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Sajida Bano
(1) Faculty of Veterinary and Animal Sciences, RGSC, BHU, Barkachha, Mirzapur, Uttar Pradesh, India
(2) Department of Veterinary Medicine, Ethics and Jurisprudence West Bengal University of Animal and Fishery Science, Kolkata, West Bengal, India

Samar Sarkar
Department of Veterinary Medicine, Ethics and Jurisprudence West Bengal University of Animal and Fishery Science, Kolkata, West Bengal, India

Onkar Nath Bhaskar
Turning Veterinary Officer, Department of Animal Husbandry, Government of Bihar, Rajpur, Buxar, Bihar, India

Corresponding Author
Sajida Bano
(1) Faculty of Veterinary and Animal Sciences, RGSC, BHU, Barkachha, Mirzapur, Uttar Pradesh, India
(2) Department of Veterinary Medicine, Ethics and Jurisprudence West Bengal University of Animal and Fishery Science, Kolkata, West Bengal, India

Bone related disorders in dog special reference to osteoarthritis: A review

Sajida Bano, Samar Sarkar and Onkar Nath Bhaskar

Abstract
Bone related disorders constitute an important cause for lameness in dogs. Some bone disorders affect the joint membranes themselves. Bone disorders may be congenital or may be the result of injury to the joint, abnormal development, immune-related conditions or infections. Pain is the most common symptom of bone disorders. Bone related disorders like Degenerative joint disease (DJD) is a common disorder of humans and animals. It is generally regarded as a non -inflammatory condition of articular cartilage resulting from natural aging, trauma, or disease. Studies using clinical sign, blood parameters and radiographic techniques for evaluating various bone related disorders in dogs will be useful in diagnosis and a proper treatment regimen.

Keywords: Bone related disorders, degenerative joint disease, dog, lameness, pain

Introduction
Historically, dogs and humans lived in cooperative relationships, where dogs were used to hunt, guard or herd animals (Coppinger & Schneider, 1995) [16]. In many circumstances, humans are the most important factor influencing an animal's welfare. This is particularly true for companion animals, because pet and owner usually live in close association in the same environment.
Health issues for aging dogs and risk of osteoarthritis increases steadily from youth and older dogs are also at risk. 50% of dogs of 10 years age or older have osteoarthritis. A major concern for dogs as it increases stress on joints. Obesity can start as a problem in young dogs and continue into later years or it can develop in an arthritic dog that can no longer exercise normally. The cycle of inflammation, degradation, and chondrocyte damage in osteoarthritis can be promoted by joint stress because of excess body weight. Obesity also increases the likelihood of other diseases in addition to osteoarthritis. In this regard, literature pertinent to bone related disorders in dogs has been reviewed as follows.

Incidence
Incidence of Rickets in dogs
McMillan *et al*., (2006) [65] reported that Rickets is an important metabolic disease commonly seen in young growing dogs due to their rapid skeletal growth (Dittmer and Thompson, 2011) [20]. Aithal *et al*., (1999) [1] reported that the number of male dogs (75%) affected with rickets was significantly higher than females (25%), as the male dogs are metabolically more active than females and may require relatively more minerals especially Ca during their growth. Sharma (2002) [86] studied that a retrospective study in 100 clinical cases of rickets in canine and found that German Shepherd (42%) was most affected breed of dogs, followed by Boxer (16%), Non-descript (12%), Great Dane (8%), Doberman (8%), Spitz (7%), Dalmatian (3%), Apso (3%) and Pointer (1%). Joshi *et al*., (2007) [42] conducted a study to find out the existing feeding practices and common nutritional deficiency disorders in dogs. The prevalence of rickets was found to be 4% among the various nutritional deficiency disorders. Kushwaha *et al*., (2009) [50] studied the incidence, and the clinical, radiographic and haemato-biochemical changes in growing dogs affected with rickets and their response to Ca-vitamin D_3 therapy. The incidence of rickets was found to be 5.16%. Satisfactory to good response was observed to the combined therapy by 45th day. Ghoke *et al*., (2012) [27] reported that Labrador (40%) as the most vulnerable breed affected by rickets followed by German Shepherd (27%), Karwan (20%) and Pomeranian (13%). Pups in age group of 5-6 months (75%) were more affected followed by 3-4 months of age (27%). Male pups (80%) were found more susceptible as

compared with females (20%) due to higher requirement of nutrition for growth in male dogs. Fourteen cases (94%) were primarily presented for bow legged condition with a single (6%) case presented for hemorrhagic gastroenteritis. Hazewinkel (1989) [34] found that dogs which dependent upon dietary vitamin D sources, can develop rickets with skeletal growth deformities when a diet deficient in vitamin D was fed under controlled conditions. Growth plates and mineralisation grade normalized on supplementing commercial dog food. Kealy *et al*., (1991) [45] conducted a study to test whether growing dogs require supplementation with cholecalciferol (vitamin D_3) in a nonpurified extruded diet. Twenty eight weanling pups were divided equally in two groups and raised for 102 weeks. One group was fed with a diet that contained no added vitamin D, while the Kaneko *et al*., (1997) [44] reported a study in which young animals that were fed diets deficient in vitamin D and housed indoors without exposure to UV radiation, developed higher incidence of rickets. Malik *et al*., (1997) [60] concluded from their study that more common causes of rickets in pups are inadequate concentration and impaired absorption of Ca and P or hypovitaminosis D due to dietary deficiency. The pups were successfully treated by feeding a nutritionally complete, vitamin D-containing ration formulated for growing pups. Kushwaha (2003) [49] found that imbalance of vitamin D, Ca and/or P might cause rickets, the most common combination being a dietary deficiency of vitamin D and Ca and/or P. Inadequate sunlight was also reported as an another important factor that causes rickets. McMillan *et al*., (2006) [65] inferred that on decreasing dietary Ca in addition to decreased P play a role in decreasing bone mineral density in puppies. As a result, lethargy, decreased long bone growth, angular limb deformity, and osteopenia occurred which resolved within 3 months with nutritional management. Kushwaha *et al*., (2009) [50] reported that in rickets, poor growth and body conformation may be attributed to disproportionate growth of the axial and appendicular skeletal system along with poor development of skeletal muscles. The other clinical signs of rickets recorded were pain, lameness, broadening of distal metaphyses, bowing of fore limbs and hind quarter weakness. Campbell (1964) [12] described pathognomonic features of rickets, which included poorly mineralized and extremely thin cortices, enlarged, compressed and laterally displaced epiphyses and metaphyses, and widening of the epiphyseal growth plate. Johnson *et al*., (1988) [41] described classical radiographic signs of rickets in a Saint Bernard dog as thinning of cortices, widening of medullary cavity, increased physeal thickness, cupping of metaphyseal border, bowing/bending of long bones and broadening of metaphyses. Dittmer and Thompson (2011) [20] found that on radiographic examination that in case of rickets, widening of the physeal growth plate is the most archetypal change. Other abnormalities seen were metaphyseal flaring, thinning of the cortex, poor mineralization of the skeleton, and pathological fractures. Enlargement of costochondral junctions, the so-called rachitic rosary, is also a classic lesion of rickets that may be seen on radiographic examination.

