CLINICO-DIAGNOSTIC AND THERAPEUTIC STUDIES ON OSTEOARTHRITIS IN GERIATRIC DOGS

By

DONKA BHAVITHA

ID No. RVM/2020-06

THESIS SUBMITTED TO

P. V. NARSIMHA RAO TELANGANA VETERINARY UNIVERSITY
IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE AWARD OF THE DEGREE OF
MASTER OF VETERINARY SCIENCE (VETERINARY MEDICINE)
IN THE FACULTY OF VETERINARY SCIENCE

DEPARTMENT OF VETERINARY MEDICINE
COLLEGE OF VETERINARY SCIENCE
P. V. NARSIMHA RAO TELANGANA VETERINARY UNIVERSITY
RAJENDRANAGAR, HYDERABAD - 500 030.
September, 2022

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September, 2022

CERTIFICATE

This is to certify that **Ms. DONKA BHAVITHA (I.D. No. RVM/2020-06)** has satisfactorily prosecuted the course of research and that the thesis entitled **"CLINICO-DIAGNOSTIC AND THERAPEUTIC STUDIES ON OSTEOARTHRITIS IN GERIATRIC DOGS"** submitted is the result of original research work done and is of sufficiently high standard to warrant its presentation to the examination. I also certify that the thesis or part thereof has not been previously submitted by her for a degree of any University.

Date: 29/12/2022

Place: Hyderabad.

(Dr. G. AMBICA)

Major Advisor

CERTIFICATE

This is to certify that the thesis entitled **"CLINICO-DIAGNOSTIC AND THERAPEUTIC STUDIES ON OSTEOARTHRITIS IN GERIATRIC DOGS"** submitted in partial fulfillment of the requirements for the degree of **Master of Veterinary Science** (Veterinary Medicine) of **P.V. Narsimha Rao Telangana Veterinary University** is a record of the *bona fide* research work carried out by **Ms. DONKA BHAVITHA (I.D. No. RVM/2020-06),** under our guidance and supervision. The subject of the thesis has been approved by the Student's Advisory Committee.

No part of the thesis has been submitted by the students for any other degree or diploma. The published part has been fully acknowledged. All assistance and help received during the course of the investigations have been duly acknowledged by the author.

The final Viva Voce examination was held on _____ and the Thesis is approved by the Student Advisory Committee.

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LIST OF ABBREVIATIONS

Abbreviation		Full form
&	:	And
%	:	Per cent
^{0}C	:	Degrees Centigrade
^{0}F	:	Degrees Fahrenheit
<	:	Less than
>	:	Greater than
@	:	At the rate of
AGE	:	Advanced glycation end products
ALT	:	Alanine Aminotransferase
AST	:	Aspartate aminotransferase
BCS	:	Body condition sore
BUN	:	Blood urea nitrogen
B. Wt.	:	Body weight
CBC	:	Complete Blood Count
CHD	:	Canine hip dysplasia
CRP	:	C- reactive protein
CT	:	Computerized Tomography
dL	:	Deci liter (s)
DJD	:	Degenerative Joint Disease
DLC	:	Differential leukocyte count
et al.	:	And associates/co-workers
ED	:	Elbow Dysplasia
EDTA	:	Ethylenediamine tetra acetic acid
Fig.	:	Figure (s)
g / gm	:	Gram (s)
GAG	:	Glycosaminoglycan
GHz	:	Giga Hertz
Hb	:	Hemoglobin
i/m	:	intra muscular
inj.	:	Injection
IU	:	International unit(s)

