

ISCHEMIC NECROSIS OF THE FEMORAL HEAD IN DOGS AND SOME CONSIDERATIONS IN HUMAN BEINGS

Necrosis isquémica de la cabeza femoral en caninos y algunas consideraciones en seres humanos

Manuel Alvarado Morillo

Surgical Medical Department
Faculty of Veterinary Sciences
University of Zulia. P.O. Box 526
Maracaibo 4001, Venezuela

ABSTRACT

Ischemic necrosis of the femoral head (I.N.F.H.) is a disease found primarily in small or dwarf dogs, characterized by ischemia induced necrosis and ultimate collapse of the subchondral bone in the head of the femur [15]. There is a disease in human being (Legg-calve-Perthes Syndrome) which has many similarities to the canine disease [7]. Most of the children affected are small for their age and often were born premature. The age of onset of clinical signs in children is usually between three and five years old. In dogs, the affected animals are presented at about four to ten months of age. It is likely, however, that the initiating incident occurs much earlier, around 6-12 weeks of age in dogs, because it takes time for the dead bone to lose its rigidity and to, finally, collapse. In dogs, the number of males and females affected is about equal. In contrast to this, the ratio in Legg-Calve-Perthes Syndrome is four boys to one girl. An explanation advanced for this difference is that boys are more active than girls and therefore, are subject to greater trauma. In the canine, the sex of the puppy does not as greatly influence the amount of activity engaged in.

Key words: Ischemic necrosis, femoral head, canine, human being.

RESUMEN

La necrosis isquémica de la cabeza femoral (N.I.C.F.) es una enfermedad que se presenta principalmente en perros de razas pequeñas o con proporciones enanas, caracterizada por una isquemia que conlleva a la necrosis y posteriormente al

colapso del hueso subcondral en la cabeza del fémur. Existe una enfermedad en los seres humanos (Síndrome de Legg-Calve-Perthes), la cual es muy similar a la enfermedad que se presenta en los caninos. La mayoría de los humanos afectados son niños pequeños en relación a la edad y casi siempre han nacido prematuramente. La edad de aparición de los signos clínicos en los niños es usualmente entre los tres y cinco años. En los animales afectados se presenta entre los 4 y 10 meses de edad. Sin embargo, es probable que el inicio del problema ocurra mucho más temprano, alrededor de 6 a 12 semanas de edad, ya que toma algún tiempo al hueso isquémico, perder su rigidez, y finalmente colapsar. En los perros, el número de machos y hembras afectados es casi similar. En contraste, la relación en el Síndrome de "Legg-Calve-Perthes" es de 4 varones por cada hembra. Una explicación para estas diferencias es que los niños son mucho más activos que las niñas y por lo tanto están sujetos a tener más traumas. En los caninos, el sexo de los cachorros no tiene una gran influencia con respecto a la actividad que ellos desarrollan.

Palabras clave: Necrosis isquémica, cabeza femoral, canino, seres humanos.

INTRODUCTION

Ischemic necrosis of the femoral head (I.N.F.H.) was first seen in humans when radiography was developed [3] in 1937. Moltzen-Nielsen first described a similar condition to Legg-Calve-Perthes Syndrome in the dog. Many theories of the etiology of this disease have been advanced. Some of this theories include: 1) juvenile osteoarthritis, 2) embolism, 3) acute or

chronic repeated trauma, 4) congenital and hereditary factors, 5) infections, and 6) endocrine disorders [1].

Embolism and trauma are mechanisms by which blood supply can be compromised. This may be the basis for evidence in support of these theories. Congenital and hereditary factors were considered because of the similarity of this disease to Morquio's and Hurler's diseases, a disease of mucopolysaccharide metabolism. Morquio's and Hurler's diseases have not been demonstrated in dogs [2]. Infections were also thought to play a part in the INFH disease in animals and in humans; until 1910, ischemic necrosis was confused with tuberculosis of the hip [3]. Ljunggren [10] thought the INFH was related to early closure of the cartilaginous growth plate, possibly under hormonal influence. Perthes, one of the men who first studied this disease, originally suggested the ischemic nature of the disease [10].

Ischemic necrosis of the Femoral Head (INFH) occurs in most cases as a unilateral condition [14]. In a few cases, both femoral heads are affected. A review of case histories from University of Pennsylvania Veterinary Clinic showed five cases to be bilateral of the forty-eight seen and The University of Zulia, Veterinary Policlinic showed 16 cases to be unilateral and one to be bilateral. FIG. 1.

The disease is characterized initially by an intermittent limp. Without treatment, the lameness becomes progressively worse. Some weight is borne on the limb when walking but in the earlier stages the limb is carried when the animal runs. In the end stage, the affected limb is carried at all gaits. The animals show resentment and pain upon palpation of the