Incidence of Hip Dysplasia in Dogs

Richardson (1992) [80] noted that in his study mentioned that Hip dysplasia is a common problem in veterinary practice, accounting for up to 30% of orthopedic cases and the frequency of the disease varies among breeds from as high as 70.5% in Bulldogs and 48.2% in St. Bernard to a low of 1.9% in Borzois. Ohlerth *et al*., (2001) [72] reported that overall occurrence of canine hip dysplasia (Federation Cynologique Internationale Grade c, d and e) was 29.6% and the heritability of total canine hip dysplasia grade was 0.44.

Smith *et al*., (2006) [90] reported that the occurrence of radiographic evidence of hip joint osteoarthritis in dogs increased linearly from an overall occurrence of 15% at 2 years to 67% at 14 years. Mele (2007) [67] reported that more than 50% of arthritic cases were observed in dogs aged between 8 to 13 years and 45% of dogs with hip arthritis were large breed dogs. Rocha and Torres (2007) [83] opined that Hip Dysplasia was one of the most common orthopedic disorders in dogs, representing approximately 30% of the total orthopedic cases. It occurred in all breeds; however the higher occurrences were seen in middle and large size breeds, and in breeds with rapid growth. In dogs, males and females were equally affected. Black *et al*., (2008) [7] and Ginja *et al*., (2009) [29] reported that in the United States, osteoarthritis was the most common cause of chronic pain of hips in dogs with more than 20% and 10 to 20 million dogs were afflicted. Fattahain *et al*., (2012) [24] stated that 105 dogs had been referred with lameness on hind limb. Dogs were younger than 1 year (50 dogs), 1 to 5 years (24) and older than 5 years. The rest of them were less than 10kg and the rest were heavier than 10kg. They also stated that degenerative joint disease (DJD) of hip joint had an effective role in dogs more than 10kg in comparison to less than 10kg. The association between DJD and the outcome wasn't statistically significant in dogs 1 to 5 years in both weights Priester and Mulvihill (1972) studied the relative risk of sex, size and breed for canine hip dysplasia and reported that males and females were equally affected. The risk in giant breeds was 50 times more than small or medium sized breeds. The authors also reported that even within a size certain breeds had excessive risk. Among large breeds, for example, Golden Retrievers had 50 times the risk of Collies. Lust *et al*., (1973) [57] reported that Labrador Retrievers had a high incidence of hip dysplasia, and the period between 3 and 8 months appeared to be important since during that time the initial diagnosis of the disease was made. The authors reported an incidence of 20 to 30% hip dysplasia in Labrador Retrievers. Riser (1973) [81] reported that severe canine hip dysplasia was evident on the standard hip extended view as early as 7 weeks of age. Lust *et al*., (1980) [56] reported that the radiographic signs of degenerative joint disease, a sequeal to hip dysplasia in dogs could be viewed from the hip extended view radiographs of the hip joints. The enumerated radiographic signs were synovial effusion, initial widening, then thinning of the radiolucent joint space, perichondrial osteophytes formation of non-weight bearing surfaces, enthesophyte formation, increased subchondral bone opacity, mineralization of intra articular and periarticular soft tissues, subchondral cyst formation and subluxation of the coxofemoral joints.

Incidence of Fracture in dogs

Harasen (2003) [32] reported that the Trauma is most common cause of fractures in small animals, and can occur due to bending, torsional, shearing and compression forces, eventually resulting in oblique, wedge fragment fracture, spiral or comminuted fractures. In dogs, fractures are commonly seen in femur, followed by tibia and radius-ulna. Robins *et al*., (1973) [82] opined that the incidence of pelvic fracture was about 25 percent of all long bone fractures in dogs. Kolata and Johnston (1975) [48] stated that most

common musculo-skeletal injury following motor vehicle trauma was pelvic fractures out of study conducted on 600 dogs met with motor accident. Phillips (1979) [76] reported that pelvic fractures accounted for about 15.8 percent of all fractures in dogs. Braden and Prieur (1986) [9] stated that the incidence of pelvic fracture in dog was about 20-31 percent of all fractures. Simon *et al*., (2010) [88] noted that the non-descript dogs were mostly affected by pelvic limb injuries (47.48%). Hoffberg *et al*., (2016) opined that most of the breeds affected with pelvic fracture were mixed breed dogs (21.68%). Denny (1978) [19] opined that the incidence of pelvic fracture was more in the age group of 2 to 3 years and most of the dogs were within 2 years of age. Denny (1978) [19] observed that the incidence of pelvic fractures in dogs was found to be more in female dogs (53%) when compared to male dogs (47%) out of 123 dogs with pelvic fracture. De Souza *et al*., (2011) [18] stated that male dogs (54.2%) were more affected than female dogs. Braden and Prieur (1986) [9] observed that acetabular fractures occurred about 29-43 percent of all the pelvic fractures in dogs.

