fibres. Changes to the remaining proteoglycans lead to the swelling of the cartilage by imbibing excess water. Thus, the biomechanical properties of the cartilage are changed, rendering both matrix and chondrocytes more susceptible to mechanical stress and enzyme attack (Morgan and Stephens, 1985; Alexander, 1992).

The loss of normal proteoglycans leads to an increased chondrocyte activity to replenish the proteoglycans. However, the newly synthesised proteoglycans are of lower molecular weight, have an altered glycosaminoglycan composition and hence, do not readily form aggregates. As a result, there is a loss of cartilage elasticity and an alteration in its shock-absorbing capacity, leading to increased friction causing blistering and ulceration (Morgan and Stephens, 1985; Alexander, 1992). Further changes in the cartilage occur because of lysosomal enzyme released from the chondrocytes, resulting in matrix destruction and further proteoglycans degradation. Once blistering and ulceration occur, the degrading enzymes from the synovial fluid enter the matrix causing further degradation. The prostaglandins suppress glycosaminoglycan synthesis while the hyaluronidase degrades both hyaluronic acid and chondroitin sulphate and other lysosomal enzymes contribute to further degradation of both matrix and proteoglycans (Morgan and Stephens, 1985).

Similarly in CHD, once the cartilage model of the acetabulum lost the congruity owing to subluxation of the femoral head, secondary DJD will occur. With continuing subluxation of the hip joint, only a small area of the articular surfaces of both the femoral head and acetabulum are actually in contact to each other. The concentration of the hip load and continued overloading on the acetabular rim are believed to produce fatigue, loss of tissue elasticity and contour and eventually, micro fractures (Alexander, 1992). Once the micro fractures occur, the acetabular cup loses its curvature and shape, causing the frayed ends at the margin to turn upward and backward. Sharpey's fibres (attachment of soft tissue to bone) rupture and bleed, which stimulates osteophyte formation around the acetabulum (Alexander, 1992). One of the hallmarks of secondary DJD in CHD is marginal formation of osteophytes. These osteophytes may be formed within the insertion lines of the joint capsule, ligaments or tendons (Morgan, 1987) and can be detected radiographically when the dog is about 20 to 36 weeks old (Henry, 1992).

DIAGNOSIS

Correct diagnosis of CHD is very important before a decision on treatment can be made. Final diagnosis is based on both physical examinations and radiography, not by radiography alone (Bloomberg, 1990).

Physical examination

Lameness is the most common clinical sign of CHD. The presentation and severity of clinical signs, however, vary widely. There appears to be poor correlation between the morphology of the hip and the severity of clinical lameness (Pharr, 1978). Two age classifications are used in describing the types of clinical signs; animals less than 1 year of age and animals greater than 1 year of age (Fry and Clark, 1992).

Affected young dogs usually have acute episodes of bilateral or occasionally unilateral pelvic limb lameness that are exacerbated by vigorous exercise or relatively minor trauma. Initial clinical signs include difficulty in rising, walking, running and climbing stairs. The animal objects to palpation or petting of the hindquarters or growls when approached by children (Morgan and Stephens, 1985; Fry and Clark, 1992). The clinical signs observed in young dogs are attributed to the distension of the joint capsule from excessive synovial fluid, synovitis, tearing or stretching of the round ligament and joint capsule and microfracture of the cranial acetabular edge. These are due to the joint laxity or subluxation of the femoral head (Burns *et al.*, 1987; Tomlinson and McLaughlin, 1996).

Many clinicians believe that there is an intermediate stage in the disease, during which stage no clinical signs are observed prior to the onset of substantial DJD. Dogs in this stage walk and run without pain. The development of DJD heralds the return of clinical signs in these animals (Fry and Clark, 1992).

Older dogs exhibiting lameness frequently shift weight to their thoracic limbs as a result of hip joint discomfort. In time, the thigh muscles atrophied because of their restricted use while the shoulder muscles hypertrophied. Stiffness, difficulties in

advancement of the limbs and hindquarter weakness contribute to a noticeable disturbance in movement of the hind limbs and a shortened stride. They often prefer sitting to standing and are reluctant to exercise (Morgan and Stephens, 1985; Fry and Clark, 1992). These clinical signs are typically due to the resultant secondary DJD.