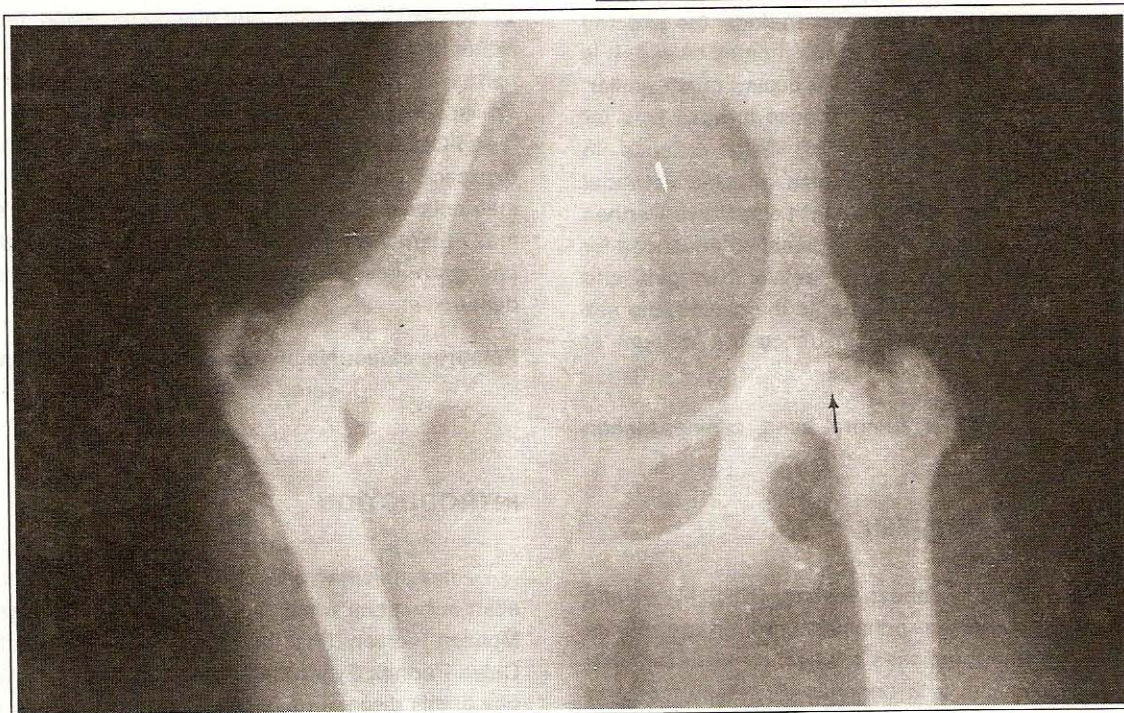
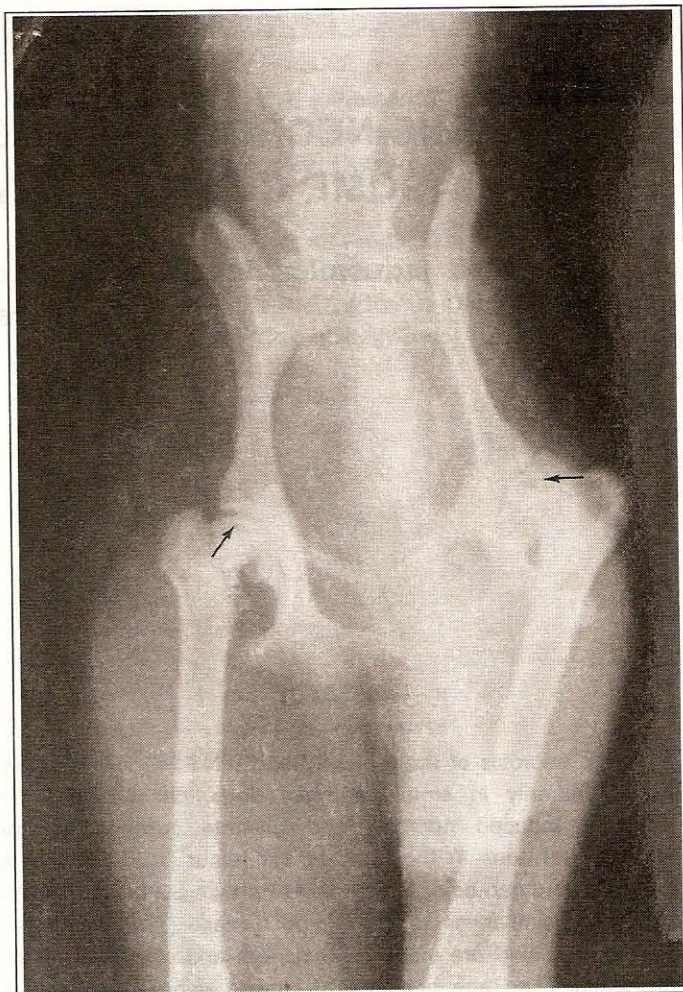


FIGURE 1. VENTRO DORSAL RADIOGRAPHS OF THE PELVIS IN A SEVEN MONTHS OLD DOG WITH ISCHEMIC NECROSIS OF BOTH FEMORAL HEADS (ARROWS).