Incidence of osteomyelitis in dogs

Piermattei *et al*., (2006) [77] found that In these cases, the bacterial contamination is the most prevalent cause of osteomyelitis and more commonly associated with Staphylococcus species, particularly *Staphylococcus intermedius* and *S. aureus*. Kwiatkowska (2011) noted that osteomyelitis is of bacterial, fungal, or possibly viral origin. Mycotic infections of bone are less common than those caused by bacteria but certain pathogenic fungi, in particular the yeasts *Cryptococcus*, *Coccidioides*, *Blastomyces*, *Histoplasma*, and *Aspergillus* may cause osteomyelitis following inhalation of spores, open fractures, surgery, bite wounds, foreign-body penetration, gunshot injury, extension from soft tissue, and hematogenous dissemination.

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Etiology of Joint disorders

Hedhammar *et al*., (1974) [35] stated that any sign of trauma involving a joint, such as luxation or trauma-induced laxity, is an indication for radiographic examination of a joint. As

trauma is a potential trigger for later changes, such as degenerative remodeling and osteoarthrosis in the affected joint, radiographs taken at the time of an injury provide a useful baseline against which to compare later changes. Mahoney and Lamb (1996) [58] reported that disorders of growing bones and trauma to long bones may have a secondary effect on the joints adjacent to the affected bone. Asynchronous growth of paired bones, such as the radius and ulna, can lead to incongruity in an adjacent joint. Mele (2007) [67] reported that osteoarthritis was most commonly caused due to various factors like age, genetic predisposition, obesity, strenuous physical activity, osteoarticular trauma and systemic diseases. Wahl *et al.*, (2010) [99] reported that certain breeds such as Labrador retriever and German shepherd dog were more predisposed to develop arthritis over and above the occurrence of underlying joint disease in those breeds. Nesic *et al.*, (2012) [70] opined that articular cartilage undergo age related changes that increased the risk of joint degeneration leading to the development of osteoarthritis and that the ability of chondrocytes to maintain metabolic homeostasis was shown to decline with age leading to alterations in the proteoglycans and collagen composition and organization.

Etiology of Hip joint disorders

Dobromylskyj *et al.*, (2000) [21] recognised vigorous exercise, particularly during growth as a predisposing factor for osteoarthritis and also joint surgery elevated the onset of hip arthritis. Fries and Remedios (1995) [25] reviewed that synovitis, articular cartilage degeneration and abrasions have been observed in the shoulder, stifle, elbow, vertebral, and mandibular joints of 30% of dogs genetically predisposed to hip dysplasia. Manley *et al*., (2007) [61] found that there were two ages at which dogs were presented with clinical signs of hip dysplasia; younger than one year of age with hip instability and overloading of some articular areas where in the pain was caused mainly by tearing or stretching of the round ligament, synovitis and acetabular microfractures and in adult dogs with chronic pain due to osteoarthritis. Ginja *et al*., (2009) [29] reported that gait abnormalities, such as stiffness, reduced height of step, shortened stride length, bunny hopping, and difficulty in rising and climbing stairs or in jumping over obstacles are the typical clinical signs. Impellizeri *et al.*, (2000) and Macphail (2000) considered obesity as an important risk factor for development of osteoarthritis of hip in dogs and occurrence of obesity and osteoarthritis was found in 25% of dog population. Kealy *et al.*, (2000) [46] described the benefits of restricted feeding on diseases of hip joint. The Radiographic evidence of osteoarthritis was lesser in restricted feeding when compared to the dogs fed with a normal diet. Smith *et al.*, (2001) [91] concluded that the excess weight overloaded the skeleton and contributed to the development of multi-factorial diseases, such as osteochondrosis, hip dysplasia. Altunatmaz *et al*., (2003) [3] opined that while genetic factors played the most significant role in the formation of hip dysplasia, other causes of hip dysplasia included; body type and development, feeding, uncoordinated development of muscle and bone, hormones and trauma exercise. Henrotin *et al.*, (2004) [36] stated that although concomitant factors including ageing and obesity were suspected to accelerate the progression of hip osteoarthritis, mechanical factors were thought to predominate in osteoarthritis. Etiopathogenesis of dogs and the abnormal mechanical strains induced osteochondral micro fractures, abnormal bone and cartilage bone remodelling and ultimately cartilage loss and bone sclerosis. Smith *et al*., (2006) [90] described that restricted feeding of dogs had lower occurrence and a later onset of hip joint osteoarthritis. Restricted feeding also delayed or prevented development of radiographic signs of hip joint osteoarthritis. Mele (2007) [67] reported that Hip osteoarthritis was most commonly caused due to various factors like age, genetic predisposition, obesity, strenuous physical activity, osteoarticular trauma and systemic diseases. He also concluded showed that weight gain was a significant predisposing cause for hip arthritis, as was increasing age. Mele (2007) [67] and Wahl *et al.*, (2010) [99] reported that certain breeds such as Labrador retriever and German shepherd dog were more predisposed to develop arthritis over and above the occurrence of underlying joint disease in those breeds. Rocha and Torres (2007) [83] mentioned that coxofemoral dysplasia was most frequently associated with the development of chronic degenerative joint disorders. He also stated that Diet had a profound influence on the onset of these pathologies, since overfeeding growing puppies causes a more rapid growth, which increases both bone length and body weight. Lascelles and Robertson (2010) [51] narrated that diets containing high levels of omega-3 fish oil especially docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) reduced damage to cartilage and slowed down progression of disease by reducing the production and activity of one of the main enzymes (aggrecanase) responsible for cartilage breakdown. Runge *et al*., (2010) [84] stated that age and weight of dogs were significant risk factors for development of hip osteoarthritis. Nesic *et al.*, (2012) [70] opined that articular cartilage underwent age related changes that increased the risk of hip joint degeneration leading to the development of osteoarthritis and that the ability of chondrocytes to maintain metabolic homeostasis was shown to decline with age leading to alterations in the proteoglycans and collagen composition and organization.