Examination of the hip joint begins while the patient awake but general anaesthesia is required for full assessment (Fry and Clark, 1992; de Hann *et al.*, 1993). The patient is observed at rest and during exercise.

A complete musculoskeletal examination should be performed. Gait abnormalities may not be a sensitive indicator of CHD (Pharr, 1978). However, "bunny hopping" while running frequently manifested in dogs with CHD because this is a more comfortable way to run by increasing motion of the spine and decreasing the motion in the hip joint (Morgan and Stephens, 1985).

The range of cranio-caudal motion of the hip joint should be measured. The normal range in anaesthetised dogs is 110 degrees. In dogs with severe DJD, the range of motion can be reduced to as little as 45 degrees, particularly the extension component (Fry and Clark, 1992; de Hann *et al.*, 1993).

Dogs with CHD exhibit pain on forced extension of the affected hips. The patients exhibit discomfort, splinting of the pelvic musculature, rapid advancement of the affected limb to a more flexed position and, in some cases, vocalisation or aggression in response to pain (Fry and Clark, 1992).

Demonstration of hip joint laxity is one of the criteria used for diagnosis of CHD. Several methods are used but laxity in itself is not necessarily indicative of CHD (Fry and Clark, 1992). The Ortolani test is a common physical manipulation to assess the severity of dorsal subluxation in patients with CHD. This test can be performed with the dog either awake or under sedation but in most instances, general anaesthesia is necessary to demonstrate the Ortolani sign adequately (Fry and Clark, 1992; de Haan *et al.*, 1993). Patients with a subluxated hip joint will have a palpable and often audible "click" or "pop" during the abduction phase of the examination. This click occurs as the subluxated femoral head is re-seated into the acetabulum. This is known as the Ortolani sign. The Ortolani sign is not present in normal hip joints (Chalman and Butler, 1985).

Patients with severe hip dysplasia may not produce the Ortolani sign. In these patients, gross destruction of the dorsal acetabular rim, thickening of the joint capsule, proliferation of osteophytes, limited range of motion and possible permanent luxation of the femoral head exist in a varying degree. Absence of the acetabular rim does not permit relocation of the femoral head and therefore, no clicking sound. Osteophyte formation and calcification within the joint capsule produce crepitus, which should not be confused with a positive Ortolani sign (Chalman and Butler, 1985). A negative Ortolani test, however, does not rule out CHD.

Young puppies with normal hip joints occasionally have an Ortolani sign due to the immaturity of the soft tissue supporting the hip joints. These puppies should be examined intermittently for evidence of improving hip stability and loss of the Ortolani sign (de Haan *et al.*, 1993).

The Barlow test is an additional physical examination parameter that gives the clinician an indication of hip laxity. It is conducted after the femoral head has been re-seated within the acetabulum. A positive Barlow sign is obtained when the reduced femoral head subluxates again following adduction of the hip. The angle of subluxation is obtained between the axes as in the Ortolani test (de Haan *et al.*, 1993). It is possible to detect coxofemoral laxity using this examination alone, but is recommended to perform with the Ortolani test to increase the reliability of the findings (Fry and Clark, 1992).

Radiography

Radiography is generally accepted as the only definitive method of diagnosing CHD after the musculoskeletal examination has been performed on the clinical CHD patients (Morgan and Stephens, 1985). Standardisation of the radiographic view by the Orthopaedic Foundation for Animals (OFA) in 1966 contributed significantly to the use of radiographic evaluation of the canine hip joint in the diagnosis and control of CHD (Henry, 1992).

The hip-extended radiographic view with the dog in dorsal recumbency (Fig. 1) has been the most commonly used radiographic position since the American Veterinary Medical Association (AVMA) panel on CHD recommended its use in 1961 (Henry, 1992; Heyman *et al.*, 1993). Chemical restraint is recommended in order to give veterinarians the best conditions for holding the dog in the required position (Corley, 1992). In young animals, the earliest radiographic sign of CHD is subluxation of the femoral head (Burns *et al.*, 1987; Morgan, 1987) (Fig. 2). As the disease progresses, radiographic changes observed are those of DJD, characterised by osteophytes formation at the insertion of joint capsule, re-modeling of the acetabulum, the femoral head and neck and sclerosis of subchondral bone of the femoral head and acetabulum (Burns *et al.*, 1987) (Fig. 3).