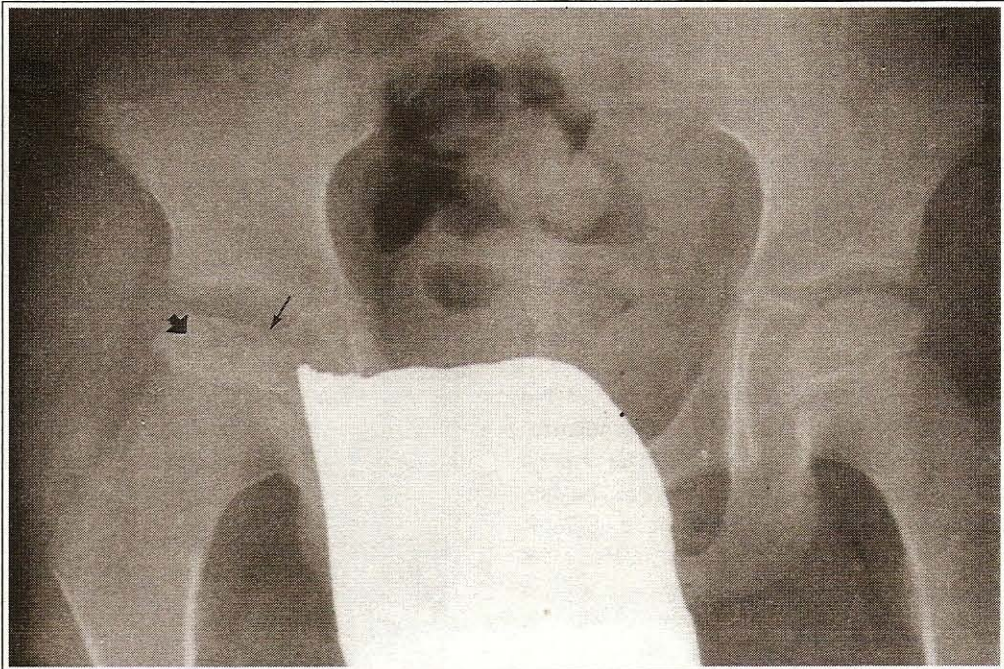


FIGURE 2. LEGG-PERTHES DISEASE OF THE RIGHT HIP IN A SEVEN YEARS OLD BOY. NOTICE THE IRREGULAR CAPITAL EPIPHYSIS AND DENSITY.

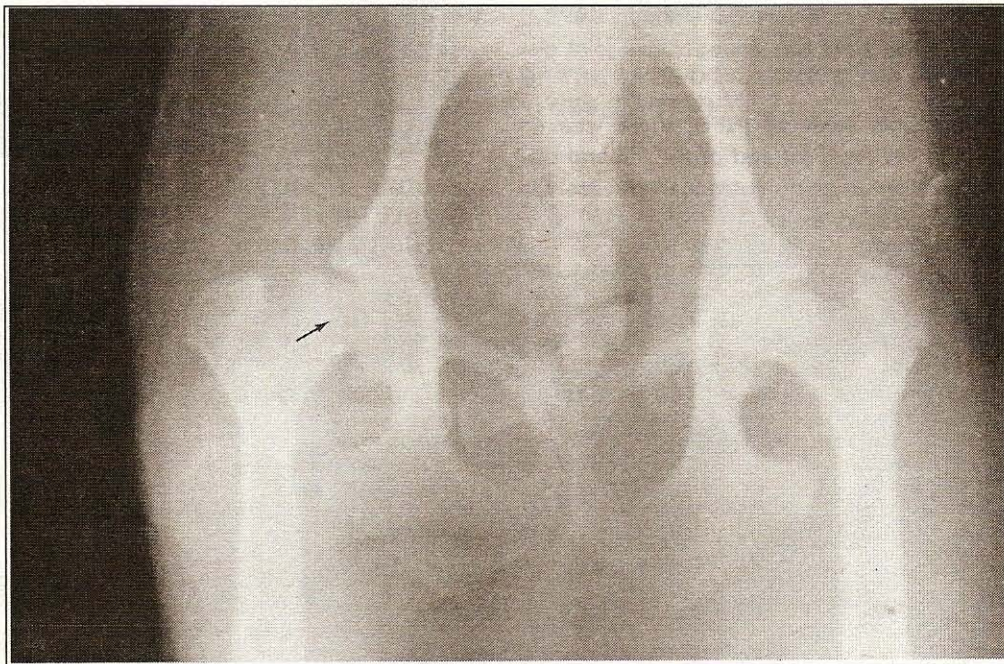


FIGURE 3. VENTRO DORSAL RADIOGRAPH OF THE PELVIS IN A TEN MONTHS OLD MALE DOG. SHOWING ISCHEMIC NECROSIS OF THE FEMORAL HEAD. NOTICE THE SIMILARITY WITH THE HUMAN.

coxofemoral joint. Crepitus in the joint may also occur. If the femoral head has collapsed, the affected limb may be noted to be somewhat shorter (about 1 cm) than the normal leg.

The muscles, especially the adductors, are in spasm. As the lameness becomes more evident, the size of the muscle mass on the affected side greatly decreases due to tissue atrophy. FIG. 1. Limited hip movements, especially internal and external rotation, are noted in the physical examination on af-

ected humans [6]. Abduction is also limited but to a lesser extent.

A tentative diagnosis can be made on the basis of the size, conformation, and age of the dog, and the presenting signs. A definitive diagnosis requires radiographic confirmation.

There are five major radiographic changes [7]. The articular surface becomes flattened and irregular, FIGS. 2 and 3. The degree of this change varies in intensity. The cause of this

flattening is the collapse of the dead bony trabeculae in the head of the femur under the stress of weight bearing. Irregular density of the epiphysis was noticed in all cases in this study, FIGS. 4,5.

Another prominent lesion was irregularity in the density of the metaphysis. These two changes are occasionally lumped together as lytic changes in the femoral head. These lytic changes are due to vascularization of the tissue. This results in resorption of the dead trabeculae and the laying down of thicker more irregularly arranged trabeculae [7, 12, 14, 15], FIG. 1.

The fourth feature of INFH is increased width of the femoral neck, FIG. 6. This change is due also to revascularization which results in deposition of new trabeculae. The change is accentuated by the collapse of the femoral head. The fifth major change is increased width of the joint space, FIG. 7. There are two theories for this radiographic finding. Kemp [6] believes it is due to a synovitis and Lee [7] to growth without calcification of the articular cartilage. The increased width is more evident as the disease progresses. Irregularities of the joint space also occur. These changes are, likewise, more evident and increased in both frequency and severity as the duration of the case increases. These acetabular changes are evidence of the progression of INFH into denegerative joint disease.

In a microangiographic study of INFH, there was an abundance of vessels in the proximal end of the femoral diaphysis and metaphysis. There was no evidence of vessels in the femoral epiphysis in diseased dogs.