Etiology of Stifle joint disorders

Capaldo *et al.*, (2005) [14] reported that the causes of cranial cruciate ligament failure could be classified as traumatic or degenerative. Isolated caudal cruciate ligament tears are extremely rare and are caused by violent trauma to the cranial aspect of the tibia while the knee is in flexion. They are most common in conjunction with other, more serious ligamentous injuries, including instability of the collateral ligaments and meniscal injury. In most cases, osteoarthritis of the stifle joint is secondary to cranial cruciate ligament insufficiency but may occur in any dog as a result of traumatic, infectious, immune-mediated, or developmental causes. Tatarunas *et al*., (2006) [93] reported that the stifle was particularly vulnerable to Osteoarthritis secondary to cruciate ligament injuries, patellar luxations, and bone axis deformities. Rupture of the cranial cruciate ligament and the resulting meniscal injuries were the most frequent lesions in the canine knee. Lopez *et al*., (2013) [53] reported that cranial cruciate ligament rupture with concurrent development of osteoarthritis was one of the most common affections of the stifle joint in dogs. The age, body weight and breed were found to be predisposing factors with the large breeds of dogs being affected even at a younger age. Julia *et al.*, (2010) [43] stated that osteoarthritic change or meniscal injury showed signs of thickening of the tissues surrounding the joint, particularly on the medial aspect (“medial buttress”), decreased range of joint motion and crepitus or a 'click' on joint manipulation. An increase in

internal rotation of the tibia with respect to femur was seen in cases of collateral instability. Tibial thrust and cranial draw sign and abnormal patellar position was seen in cranial cruciate ligament ruptures.

Diagnosis of joint disorders

Physical examination

Pain assessment

Boothe *et al.*, (1996) [8]; Peach *et al.*, (2005) [75] and Corfield *et al.*, (2007) reported that the clinical diagnosis of osteoarthritis was predominantly on symptoms of pain, stiffness and associated functional impairment. Jaegger and Marcellin-Little (2002) [40] and Goldring and Goldring (2006) reported that joint pain was one of the hallmarks of osteoarthritis and the major cause of lameness associated with the disease and the symptoms of osteoarthritis were often associated with significant functional impairment as well as signs and symptoms of inflammation including pain, stiffness and loss of mobility. Manley *et al*., (2007) [61] found that there were two ages at which dogs were presented with clinical signs of hip dysplasia; younger than one year of age with hip instability and overloading of some articular areas where in the pain was caused mainly by tearing or stretching of the round ligament, synovitis and acetabular micro fractures and in adult dogs with chronic pain due to osteoarthritis.

Lameness and gait assessment

Macphail (2000) recorded that obesity was a risk factor for osteoarthritis and increased loads placed on an arthritic joint contributed to cartilage deterioration and that weight control might alleviate the severity of clinical signs of osteoarthritis by decreasing the amount of abnormal force put on the joint leading to significant lameness ranging from mild to severe. Nielson *et al.*, (2005) [71] noted that the orthopedic examination consists of three major components, including observing the animal at rest, observing in motion, and physical examination of the animal both standing and in lateral recumbences. He also stated that Visual observation of lameness will include change in stride length, change in joint angles both in stride and stance phases of gait, weight shifting, motion of the head and trunk, and changes in gait symmetry from normal. Kerwin (2010) [47] described that at the walk, one may observe a “head bob” with the head going up as the affected limb strikes the ground. Typically, the more proximal (andmore severe) the lesion, the more pronounced the head bob. He also described that some lameness may not show up at the walk but is more apparent at the trot, conversely, some lameness may most obvious at a slow walk. The animal should be observed from the rear, front and both sides. Farrell *et al.*, (2007) [23] reported that in case of Osteoarthritis crepitus might be detected during palpation of hip joint and the range of motion might be decreased due to the presence of osteophytes, capsular fibrosis, subluxation or fixed luxation. Julia *et al*., (2010) [43] stated that osteoarthritic change or meniscal injury showed signs of thickening of the tissues surrounding the joint, particularly on the medial aspect (“medial buttress”), decreased range of joint motion and crepitus or a 'click' on joint manipulation.

Hematological and biochemical parameters of bone disorders

Lipowitz and Newton (1985) [52] reported that there were no significant changes in haematological parameters in dogs with degenerative joint disease. However, in a few cases there was a non -significant increase in total white cell count which might indicate a low grade inflammatory process. Mala (2006) [59] and Vishal (2011) stated that there was no significant change in hemoglobin (Hb), total erythrocyte count (TEC), Total Leukocyte Count (TLC) values both between normal and joint affected dogs. Lust (1973) [57] observed no changes in hematological and biochemical parameters between diseases free and hip dysplastic dogs.

Radiography

Radiography of Elbow joint

Haudiquet *et al.*, (2002) [33] reported that the use of oblique radiographic projections has been recommended to increase the likelihood of identifying free fragments and radiographic diagnosis of Medial coronoid process of elbow joint is commonly based on recognition of nonspecific characteristics of degenerative joint disease like periarticular osteophytosis combined with exclusion of other primary disease processes like Osteochondrosis dessicans and ununited anconeal process. Trostel *et al.*, (2003) [96] concluded that some affected dogs may be clinically lame or have an abnormal gait. In dogs that were clinically lame, varying degrees of lameness maybe exhibited and exacerbation with activity is seen. Gait abnormality usually was present in dogs with bilateral disease, unless one elbow is worse than the other, making aunilateral lameness evident. Affected limbs were usually rotated inward with elbows rotated outward. Manipulation of the elbow will reveal a decrease in range of motion. Crepitation, joint effusion, joint capsule thickening and muscle atrophy are variable. Capaldo *et al*., (2005) [14] stated that the typical radiographic finding is a region of subchondral bone loss on the distal and medial portion of the humeral condyle that is best appreciated on a cranio-caudal radiograph. More advanced findings included sclerosis around the affected region and osteophyte production in the proximal aspect of the anconeal process, which is best visualized on a flexed lateral view. Vermote *et al.*, (2010) [98] described that radiographic suspicion of Fragmented coronoid process was often based on secondary changes: unclear delineation of the medial coronoid process, sclerosis of the ulnar notch, and secondary signs of osteoarthritis (OA) of the elbow.

Reuss-Lamky (2012) [79] stated that Clinical signs of elbow dysplasia were first noted at 4 to7 months of age although they may not appear until the dog is older and developed signs of osteoarthritis. An orthopaedic examination may reveal elbow joint swelling, crepitus and pain on elbow flexion and extension with reduced motion and muscle atrophy.