IL	:	Interleukin
Kg	:	Kilo gram(s)
L	:	Liter(s)
LWD	:	Long wave diathermy
MCH	:	Mean Corpuscular Hemoglobin
MCHC	:	Mean Corpuscular Hemoglobin concentration
MCV	:	Mean Corpuscular volume
MHz	:	Mega Hertz
MPV	:	Mean Platelet Volume
Mg	:	Milli gram(s)
Min	:	Minute(s)
ml	:	Milli liter(s)
Mm	:	milli mole(s)
MW	:	Micro waves
MWD	:	Micro wave diathermy
No.	:	Number
NSAIDs	:	Non-steroidal anti-inflammatory drugs
OA	:	Osteoarthritis
OCD	:	Osteochondrosis dissecans
OD	:	Once daily
PCV	:	Packed cell volume
P/O	:	per os (orally)
RBC	:	Red blood cell (corpuscle)
SE	:	Standard error
SW	:	Short wave
SWD	:	Short wave diathermy
Tab.	:	Tablet
TEC	:	Total erythrocyte count
TENS	:	Transcutaneous electric nerve stimulation
TLC	:	Total leucocyte count
TNF	:	Tumour necrosis factor
TP	:	Total protein
U	:	Units
USG	:	Ultrasonography

VCC	:	Veterinary Clinical Complex
Wt.	:	Weight

DECLARATION

I, **Ms. DONKA BHAVITHA (I.D. No. RVM/2020-06)** hereby declare that the thesis entitled **"CLINICO-DIAGNOSTIC AND THERAPEUTIC STUDIES ON OSEOARTHRITIS IN GERIATRIC DOGS"** submitted to P.V. Narsimha Rao Telangana Veterinary University for the degree of **MASTER OF VETERINARY SCIENCE** is a result of original research work done by me. It is further declared that the thesis or any part thereof has not been submitted for any other degree or diploma.

Date: 29/12/2022

Place: Hyderabad

D Bhavitha

(DONKA BHAVITHA)

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Major Advisor	:	**Dr. G. AMBICA**
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Year of submission	:	**2022**

ABSTRACT

The present study "Clinico - Diagnostic and Therapeutic studies on Osteoarthritis in Geriatric dogs" was under taken with an aim to study the incidence, clinical signs, risk factors, diagnosis and to assess the efficacy of herbal preparations and physical rehabilitation techniques to manage osteoarthritis in geriatric dogs. A total of 3040 geriatric dogs were presented to Veterinary Clinical Complex, Campus Hospital, College of Veterinary Science, Rajendranagar; Veterinary Hospital, Bhoiguda and from peripheral Hospitals during the period of January 2022 to June 2022.

Among 3040 geriatric dogs, 350 presented with the signs of lameness, inability to bear weight and exercise intolerance were diagnosed positive for OA after subjecting to various imaging techniques, accounting the overall incidence of 11.51%. In the present study, the incidence of osteoarthritis reported highest in 220 dogs (62.86%) aged between 10-15 years, followed by 105 (30%) in 6-10 years and least in 25 dogs (7.14%) having more than 15 years age. Breed wise incidence was found to be higher in Labrador Retriever 160 (45.71%) followed by German Shepherd 72 (20.57%), Rottweiler 38 (10.86%), non-descript 33 (9.42%), Pug 25 (7.14%), Spitz 13 (3.71%), Golden Retriever 8 (2.29%) and least was recorded in Dalmatian 1 (0.29%). Gender wise incidence was higher in males (60%) compared to females (40%). Joint wise incidence was found to be high in hip joint 75 (82.57%), followed by elbow joint 54(15.43%) and least in stifle 07 (2%). In the present investigation, among the risk factor involved in occurrence of OA in geriatric dogs, overweight (57.71%) was found to be major factor, followed by neutering (12.86%), slippery floor (9.43%), over use of calcium (8.57%), heavy exercise (5.71%), underlying joint diseases (4.29%) and Hypothyroidism (1.42%).

In the present study, the major clinical signs observed in osteoarthritic affected dogs were pain in 350 (100%), reluctant to move in 310 (88.57%), lameness in 250 (71.43%), sluggish rising in 180 (51.43%), difficulty in walking upstairs or jumping in 150 (42.86%), stiffness in 50 (14.29%) and reduced appetite in 20 (5.71%) dogs, indicating pain as a major clinical sign associated with osteoarthritis in geriatric dogs.