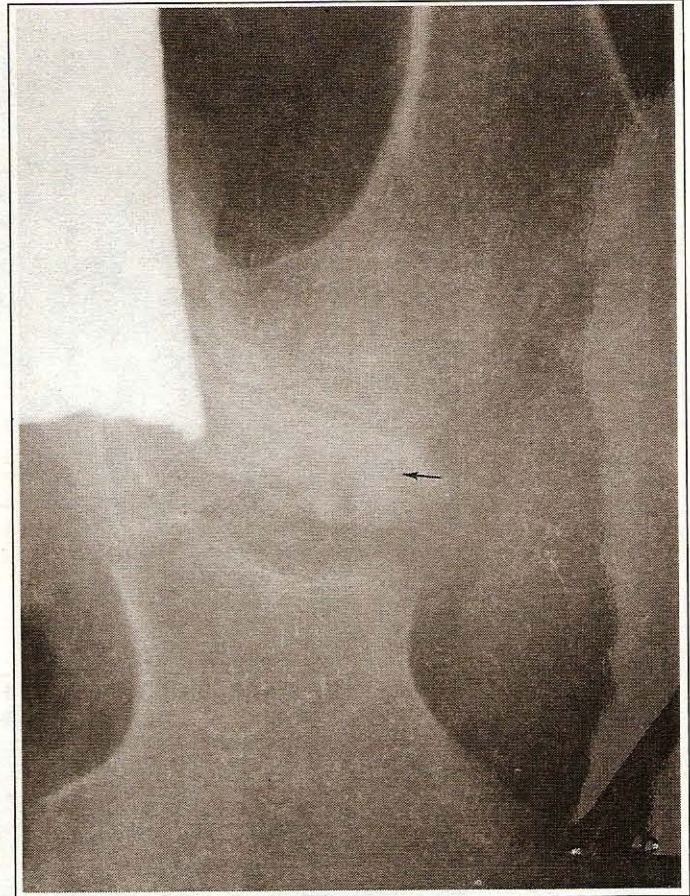


FIGURE 4. ANTERO-POSTERIOR RADIOGRAPH OF THE RIGHT HIP OF THE SAME BOY. NOTICE THE INCREASE DENSITY OF THE FEMORAL HEAD (ARROW).

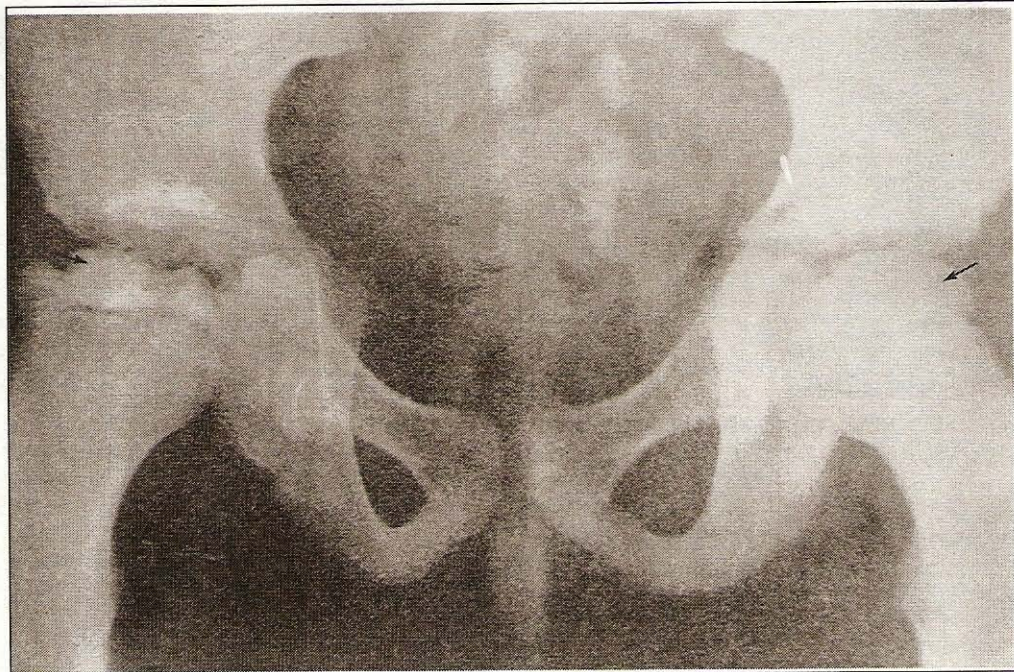


FIGURE 5. LEGG-PERTHES DISEASE OF THE RIGHT HIP. THE CAPITAL EPIPHYSIS IS FLATTENED AND IRREGULAR IN DENSITY, THE ARTICULAR SURFACE IS ROUGHENED AND THERE ARE AREAS OF ABSORPTION. COMPARE WITH THE OPPOSITE NORMAL HIP.