Radiography of Hip joint

Lust and Summers (1981) [55] noticed that radiographically there was incongruence between the articular surfaces with razing of acetabulum, flattening of the femoral head, coxofemoral sub laxity or laxity and secondary osteoarthritic alterations. Slocum and Slocum (1992) [89] reported that radiographic evaluation was used to confirm the diagnosis of hip dysplasia. Radiographic views included standard ventrodorsal and lateral projections of the pelvis. As the disease progressed, radiographic changes included coxofemoral subluxation and osteoarthritis. Briet *et al*., (2004) [10] reported that ventrodorsal radiographs of the pelvis were most commonly used for evaluation of canine hip joints. Peach *et al.*, (2005) [75] concluded that x-ray analysis of the joint was the gold standard measure of osteoarthritis. He also

stated that in osteoarthritic joint radiograph, the opposing surfaces of the two bones were closer together as the cartilage was lost. Rademacher *et al.*,(2005) [78] reported that hip extended radiographs were evaluated based on the contour of cranio-lateral acetabular rim, the thickness of the subchondral rim, the thickness of subchondral bone, changes of femoral head and neck and presence of a Morgan line to assess the extent of degenerative joint disease. Lorenz and Richter (2006) [54] stated that radiographically visible changes were narrowing of the joint space, forming of osteophytes and changes in the subchondral bone like thickening, fibrillation and cysts. Smith *et al.*, (2006) [90] noted that radiographic and clinical evidence of coxofemoral osteoarthritis was a common sequel of canine hip dysplasia especially in older dogs and that left and right hip joints of each dog were scored independently on the basis of sclerosis of the craniodorsal portion of the acetabular subchondral bone, osteophytes on the cranial aspect of the acetabular margin and femoral periarticular osteophytes. Rocha and Torres (2007) [83] opined that Canine hip dysplasia was an osteoarthritic disease that was most common in large-breed dogs. Dogs may have no clinical signs or lameness of variable degrees, depending on the severity of the disease. Affected dogs may also have difficulty rising and a bunny-hopping gait. In most clinical cases, there is hind-limb muscle atrophy and pain during manipulation of the hip. Lascelles and Robertson (2010) [51] observed significant new bone on the cranial effective acetabular rim in ventrodorsal view of pelvis in dogs with bilateral DJD. Shallow acetabulum bilaterally and a new bone formation at the attachment of the joint capsule on the neck of the femur was also noticed. Runge *et al.*, (2010) [84] reported that ventrodorsal hip extended radiographic projection of pelvis was utilized to determine the presence of hip osteoarthritis and the evaluation was based on the presence of periarticular osteophytes, subchondral bone sclerosis and joint remodeling

Radiography of Stifle joint

Burke and Ackermann (1986) [11] described that cranial cruciate ligament rupture with displacement of the proximal tibia cranially in relation to the femur and distal displacement of the popliteal sesamoid bone radiographically. The authors also recorded the radiographic features of degenerative joint disease to show periosteal proliferation at the site of the joint capsule and at the margins of the articular cartilage. This proliferation was usually smooth and uniformly mineralized with well-defined margins. The subchondral bone was thinned, thickened, dense or irregular. Intra-articular bone densities were also seen. mal-alignment of articular surfaces or joint subluxation was seen in specific conditions. De Rooster *et al.*, (1998) [17] reported that radiographic evaluation of the stifle joint can be an aid in confirming the diagnosis. Osteoarthritic changes are a nonspecific and inconsistent finding in the cranial cruciate-deficient patient, and they are inevitably more severe when evaluated on radiography. A triangle of increased radiographic lucency caudal to the patellar tendon on lateral projection of the stifle is associated with the infrapatellar fat pad, and it has been suggested as an indicator of cruciate disease. Capaldo (2005) [14] concluded that radiographic findings were rarely normal in dogs with partial tears, although abnormal findings may be subtle. In acute cases, lateral radiographs show soft tissue swelling in the caudal joint causing obliteration or caudal deviation of the fat line of the gastrocnemius muscle planes in the popliteal fossa. Similar soft tissue swelling causes loss of the triangular detail of the infra-patellar fat pad in the cranial joint. In sub-acute cases (i.e., 3 to 6 weeks), early osteophyte formation may be noted, especially on the proximal and distal poles of the patella and the medial and lateral trochlear ridges of the femur. In chronic cases (i.e., months), these osteophytes may be large and accompanied by similar changes along the medial, lateral, and caudal aspects of the tibia. Subchondral sclerosis may also be present. Abercromby *et al.*, (2007) found that typical radiological findings in Osteoarthritis of stifle were osteophytes, bone sclerosis, cysts and subluxations.

Osteoarthritis

Sokoloff L *et al*., (1969) reported that Degenerative joint disease (DJD) is a common disorder of humans and animals. It is generally regarded as a non- inflammatory condition of articular cartilage resulting from natural aging, trauma, or disease. Many names have been applied to the condition; however, osteoarthritis and degenerative joint disease are two of the most common and will used interchangeably. DJD has been recognized in animals for a long time. Paleopathologic examination of dry-bone preparations has shown lesions comparable to those of DJD in fossil reptiles from the Mesozoic. Pederson NC *et al*., (1978) [75] reported that there are two major categories of articular disease are described; non inflammatory and inflammatory. OA is placed in the non-inflammatory category although an intermittent inflammatory component is recognized.

Incidence of OA

Bennet (1984) [4] reported that in dog with arthritis, OA accounted for 78% of cases. As many cases 20% of dogs above 1 year of age are affected with OA (Mc Laughlin and Roush, 2002) [66]. Harari (1997) opined that neutered large breed dogs such as Rottweilers, New foundlands and Retrievers were more Susceptible to cranial cruciate ligament rupture and subsequent OA of stifle joint. McLaughlin and Roush (2002) [66] reported a patient's breed, age, Sex and body weight provide valuable when the patient's elat1ve risk for OA is assessed. McLaughlin and Roush (2002a) [66] observed that small breed dogs are more often affected with patellar luxation or aseptic necrosis of the femoral head, both of which may lead to OA

Anamnesis of OA

McLaughlin and Roush (2002a) [66] found that the medical histories obtained from the owners of osteoarthritic patients vary depending on the joint or joints involved, severity of the degenerative changes as well as the duration and cause of OA. In general, a patient will have a history of an intermittent, slowly progressive lameness or gait change and exercise intolerance is frequently reported. Lameness often goes unnoticed and owners often indicate that their pets are Unwilling to jumpy run or climb stairs and that they prefer to sit or lie down rather than stand. Difficulty in rising and stiffness after resting are common complaints. Many OA patients have a history of earlier acute painful, non-weight bearing lameness, which gradually improved and resolved, because OA mostly develop secondary to trauma or injury.