Dogs with osteoarthritis were divided randomly into two groups containing 10 in each and investigated further. However, 10 apparently healthy dogs were considered for comparison. Physiological parameters, haematological and serum biochemical parameters were estimated on day 0, 15 and 30 days and were within the normal range in both the groups before and after therapy and showed non significant difference ($P>0.05$) when compared to apparently healthy dogs. Further, clinical parameters like weight bearing, joint mobility, lameness scores and pain scores showed significant increase ($P<0.01$) when compared to apparently healthy dogs before the therapy in both the groups. After therapy, significant reduction ($P<0.05$) in weight bearing, joint mobility, lameness scores and pain scores were seen in both group I and group II dogs, however reduction is more significant in group II dogs compared to group I dogs. Further, synovial analysis revealed normal volume, viscosity ranging from normal to decreased in severely affected OA dogs, colour ranging from yellow to red tinged with a significant increase in total protein ($P<0.05$) when compared to apparently healthy dogs, while synovial TNCC (cells/ml) and DLCC (%) does not show any significant difference ($P>0.05$) when compared to apparently healthy dogs in both groups before and after therapy.

Radiography of OA dogs in the present study revealed the presence of advanced osteoarthritic signs like formation of osteophytes, joint space narrowing, loss of femoral head contour and certain early osteoarthritic signs were not visible under radiography, so further ultrasonography was done in these dogs to know the early osteoarthritic signs and USG of osteoarthritis dog revealed the presence of joint effusions, myositis and tendinitis. Further, CT was done in the severe OA dogs to know the progression of disease and the CT revealed the presence of hypertrophied femoral head with osteophytes at the neck of femur, which are not visible under plain radiography and ultrasonography.

Two groups OA dogs managed clinically with two treatment regimens. Group I treated with Inj. Prednisolone @ 0.5mg/kg i.m for 5 days, followed by Lubrihans @ 1 tab/10 kg B. wt. oral for 30 days and Diathermy applied twice a day in more severe cases and twice a week in less severe cases for 4 weeks and Group II received Tab. Carprofen @ 4.4 mg/kg for 5 days, followed by Ashwagandha powder @ 500-1000 mg/kg oral for 30 days and TENS applied twice a day in more severe cases and twice a week in less severe cases for 4 weeks. Clinical signs started disappearing early in group II by day 15 as compared to group I and therapeutic response was significantly high in Group II (80%) compared to group I (50%).

Therefore, in the present study, it can be concluded that the diagnostic efficacy of radiography alone may not be sufficient and should be combined ultrasonography and CT to know the early osteoarthritic signs and the progression of disease. Regimen comprising Carprofen, Ashwagandha and TENS could be recommended safely in geriatric dogs with no adverse effects.

CHAPTER I

INTRODUCTION

Dog domestication began with the dawn of humanity and they are today an essential component of many families throughout the world. Dogs are one of the most loyal creatures on the planet because they bring companionship, unconditional affection and utility. However, dogs, like all other living creatures become old following some of the geriatric changes. Geriatric age is one period in which they require extra attention. A deviation from normal bodily physiology is an indicator of an illness or disease during geriatric change. A profound relationship forms between pets and their owners during the geriatric age. Because of this strong link, a comprehensive diagnosis and management of geriatric disorders is critical for the well-being of aged dogs as well as pet owners.

Aging process is influenced by various factors like genetics, nutrition, exercise and breed size, which makes the chronological age different from physiological age and in dogs, the senior age starts at 7 years, while geriatric age at 10 years and also breed size was one of the most notable factors for various diseases in geriatric dogs (Goldston and Hoskins, 1995). Small-breed dogs reach geriatric status at around 11 years, medium-breed at 10 years, large-breed at 8 years and giant-breed dogs at 6 years (Siegal Mordecai, 1995).

Among all the different geriatric ailments, osteoarthritis (OA) is a notable condition. It is the most frequent kind of arthritis in dogs and is the leading cause of persistent discomfort in older canines. The prevalence of osteoarthritis in dogs over a year old can be as high as 20%, with middle-aged and older canines being more vulnerable (Bland, 2015).