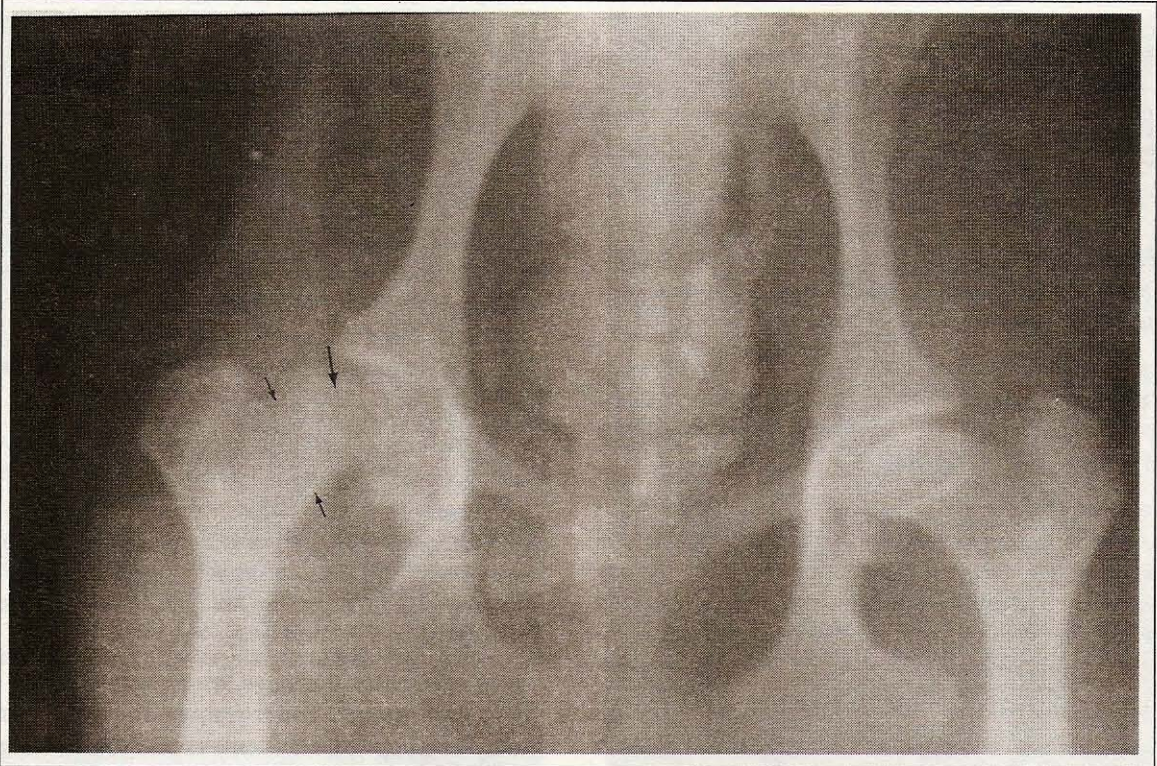


FIGURE 6. VENTRO DORSAL RADIOGRAPH OF THE PELVIS OF THE SAME DOG. NOTICE THE IRREGULAR SHAPE OF THE PROXIMAL EPIPHYSIS (ARROW).

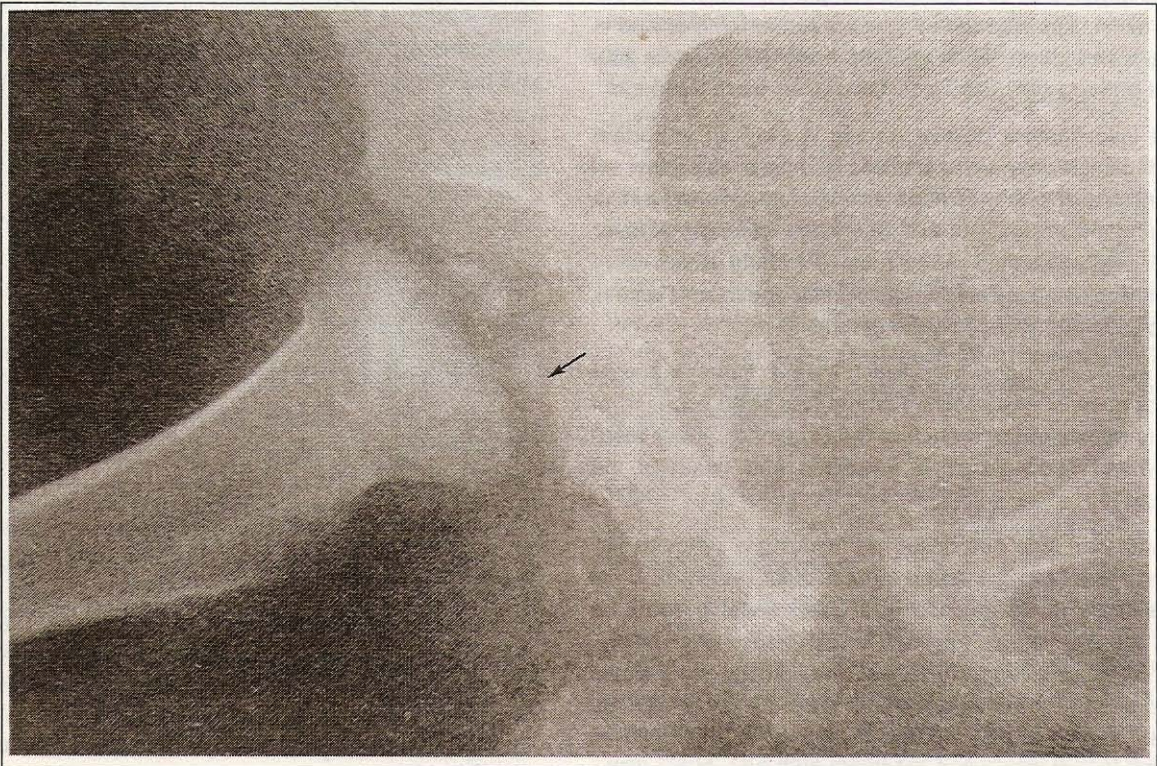


FIGURE 7. SAME HIP OF FIGURE 5 EIGHT MONTHS LATER. THERE HAS BEEN PROGRESSIVE ABSORPTION OF BONE, LEAVING ONLY SMALL FRAGMENTS OF OSSIFICATION.

The death of the femoral head tissue is followed by repair. The dead bone is characterized by empty lacunae [8]. Osteocytes lay down new bone around a central area of dead trabeculae [6]. This dead core is gradually hollowed out by the action of the osteoclasts.