Etiology of OA

Bennet (1991) [5] and May (1994) reported that primary OA was said to occur in particular breeds such as the Chow,

Dalmatian, Samoye, Retriever and Spaniels and the exact cause was not known. It might be related to metabolic abnormality of cartilage or wear and tear with a hereditary or genetic basis. Clyne (1987); Bennet (1991) [5] and May (1994) observed that secondary degenerative joint disease was said to be usually associated with as unstable, deformed joint which was usceptible to minor, repeated, trauma leading to development of certain well defined pathological changes. Any abnormality affecting articular surface Congruity and joint stability as well as the even distribution of forces across the joint surfaces led to cartilage damage and affected, cartilage horneostasis. Examples of such conditions are hip dysplasia, rupture of the anterior cruciate ligament of the stifle joint

Clinical signs of OA

Bennet (1991) [5] and May (1994) reported that many joints affected with OA may not show clinical signs at least in the early stages. Lameness, is of a chronic nature, of insidious onset, and there is usually a progressive deterioration with time Lameness and stiffness often follows periods of exercise and will initially resolve with rest. As the disease progresses, stiffness becomes more pronounced following rest. Acute flare-ups occur by excessive exercise or trauma to the joint

Physical examination

McLaughlin and Roush (2002a) [66] recorded thata complete physical examination must be performed in a patient suspected to have OA to rule out neurologic, metabolic, and cardiopulmonary abnormalities as causes of lameness, pain, and exercise intolerance. Then a complete orthopaedic examination must be performed to identify the musculoskeletal abnormalities. Patients are to be observed for weight bearing stance, muscle mass, and symmetry and lameness when ambulating. The lameness should be evaluated when the patient is moving at a slow walk, a fast walk, and a trot. Having the patient move the duration of weight bearing on the affected limb should be looked for. The affected limb is to be carefully palpated beginning distally and working proximally. Evaluate each joint for evidence of pain, swelling, effusion, crepitating, fibrosis, laxity or reduced range of motion. Examine muscles for atrophy or pain. Additional tests are performed to confirm the presence of OA.

Pathology of OA

Gardner DL (1980) [26] reported that the characteristic structural changes that typify OA are disorganization and loss of articular surfaces and proliferation of tissues in and adjacent to these surfaces. The precise sequence of histologic change in affected articular tissues is not known with absolute certainty. Much of the sophisticated histologic and biochemical research into OA has concentrated on articular cartilage. Gardner DL (1980) [26] reported that advancing age is important in the pathogenesis of DJD but is probably not the primary cause of the disease. While cartilage breakdown is more frequent in older humans and animals, all joints are not affected concomitantly, and it is evident that local factors play a major pathogenic role. With advancing age a considerable number of chemical, biologic, and mechanical alterations occur in normal articular cartilage. It has been stated that if aging itself is considered normal, so then too should these changes. Palmoski MJ *et al.*, (1980) [73] observed that load bearing is important in the normal biology of joints. Langenskiold A *et al.*, (1979) observed that the nutrient transport to articular cartilage via the synovial fluid is dependent on joint motion and weight bearing. In fact, degenerative changes of articular cartilage have been demonstrated in studies in which limbs were immobilized for varying periods of time. McCarty DJ *et al*., (1979) [63] recorded that the affected joints were allowed no motion or weight bearing. At the end of 5 weeks, definite changes in articular cartilage, some indistinguishable from DJD, were noted. Conversely, heavy use of well aligned joints rarely contributes to cartilage breakdown. McCarty DJ *et al*., (1979) [63] observed that the placement on joints of abnormal physical stresses that accompanies such conditions as chronic subluxation, hip dysplasia, slipped epiphyses, and aseptic necrosis' plays a vital pathogenic role in the development of DJD. Studies have shown that joint remodeling normally occurs with advancing age, thus changing joint contour and articular surface congruity. Joints normally are not entirely congruous but rather have areas of "contact" and "noncontact." With increased loads on joints, the areas of contact increase in size, and greater joint surface congruity is achieved. With advancing age, minor alterations in cartilage allow greater congruity with smaller and smaller loads, thus bringing areas of normal noncontact in the intact joint into contact. Such alterations in congruity lead to erosive and osteophytic changes McCarty DJ *et al*., (1979) [63] stated that the classic morphologic changes of osteoarthritic articular cartilage begin with fibrillation. Weiss C (1979) [101] described that a local surface disorganization involving a splitting of the superficial layers of the cartilage. The early splitting is tangential with the cartilage surface, following the axes of' the predominant collagen bundles. Horizontal flaking of cartilage occurs along with the development of shallow pits or clefts perpendicular to the cartilage surface. Eventually, the splitting extends deeper, altering the normal arrangement of the collagen bundles. As the disease progresses, the clefts extend entirely through the cartilage to its junction with subchondral bone. Weiss C (1979) [101] recorded that proteoglycan of normal articular cartilage is constantly degraded and resynthesized; its half-life has been estimated at 150 days. Pederson NC (1978) [75] reported that DJD changes are first recognized as a focal area of dullness on the articular cartilage surface. This is accompanied by a colour change from the more normal glistening white to a mottled gray or yellow. Because of alteration in the matrix composition, these areas also become softer than normal. Chondrocytes adjacent and superficial to the softened areas become more numerous, while those within the Softened areas decrease in number. Increased numbers of cartilage cells at the initial site of injury has been viewed by some as an attempt at intrinsic cartilage repair Sokoloff L (1969) remarked that bony change that often accompanies DJD is that of exophytic growth at the margins of the articular surface. Marginal osteophytes occur at the junction or interface between the articular cartilage and synovial membrane. They may appear as protuberances into the joint space or develop within capsular or ligamentous attachments at the joint margins. Gilbertson EMM (1975) [28] stated that osteophyte formation begins as a deposition of mineral outside the existing bony cortex. Further deposition of new bone, resorption, and remodeling ultimately produce a mature osteophyte. Capped by a hyaline or fibrocartilage surface, mature osteophytes communicate freely with the marrow spaces of the bone from which they arose. Sokoloff L (1980) [92] noted that a third significant bony change of DJD is the development of sclerosis in the subchondral area. The

bone in this area becomes denser with increasing loss of the articular cartilage above. This sclerosis has been described as being the result of increased loads placed on the bone because of the articular cartilage loss.