The term arthritis is derived from two Greek words*, 'arthon*' means joint and '*itis',* means inflammation, which is a severe problem in middle-aged and elderly dogs however can occur in dogs of any age. Arthritis can be classified as either degenerative (or) inflammatory (May, 1995). In dogs, inflammatory arthropathies are less prevalent than 0steoarthritis (Bui and Bierer, 2003). The most frequent kind of joint illness in dogs is degenerative type i.e., osteoarthritis (OA), which is complicated, progressive synovial joint disease characterised by articular cartilage deterioration and the production of new bone at the joint borders.

Osteoarthritis in dogs is a slowly progressing, degenerative and dynamic condition that can result in significant discomfort, lameness and incapacity. Arthritis can damage one or more joints, causing pain, stiffness, swelling, lameness and decreased movement (Bennett and May, 1995).

OA is a chronic degenerative condition of the joints that causes pain, stiffness, edoema and lameness (McLaughlin, 2000) and affects dogs more frequently than rheumatoid arthritis (Hielm-Bjorkman *et al*., 2003). Clinically OA is characterised by lameness and joint pain (Mortellaro, 2003) and is frequently encountered in canine practise, associated with canine hip dysplasia (CHD) and elbow dysplasia (ED) (Innes. 2006). OA has been divided into two forms: primary and secondary. Primary is the result of defective articular cartilage structure and biosynthesis and is uncommon in dogs (Bennett and May, 1995), whereas secondary results from abnormal forces acting on a normal joint like overweight, fracture, luxation, infection, crystal arthropathy or immune mediated inflammation or normal forces acting on an abnormal joint like abnormal joint conformation, osteochondrosis, hip and elbow dysplasia (Bennett and May, 1995; Innes, 2006).

Arthritis, if left undetected and untreated, causes lasting damage and can hinder pets from completely engaging in daily activities such as walking, jogging and swimming (Ranganath, 2012).

Osteoarthritis is often managed with a multimodal strategy that may include activity control, weight management, nutritional assistance, physical therapy and the use of nonsteroidal anti-inflammatory medicines, analgesic pharmaceuticals and nutraceuticals (Aragon *et al.*, 2007). Ayurveda is an ancient Indian medicinal science that has been practised for over 5000 years and is Indian traditional medical system. Because of the toxicity and side effects of allopathic drugs, the usage of herbal medicine has resulted in a surge. According to current estimates, around 80% of people in underdeveloped nations rely on conventional medications for basic health care (Devkar, 2015).

The diagnosis and management of OA in geriatric dogs is very important as it is one of most common clinical condition encountered and the reports on the clinical management of osteoarthritis in geriatric dogs are partly. Hence, the present study has been conducted with the following objectives:

1. To evaluate various factors involved in occurrence of osteoarthritis in geriatric dogs

2. To evaluate the various diagnostic techniques for osteoarthritis in geriatric dogs

3.To manage the osteoarthritic geriatric dogs with appropriate therapy

4.To assess the incidence of osteoarthritis in geriatric dogs

CHAPTER II

REVIEW OF LITERATURE

Osteoarthritis is one of the common conditions encountered in geriatric dogs. Unfortunately, the disease goes undiagnosed by most of the owners as well as clinicians because of the thought that it is common due to old age. However, arthritis is not just a geriatric dog problem, it can also be seen in young dogs due to underlying arthropathic disorders (Ranganath, 2012). Osteoarthritis can be well managed in young as well as in geriatric dogs with the help of conventional and non-conventional therapy. Herbal preparations and physiotherapy are a new concept of managing osteoarthritis in geriatric dogs in terms of relieving pain and stress and it has given a new hope in geriatric dogs suffering from arthritis.

2.1 ANATOMY OF THE NORMAL JOINT

2.1.1 Forelimb Anatomy

Scott and Witte (2011) in their work reported that around 25 percent of lameness caused due to musculoskeletal problems of the forelimb.