Infraction, characterized by disruption of trabecular continuity, often occurs later. Isolated trabecular fragments show osteocyte degeneration and death. Recurrent infraction can be recognized by the intermingling of trabeculae fragments in different stages of osteocytic involvement [6].

The normal intratrabecular tissue is replaced in the area of infarction by fibroblastic proliferation and occasional fat cells. Hemopoietic tissue is relatively depleted. Blood vessels are present and appear normal.

The articular cartilage surface is irregular and roughened. It may occasionally be torn or detached. The femoral head may also show a depression on the anterolateral surface which corresponds to the acetabular lip. The normal rounded appearance is not present. The head is flattened to some degree depending on the severity of the case and the stage of repair [16, 17].

The femoral neck is composed mainly of compact bone [8]. The normal marrow elements are slightly replaced by fibrous tissue. Defects in the femoral neck are also filled with this highly vascularized fibrous tissue. This is especially prominent in the areas from which normal tissue has been resorbed.

There appears to be a genetic component involved in the etiology of INFH. The disease appears to run in families and is confined to small dogs [6]. A genetic basis for humans has been found [5].

Most investigators believe anoxia to play an important role also. Most likewise agree that the anoxia is somehow induced by trauma. (Hormones have also been mentioned as the factor causing the anoxia, but this theory has fallen out of favor because it was applied to many bone disorders which have been since explained by more appropriate theories. For this reason, the hormonal theory will be ignored here).

The actual mechanism by which trauma induces anoxia is the major point of contention. Trauma has been accused of causing thrombosis of the branch of the circumflex artery feeding the femoral head, resulting in anoxia and death of the femoral head [1].

Trauma has also been cited as inducing a synovitis [6]. The synovitis results in increased intracapsular pressure. If the blood vessel within the capsule had weak walls, it could be closed by this physiologic increase in intracapsular pressure. When the pressure closes the vessel, the femoral head would become anoxic. After the intracapsular pressure fell to normal, the vessels would reopen. By this theory INFH and synovitis are two extremes of the same scale.

Although the pathogenesis is still unsettled, INFH has been shown to have a genetic basis [5, 8]. On the basis of this alone, breeding of the affected animals should be avoided. If one accepts Kemp's theory, the control of the manifestation of the disease can be accomplished by preventing either the genetic or trauma factors from operating. Control of trauma may be accomplished by severely limiting exercise during the susceptible age. This method might result in increased numbers of susceptible dogs. Recurrence of the disease would result if trauma control was suddenly stopped [19]. On the other hand, control of the genetic factor by not breeding the affected animals would lower both the incidence of the disease and the number of susceptible animals.

To accomplish either of these programs would require the cooperation of the owners and breeders of these animals. Individuals who raise large numbers of puppies are the ones who need to be converted to this plan. If economics or winning in the ring must be compromised to accomplish elimination of the disease, the plan may not gain sufficient support for it to be carried out.

Many dogs that have this disease show no signs of spontaneously recover. The treatment of INFH depends of the severity of signs. If there is only slight dysfunction or a regression of signs, conservative methods are utilized. This includes a period of limited exercise and, analgesics to relieve pain and improve gait. The problem with this treatment procedure is that the epiphysis will always be irregular and will often continue to develop a more painful osteoarthritis. In humans, enforced long term bed rest (15 to 20 months) is a treatment of choice and results in a better prognosis than can be expected in dogs [9]. The head of the femur should move inside of acetaboli, without weight bearing and in abduction.

Another method of treatment used in humans when the diagnosis is made before the head collapses, is to hold the limb in abduction with a mechanical device [4]. Weight bearing is allowed during this time when the femoral head is reforming. Under this system, the head reforms nearly round. The results from this treatment are better than with the conventional bed rest. To apply this method of treatment to dogs, early diagnosis is required. This could most likely be accomplished by radiographically screening susceptible animals at an appropriate age for diagnosis, even though this method of treatment would be relative expensive.

At this time, surgical treatment is the treatment of choice in dogs. The head and neck of the femur are excised [20]. These light weight dogs improve dramatically post-operatively [18]. The limb shortens slightly. A pseudoarthrosis forms between the femoral greater trochanter and the gluteal muscle mass. There is a no gait abnormality noticeable and there is no pain on palpation of the coxofemoral joint. A slight drawback to this procedure is that the hip is not as stable as before. This, however, is of little concern because of the light weight of these animals.

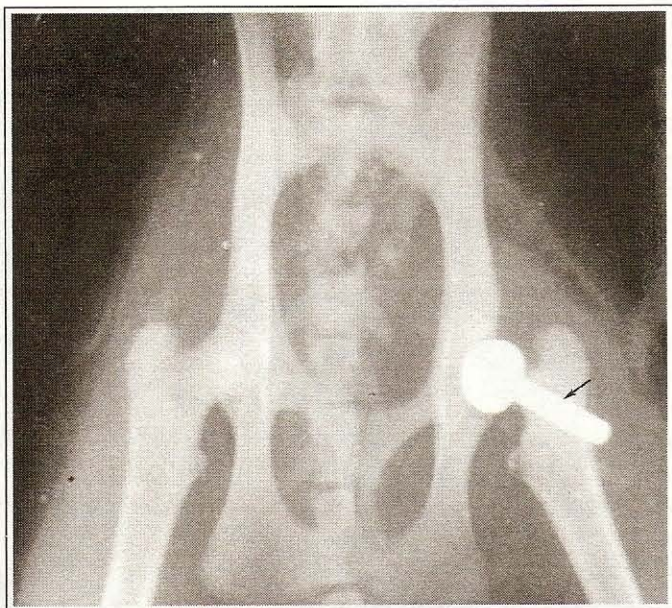


FIGURE 8. VENTRO DORSAL RADIOGRAPH AFTER THE IMPLANTATION OF A FEMORAL HEAD PROSTHETIC DEVICE.