There are many methods in assessing the radiographs; the subjective, objective and semi-

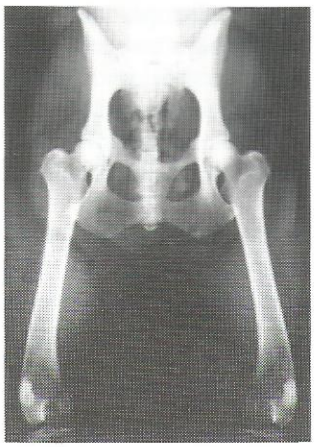

Fig. 1. The hip-extended radiographic view of a dog with normal hip joints

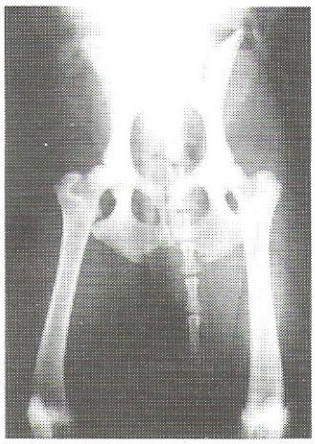

Fig. 2. The hip extended radiographic view of a dog with subluxation of the femoral head

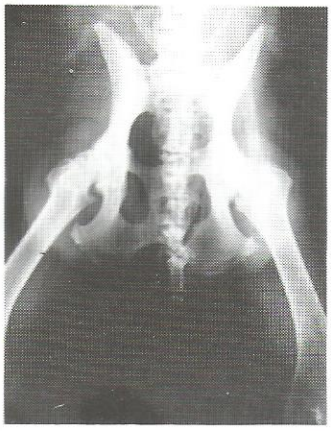

Fig. 3. The hip extended radiographic view of a dog with severe secondary DJD

quantitative methods. However, the basis of the diagnosis is still based either on the presence of hip subluxation, DJD or both (Smith and McKelvie, 1995).

Stress radiography was introduced in the early 90s (Smith *et al.*, 1990). This technique is based on the premise that joint laxity is the earliest sign of CHD (Burns, *et al.*, 1987; Morgan, 1987) and is a risk factor for the development of DJD (Smith *et al.*, 1995). Two radiographs are needed in stress radiography; the compression and distraction views. Sedated dogs are placed on dorso-ventral position with hips in the neutral orientation. To obtain a compression view, a medially directed compressive force is applied to the trochanteric area in order to firmly seat the hips in their most congruent position for the radiographic exposure. The distraction view is made with hips in the same neutral orientation but with an adjustable distractor positioned between the hind limbs and a distractive force is applied to determine maximum passive laxity. Hip laxity is quantified by means of the distraction index. The compression view serves as a reference to identify landmarks used in determining the distraction index (Smith *et al.*, 1990; Smith *et al.*, 1995).

In 1993, the University of Pennsylvania Hip Improvement Program (PennHIP) was established based on the stress radiography. This program is aimed at detecting CHD at an earlier stage than is previously possible using the current radiographic schemes and to reduce the frequency of CHD (Madson and Svalastoga, 1995). Currently, studies are being undertaken to determine the effectiveness of stress radiography as a screening test in breeding programmes and the heritability of the distraction index for different breeds affected by CHD (Smith and McKelvie, 1995).

REFERENCES

Alexander, J.W. (1992). The pathogenesis of canine hip dysplasia. *Vet. Clin. North Am. Small Anim. Pract.* **22**: 503-511.

Bloomberg, M.S. (1990). Canine hip dysplasia: What's old, what's new, what's true. *Vet. Tech.* **11**: 303-309.

Burns, J., Fox, S.M. and Burt, J. (1987). Diagnostic radiography: the only definitive determination of CHD. *Vet. Med.* **82**: 694-700.

Chalman, J.A. and Butler, H.C. (1985). Coxofemoral joint laxity and the Ortholani sign. *J. Am. Ani. Hlth. Assoc.* **21**: 671-676.

Clyne, M.J. (1987). Pathogenesis of degenerative joint disease. *Equine Vet. J.* **19**: 15-18.