Riegger-Krugh *et al.* (2014) stated that the forelimbs support 60 percent of the dog's weight and forelimb skeleton is made up of the thoracic or pectoral girdle and forelimb bones and because of the higher range in size among dog breeds, the size of forelimb bones varies greatly.

2.1.2 Hindlimb Anatomy

Riegger-Krugh *et al.* (2014) in their writing informed that the hindlimb skeleton consists of the pelvic girdle, which was made up of fused ilium, ischium and pubis bones, as well as the bones of the hindlimb and the size of the hindlimb bones vary greatly due to the wide range of dog breed sizes and the back legs support 40 percent of the dog's body weight.

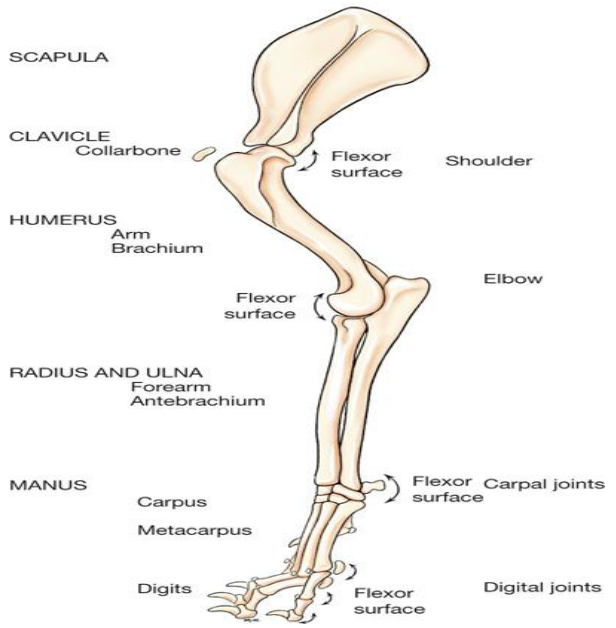

Fig. 2.1: Anatomy of the Forelimb Skeleton (Evans *et al.*, 2013).

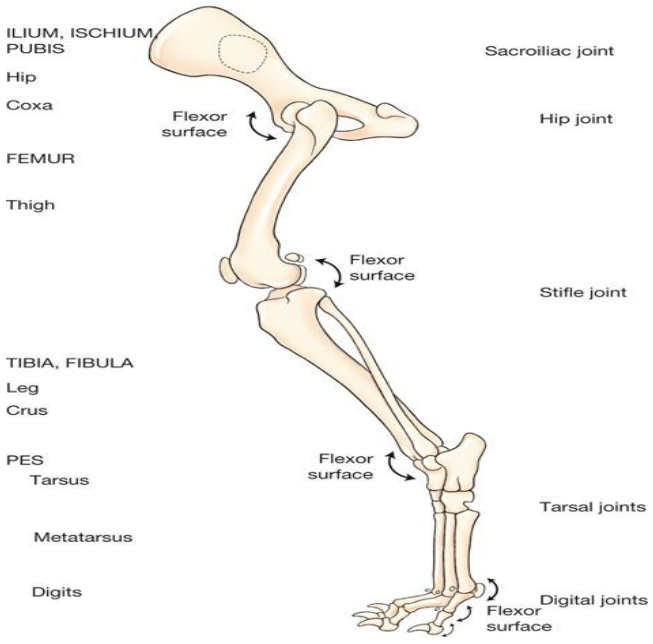

Fig. 2.2: Anatomy of the Hindlimb Skeleton (Evans *et al.*, 2013).

2.1.3 Normal structure of synovial joints

Dyce *et al.* (1996) in their writing reported that in synovial joints, the articulating bones will be separated by a fluid filled space known as joint cavity and is surrounded by a sleeve of delicate tissue known as the synovial membrane.