FIGURE 9. VENTRO DORSAL RADIOGRAPH SHOWING UNILATERAL ENTIRE JOINT FEMORAL HEAD AND NECK PROSTHETIC DEVICE.

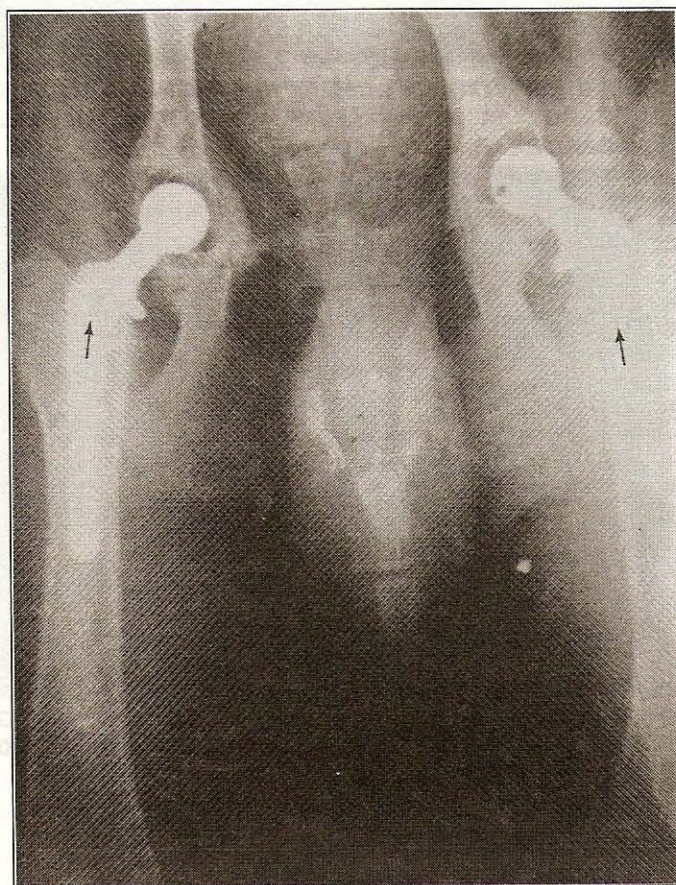


FIGURE 10. VENTRO DORSAL RADIOGRAPH OF A DOG SHOWING A TOTAL HIP PROSTHESES (BILATERAL).

For humans prosthetic devices for replacing either only the femoral head or the entire joint are being perfected and being used with relative success. In dogs, femoral head prostheses have been developed and used with good to excellent results [11, 13],

FIGS. 8 y 9. The use of these requires a normal acetabulum. Total hip prostheses have been almost perfected for use in the dog. The results of total hip prostheses in the dog are only fair to good initially, FIG. 10. However both types of prosthetic devices do not last longer than a year in most instances. For this reason, they are rarely used (especially since removal of the femoral head gives such good results), FIGS. 11 y 12. There is also another reason, these devices are too expensive to use in dogs.

RESULTS

Statistic of occurrence

From a of 28,000 canine cases seen between November 1968 and November 1976 at the University of Pennsylvania and 11,210 canine cases seen between January 1968 and November 1995 at the University of Zulia Veterinary Policlinic Department of Diagnostic Radiology, there are 64 cases reported with INFH. The data might not accurately reflect the prevalence of the disease due to mild or subclinical cases and to the referral nature of the clientele.

RADIOGRAPHIC RESULTS

Flattened head	64/64	100%
Lytic areas on head	60/64	93%
Widened neck	58/64	90%
Acetabular changes	50/64	79%
Others	2/64	3%

TABLE I

ISCHEMIC NECROSIS OF THE FEMORAL HEAD AND ITS RELATIONSHIP WITH DWARF BREEDS

Breeds	Number of cases	Percentage
Poodles	32	50%
Terriers		
Yorkshire	07	10.9
Welsh	04	6.2
Fox	04	6.2
Schnauzer	01	1.5
Others		
Chihuahua	03	4.6
Pekingese	08	12.5
Pomeranian	01	1.5
Dachshund	04	6.2

TABLE II

ISCHEMIC NECROSIS OF THE FEMORAL HEAD AND ITS RELATIONSHIP WITH AGE

Age at presentation	Number of cases	Percentage
3 months - 6 months	07	10.9
6 months - 1 year	35	54.6
1 year - 2 years	19	29.6
2 years - 3 years	03	4.6

TABLE III

ISCHEMIC NECROSIS OF THE FEMORAL HEAD AND ITS RELATIONSHIP WITH SEX

Sex distribution	Number of cases	Percentage
Males	32	50
Females	31	47.9
Unknown	01	2.1