Corley, E.A. (1992). Role of the Orthopedic Foundation for Animal in the control of canine hip dysplasia. *Vet. Med. North Am. Sm. Anim. Prac.* **22**: 579-593

Corley, E.A. and Hogan, P.M. (1985). Trends in hip dysplasia control: analysis of radiographs submitted

to the Orthopedic Foundation for Animals, 1974-1984. *J. Am. Vet. Med. Assoc.* **187**: 805-809.

de Haan, J.J., Beale, B.S. and Parker, R.B. (1993). Diagnosis and treatment of canine hip dysplasia. Part 1 and Part 2. *Canine Prac.* **18**: 24-28, 28-32

Fries, C.L. and Remedios, A.M. (1995). The pathogenesis and diagnosis of canine hip dysplasia : A review. *Can Vet. J.* **36**: 494-502.

Fry, T.R. and Clark, D.M. (1992). Canine hip dysplasia: clinical signs and physical diagnosis. *Vet. Clin. North Am. Small Anim. Pract.* **22**: 551-558.

Henry, G.A. (1992). Radiographic development of canine hip dysplasia. *Vet. Clin. North Am. Small Anim. Pract.* **22**: 559-578.

Heyman, S.J., Smith, G.K. and Cofore, M.A. (1993). Biomechanical study of the effect of coxofemoral positioning on passive hip joint laxity in dogs. *Am. J. Vet. Res.* **54**: 210-215.

Madsen, J.S. and Svalastoga, E. (1995). Early diagnosis of hip dysplasia – A stress-radiographic study. *V.C.O.T.* **8**: 114-117.

Morgan, J.P. (1987). Canine hip dysplasia: Significance of early bony spurring. *Vet. Radio.* **28**: 2-5.

Morgan, J.P. and Stephens, M. (1985). *In:* Radiographic Diagnosis and Control of Canine Hip Dysplasia. Iowa State University Press, Ames, USA, 145pp.

Pharr, J.W. (1978). Canine hip dysplasia, yet another radical view. *Vet. Clin. North Am. Small Anim. Pract.* **8**: 309.

Richardson, D.C. (1992). The role of nutrition in canine hip dysplasia. *Vet. Clin. North Am. Small Anim. Pract.* **22**:529-540.

Schnelle, G.B. (1935). Cited by Henry, G.A. (1992) in Radiographic development of canine hip dysplasia. *Vet. Clin. North Am. Small Anim. Pract.* **22**: 559-578.

Smith, G.K., Biery, D.N. and Gregor, T.P. (1990). New concepts of coxofemoral joint stability and the development of a clinical stress-radiographic method for quantitating hip joint laxity in dog. *J. Am. Vet. Med. Assoc.* **196**: 59-70.

Smith, G.K. and McKelvie, P.J. (1995). Current concepts in the diagnosis of canine hip dysplasia. *In*: Kirk's Current Veterinary Therapy, Small Animal Practice. Bonagura, J.D. (Ed.) XII ed., W.B. Saunders Co., Philadelphia. pp1180-1187.

Smith, G.K., Popovitch, C.A., Gregor, T.P. and Shofer, F.S. (1995). Evaluation of risk factors for degenerative joint disease associated with hip dysplasia in dogs. *J. Am. Vet. Med. Assoc.* **206**: 642-647.

Tomlinson, J. and McLaughlin, R. (1996). Medically managing canine hip dysplasia. *Vet. Med.* **91**: 48-53.

RINGKASAN

DISPLASIA PINGGUL KANIN: PATOGENESIS DAN DIAGNOSIS

Displasia pinggul kanin (CHP) merupakan penyakit terwaris yang melibatkan anjing-anjing baka besar. Anjing-anjing muda yang terlibat selalunya memaparkan beberapa petanda klinikal berkaitan dengan subkehelan pada kepala femur, sambil pada anjing-anjing tua pula, petanda klinikalnya berkaitan dengan penyakit sendi jana rosot (DJD). Kolerasi di antara perubahan radiografi dengan petanda klinikal adalah buruk. Dengan ini, diagnosis untuk CHD tidak boleh berasaskan kepada sama ada pemeriksaan fizikal atau radiografi sahaja, tetapi mesti berasaskan kepada kedua-duanya sekali