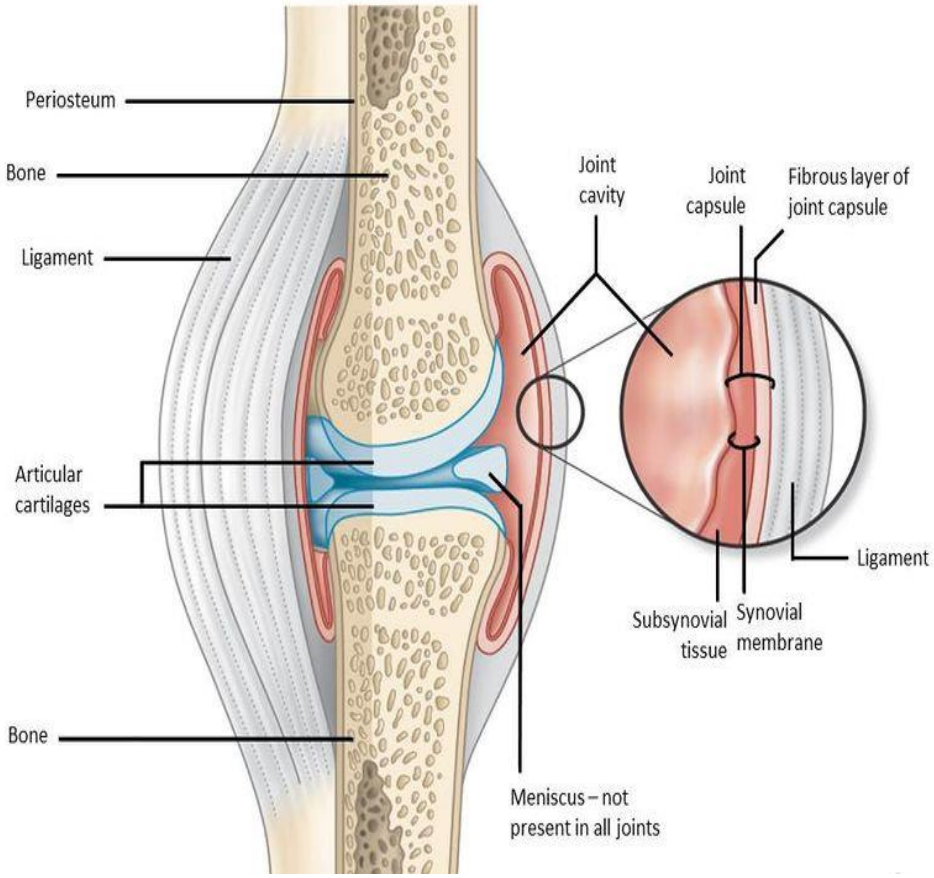

Fig 2.3: Anatomy of the synovial joints (Aspinall, 2011).

2.1.4 Composition of articular cartilage

Roush *et al.* (2002) studied the composition of articular cartilage and reported that articular cartilage is an avascular, aneural and alymphatic tissue found at the end of long bones and is composed of chondrocytes and extracellular matrix. Chondrocytes occupy less than 2 percent of articular cartilage by volume and maintain the extracellular matrix and by weight,

the matrix is composed predominantly of water (65-80%) in the form of filtered synovial fluid, collagen (20- 30%) and proteoglycans (5-10%).

MacWilliams and Friedrichs (2003), stated the articular cartilage is a hyaline cartilage that covers the bones and the thickness varies from joint to joint and even within the same joint and normal cartilage is translucent with a bluish gleam. Further, they stated that articular cartilage has a high elasticity and compressibility, which helps the bones to resist external stresses and is made up of proteoglycans, which are polypeptide cores with one or more negatively charged glycosaminoglycan (GAG) side chains.

2.2 Osteoarthritis

Anderson *et al.* (2018) defined osteoarthritis as one of the most frequent joint diseases in both humans and veterinary medicine, characterised by gradual deterioration and remodelling of synovial joints, resulting in reduced mechanical function and discomfort.