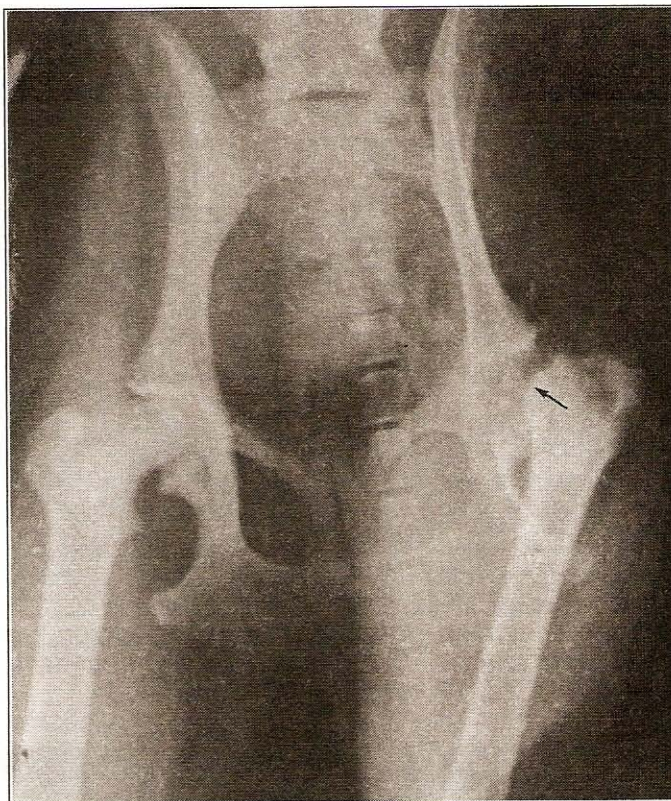


FIGURE 11. VENTRO DORSAL RADIOGRAPH OF THE SAME DOG IN FIG. 1 AFTER OSTEOTOMY OF THE FEMORAL HEAD.



FIGURE 12. VENTRO DORSAL RADIOGRAPH OF A DOG AFTER OSTEOTOMY OF THE FEMORAL HEAD AND NECK.

CLINICAL PRESENTATION. SELECTED CASE HISTORIES

Case 1

A male toy poodle of 9 months was presented with lameness in the left hip of four months duration. No weight was

borne on the left leg while running, but some was borne when walking. Pain and crepitus was noted on palpation of the coxofemoral joint.

Radiographic studies showed multiple lytic areas within a malformed femoral head. The head was flattened and the femoral neck was thickened. Remodeling changes were seen in the acetabulum.

Femoral head resection was done. Re-examination three days post operatively showed the animal to be using the leg slightly. No pain was evident on palpation.

Case 2

A 15 months male wire-haired fox terrier was presented with lameness of the right hindleg of about 1 year duration. The animal did not bear weight on the affected leg at any gait. The affected leg was shorter than the normal leg and the muscle mass was greatly decreased. Palpation of the coxofemoral joint caused pain.

Radiographs showed a mottled, collapsed femoral head. The neck was widened and periosteal proliferative changes were evident at the junction of the neck and diaphysis. Little change was noted in the acetabulum.

A femoral head osteotomy was performed. Re-examination showed the dog to be using the limb well 5 days later.

REFERENCES

- [1] Aegerter and Kirkpartirck. Orthopedic Diseases: Saunders Co. London. Pp. 175-179. 1958.
- [2] Campbell, C.S. Proceeding of the Conference on Aseptic Necrosis of the Femoral Head; held in St. Louis, Mo. United States Public Health Service. 37 p. 1964.
- [3] Edeiken, J. and Hodes, P.J. Roentgen Diagnosis of Diseases of Bone. Williams and Wilkings. 105 p. 1967.
- [4] Gibson, K.L.; Lewis, D.D. and Peckman, R.D. Use of external Coaptation for the treatment of avascular necrosis of the femoral head in the dog. Journal of the American Veterinary Medical Association. 197(7): 868-870. 1990.
- [5] Gray, I.M. Incidence and Genetics of Legg-Perthe Disease in British Columbia: Evidence of Polygenetic Determination. Journal of Medical Genetics. 9:197-202. 1972.
- [6] Kemp, H. Perthes Disease. (Thesis for Master of Surgery). University of London. 73 p. 1974.
- [7] Lee, R. Study of the Radiographic and Histological Changes Occurring in Legg-Calve-Perthe Disease in the Dog. Journal of Small Animal Practice 11:621-638. 1970.
- [8] Lee, R. and Fry, W. Some Observations on the Occurrence of Legg-Calve-Perthe Disease in the dog and an Evaluation of Excision Arthroplasty as a Method of Treatment. Journal of Small Animal Practice 10:309-317. 1969.
- [9] Lewis, D.D.; McCarthy, R.J. and Pechman, R.D. Diagnosis of common development orthopedic condition in canine pediatric patients. Compendium on Continuing Education for the Practicing Veterinarian. 14(3): 287-301. 1992.
- [10] Ljunggren, G. Legg-Perthe Disease in the Dog. Clinical Orthopedics and Related Research 62:31-36. 1969.
- [11] Mann, F.A.; Hathcock, J.T. and Wagner-Mann, C. Estimation of soft tissue interposition after femoral head and neck excision in dogs using neutrodorsal pelvic radiography. Veterinary Radiology and Ultrasound 34(4): 230-234. 1993.
- [12] Montgomery, R.D.; Finn, S. and Cooper, R. What is your diagnosis? Journal of the American Veterinary Medical Association. 203(11): 1535-1536. 1993.
- [13] Newton, C.D. Printed Lecture Notes for Surgery 8001, at the School of Veterinary Medicine. University of Pennsylvania. 1975.
- [14] Riser, W.H. Necrosis of the Femoral Head. Journal of the American Veterinary Medical Association 142:1024-103. 1963
- [15] Salter, R.B. and Ball, M. The Pathogenesis of Deformity in Legg-Perthe Disease: an Experimental Investigation. Journal of Bone and Joint Surgery 50B:767-773. 1971.
- [16] Schiller, M.G. and Aixer, A. Hypertrophy of the Femoral Head in Legg-Calve-Perthe4 Syndrome. Acta Orthopedic Scandinavia 43:45-55.1972.
- [17] Thibaut, J.; Deppe, R. and Munzenmayer, V. Surgical Treatment of bilateral femoral head osteonecrosis in a dog. Archivos de Medicina Veterinaria. Universidad Austral de Chile. 25:1, 95-99. 1993.