Biochemical changes of OA

McCarty DJ *et al*., (1979) [63] reported that the water content of normal articular cartilage is between 72% and 78%.McCarty DJ: Differential diagnosis of arthritis: Analysis of signs and symptoms. In OA not only is the water content increased but the water is more tightly bound and therefore less freely exchangeable with the joint space._McCarty DJ (1979) [63] reported thatit is generally accepted that the total proteoglycan content of osteoarthritis articular cartilage is decreased and that the decrease is directly proportional to the disease severity. However, while total GAG content of affected cartilage is diminished, not all GAG components are affected equally. When compared with normal cartilage, there is a relative decrease in keratin sulfate and an increase in chondroitin 4-sulfate such as is found in normal young or immature cartilage. Thus, chondrocytic synthesizing activity continues in affected cartilage but the products produced are different. Thompson RC (1979) [94] described thatin later stages of OA, there is a continued decrease in GAG synthesis and a relative decrease in chondroitin sulfate compared with keratin sulfate.

Risk factors

Lust *et al*., (1973) [57] considered hip joint laxity as a major risk factor leading to abnormal weight bearing forces and subsequent development of OA. Smith *et al.*, (2001) [91] opined that sex was not a significant risk factor for OA. German shepherd dogs were found to have 4.95 times more risk than the risk of Golden Retriever and Rottweiler put together. Allan (2002) [2] reported that hip joint was the most frequent location of OA occurring secondary to hip dysplasia, followed by shoulder and stifle. Melaughlim and Roush, (2002) reported that obese animal with OA might be more likely to show clinical sings and may be more debilitated by the disease

Haemato-biochemical findings

Lipowitz and Newton (1985) [52] studied that results of haemogram, urinalysis and blood parameters determinations are usually within normal results in OA, unless other conditions exist.

Diagnosis

Radiography

Peach *et al.*, (2005) [75] concluded that x-ray analysis of the joint was the gold standard measure of osteoarthritis. He also stated that in osteoarthritic joint radiograph, the opposing surfaces of the two bones were closer together as the cartilage was lost. Lorenz and Richter (2006) [54] stated that radiographically visible changes were narrowing of the joint space, forming of osteophytes and changes in the subchondral bone like thickening, fibrillation and cysts. Lascelles and Robertson (2010) [51] observed significant new bone on the cranial effective acetabular rim in ventrodorsal view of pelvis in dogs with bilateral DJD. Shallow acetabulum bilaterally and a new bone formation at the attachment of the joint capsule on the neck of the femur was also noticed. Runge *et al.*, (2010) [84] reported that ventrodorsal hip extended radiographic projection of pelvis was utilized to determine the presence of hip osteoarthritis and the evaluation was based on the presence of periarticular osteophytes, subchondral bone sclerosis and joint remodeling Mclaughlin and Roush (2002a) [66] opined that a diagnosis of OA can be based on a careful evaluation of a patient's significant, medical history, clinical signs, physical examination findings, laboratory data, and radiographic findings. Allan G (2002) [2] reported that the gamut of radiographic changes seen in OA are synovial effusion, initial widening of joint space, perichondral osteophyte formation of non-weight bearing surface, enthesiophyte formation, increased subchondral bone opacity, remodeling of subchondral bone, mineralization of intra-articular and peri-articular soft tissues, subchondral cyst formation (rare) and subluxation for the coxofemoral joint) Mc Laughlin and Roush (2002a) [66] found that lateral and Craniocaudal views can be taken to diagnose OA. Heavily sedated patients allow proper radiographic positioning.

Treatment regimen of Osteoarthritis

Remedios and fires (1995) [25]; Hulse (1998) [38]; Doig *et al*., (2000) [22] and Anderson (2001) reported that it is currently not possible to cure OA, once it becomes clinically evident. Current treatment Strategies are directed towards slowing the progression of the disease and improving the quality of life and exercise tolerance by reducing joint pain, increasing joint mobility, and reducing cartilage destruction. The main goal of medical therapy in the treatment of OA is to restore and maintain normal joint function by alleviating joint pain and inflammation and protecting cartilage from further damage. This goal is generally achievable with a combination of weight control, activity moderation, anti-inflammatory medications and chondroprotective agents. Canapp *et al*., (1999) [13]. Found that treatment for animals with joint disease has met with limited success once bony changes associated with degeneration have developed. Preventing or minimizing the inflammatory process within the synovium may interrupt the cascade of events that can lead to cartilage degeneration. Controlling or preventing synovitis early in the disease process may slow the progression of OA.

Non-steroidal anti-inflammatory drugs (NSAIDs)

Meloxicam

Hulse D (1998) [38] repoted that Anti-inflammatory medications are the foundations for medical treatment of OA in dogs. These agents have an important role in palliation of pain associated with OA, decrese synovitis and improve lameness. Thus these agents improve mobility and quality of life in just a short amount of time. The most commonly used anti-inflammatory medications for the treatment of OA are non-steroidal anti-inflammatory drugs (NSAIDS). Mathew *et al.*, (2001) reported that Meloxicam is a non-steroidal anti-inflammatory drug of the oxicam group and it is derived from enolic group. Doig, *et al.*, (2000) [22] observed that meloxicam is a potent inhibitor of prostaglandin synthesis and has anti-inflammatory, analgesic and antipyretic properties.

Collagen peptide

McAlindon *et al*., (2011) [62] indicated forthe first time that orally administered collagen peptides have the potential to stimulate proteoglycan synthesis in the joints with mild forms of osteoarthritis. The Effectiveness of Specific Collagen Peptides on Osteoarthritis in Dogs-Impact on Metabolic Processes in Canine Chondrocytes clinical trials were made and the beneficial effect of orally administered collagen

peptides in osteoarthritic dogs has been clearly demonstrated Weide N (2004) [100] observed that the reduction in lameness and increased mobility in dogs after collagen peptide treatment were associated with a statistically significantly lowered plasma content of MMP-3, which is involved in collagen degradation. Beynen, A.C. *et al*., (2010) [6] reported that the treatment of osteoarthritic dogs of similar age and body weight with collagen peptides were required to achieve a positive impact in osteoarthritis treatment.

Vitamin D

Garfinkel, R. J *et al*., (2017) reviewed that the activated form of vitamin D (1a, 25 (OH) 2D3), plays a role in articular cartilage degeneration. Vitamin D binds to vitamin D receptors, triggering a signaling cascade that leads to chondrocyte hypertrophy. In clinical trials, vitamin D deficiency poses a risk factor for OA, and those with decreased cartilage thickness are more likely to be vitamin D–insufficient. Guillot X. *et al*., (2010) [30] observed that Vitamin D is a key immune regulator in the reduction of inflammation, and it has been shown to exert influence on T and B lymphocytes, macrophages, and dendritic cells.

Nagpal S *et al*., (2001) [68] recorded that because of anti-inflammatory nature of vitamin D, it is sometimes used as a medication for patients with chronic diseases such as rheumatoid arthritis, osteoarthritis, multiple sclerosis, and systemic lupus erythematous. Zhang *et al*., (2014) [102] have found that vitamin D deficiency increases the risk of patients developing OA. In a prospective study, it was found that individuals with similar characteristics who were deficient in vitamin D and who were assessed radiographically for OA had an increased risk for OA, as assessed by radiologists.

Calcium

Triveni A. Jambale *et al*., (2017) [95] concluded that patients with decreased blood serum levels of calcium due to prolonged inadequate calcium intake results in osteoarthritis.

Glucosamine and Chondroitin sulphate

Canapp *et al*.,(1999) [13] found that the oral disease modifying agents have glucosamine and chondroitin sulphate in various forms Glucosamine and chondroitin sulphate have different benefits due to different mechanisms of action. Hence, the combined use of these gents has advantage over their individual use and has been shown to have synergetic effect. Simoens. S and Laekeman. G (2010) [87] described that glucosamine is one of the most commonly used nutraceuticals, especially for arthritic patients, due to it being involved in the body's production of joint lubrication and shock absorption and maintaining healthy cartilage and joint function. Narcy SJ, Vangsness CT (2010) [69] reported that glucosamine supplements are often combined with chondroitin sulfate. Chondroitin sulfate, a type of gylcoaminogy can, addresses the disease process of arthritis by aiding in the repair of damaged connective tissue. Chondroitin sulfate is one of the most abundant glycosaminoglycan in joint cartilage, bones, tendons, cornea, and heart valves.[21]It is also beneficial to stress injuries, by keeping joints hydrating and protecting existing cartilage breakdown. Studies has theorized that supplementation of chondroitin sulfate will maximize blood circulation to subchondral bone and synovial joints. Chondroitin sulfate is vital for articular cartilage and joint structure because it can bind collagen fibrils and is used as a chondroprotective agent by inhibiting the degradations of cartilage matrix and synovial fluid. Supplementation of chondroitin sulfate is important because as the body ages, less chondroitin sulfate is produced and other glycosaminoglycan, such as keratin sulfate, are produced which predisposes the joint to osteoarthritis. In addition to the joint benefits, chondroitin sulfate supplements are noted to have up to 70% bioavailability when taken orally, this is significantly more than the bioavailability of other supplements and nutraceuticals. Overall, glucosamine chondroitin sulfate and other joint related glycosaminoglycans, seem to be relatively safe and do not display any long term side effects. Therefore, making glycosaminoglycan a popular alternative treatment for osteoarthritis in canines.

Sunthi *(Zingiber officinale)*

Pratishtan. B.T (1999) deliberated that Ginger is known as Sunthiin Ayurveda and description of the plant appears in the old text like Charaka, Sushruta, Vagbhatta and Chakra-dutta. Sharma K P V and Visvabharti C (1999) reported that in Ashtanga Hridaya, the plant has been used in Rasna Saptak Quath (a decoction based on seven medicinal herbs), and a traditional remedy for arthritis, Semwal, R.B et al., (2015) [85] reported that ginger is an anti-inflammatory and antirheumatic agent used in holistic medicine, and it contains bioactive molecules such as gingerols and shogaols. Frondoza, C.Get al., (2004) found that In vitro, ginger extract suppressed TNF-α and inhibited COX-2-mediated synthesis of proinflammatory cytocynes. Vd. Mukund Sabnis (2006) [97] reported that sunthi has anti-inflammatory activity as itblocks inflammatory prostaglandins and thromboxane. These anti-inflammatory actions are the result of inhibition of prostaglandin release and hence ginger may act in a similar manner as NSAID.

Conclusion

In the present investigation, incidence of different bone related disorders were studied. Further sex wise incidence, breed wise incidence, joint wise incidence and age wise incidence were also studied. It was found during the study that incidence of different bone related disorders were highest in fracture (38.00%), followed by hip dysplasia (35.33%) and OA was found to be 8.86%. In sex wise incidence of dogs male was found to be more prone to OA than female bitches (50.53%). In breed wise incidence of OA Labrador was found to be the highest affected breed which was 29.66%. As compared to the other breed of dogs. In joint wise incidence hip joint was found to be highest (70%) followed by stifle joint. In age wise incidence seven to nine years was the highest affected with OA (24.00%) followed by ten years (21.50%).

In the present investigation, based on results obtained or changes were observed during the above studies it can be concluded as follows. Osteoarthritis is considered to be a problem of geriatric dog population. Clinical signs such as pain, lameness, stiffness were reduced. In Radiograph osteophyte formation, increased bone opacity, development of sclerosis, loss of the articular cartilage and narrowing of joint spaces were reduced. Ginger seems to have potent ant inflammatory and analgesic property in conjunction with other conventional drugs that have evidence in the clinical, biochemical and radiological studies of osteoarthritic dogs. It was found that herbal drugs are economic, has low risk of side effects, can be used for a long duration and complementary

and alternative medicine. Treatment with collagen peptide along with combination of Sunthi, calcium and Vitamin D in oral form showed good therapeutic response and is better than conventional treatment alone.

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