Kim *et al.* (2019) reported that dogs most frequently experience chronic discomfort from osteoarthritis, also known as degenerative joint disease, a degenerative illness that is difficult to treat since adult articular cartilage has a limited capacity for regeneration.

Monteiro *et al*. (2019) defined osteoarthritis as a disease that affects the synovial joints and progresses over time and causes structural and functional changes as a result of inflammatory, biomechanical and metabolic factors.

Johnson *et al*. (2020) in their work informed that osteoarthritis as a progressive painful condition of synovial joints, characterised by articular cartilage degradation with proteoglycan and collagen loss, subchondral bone sclerosis, periarticular proliferation of new bone and persistent inflammation of synovial membranes and expected to affect 20 percent of dogs before the age of one year and 90% of dogs over the age of five years.

Aulakh *et al.* (2021) defined osteoarthritis as a degenerative joint condition marked by decreased joint range of motion, discomfort, cartilage breakdown and the production of osteophytes.

2.3 Incidence

Prasad *et al.* (2012) conducted study on 159 dogs and reported the incidence rate of osteoarthritis as percent.

Triakoso (2013) conducted study on the incidence of geriatric dog diseases and reported 7.2 percent prevalence of musculoskeletal diseases.

Millis *et al.* (2014) in their study concluded that, out of 28 dogs tested, 19 (68%) had at least one joint with radiographic osteoarthritis.

Jain *et al.* (2015) conducted study on diseases in geriatric dogs and revealed that the prevalence of arthritis accounts for 2.47 percent.

Lamani *et al.* (2019) conducted study on 200 dogs suffering from coxofemoral joint affections and found 64 dogs with osteoarthritis with an incidence rate of 32 percent.

Anderson *et al.* (2020) collected data from 200 veterinarians and reported that the prevalence of osteoarthritis in North America as 20 percent and in UK from 2.5 to 6.6 percent.

2.3.1 Age wise incidence

DeGroot *et al.* (2004) reported that as the age advances, there will be accumulation of AGEs, which shown to increase the tissue stiffness, decrease synthesis and increase the degradation of the extracellular matrix and also affects the cellular process within the joint and may predispose for the development of osteoarthritis in aged dogs.

Smith *et al*. (2006) reported that the prevalence of osteoarthritis will be more in aged dogs and the incidence increases as the age advances and the large active muscle mass may inhibit the transformation of passive laxity into the functional laxity as the dog make attempts to move and which decreases the stresses on the articular cartilage that leads to degenerative changes in the joints.

Elliott *et al.* (2007) reported osteoarthritis as a common condition in middle aged to geriatric dogs affecting 1 in every 5 dogs and giant to large sized breeds appear to have an increased prevalence of orthopaedic diseases which further progress to osteoarthritis in the later stages of life.

Mele (2007) reported that osteoarthritis is a common condition seen in dogs aged > 6 years.

Prasad *et al*. (2012) conducted study on 159 osteoarthritis dogs and reported that the incidence of osteoarthritis is high in dogs aged > 4 years (82 percent).

Bland (2015) in his study revealed that the prevalence of osteoarthritis can reach 20 percent, with middle-aged and older canines being at increased risk.

Lamani *et al.* (2019) in their study on osteoarthritis reported the highest incidence of in dogs aged between 5-10 years (62.5 percent).

Wright *et al. (*2019) reported that the mean age recorded in the osteoarthritis cases is 7.61 years and also reported that 37 percent of the osteoarthritic cases diagnosed were of > 1year aged dogs.

Anderson *et al*. (2020) reported the prevalence of osteoarthritis in North America as 20 percent in the dogs aged > 1 year and in UK 2.5 to 6.6 percent irrespective of age and 20 percent in dogs > 1 year age.

2.3.2 Breed wise incidence

Elliott *et al.* (2007) reported that giant to large sized breeds appear to have an increased prevalence of osteoarthritis in the later stages of their life.

Mele (2007) stated that the musculoskeletal diseases are common in geriatric patients and reported that >50 percent of the arthritic cases are seen in giant dog breeds, 28% in medium sized dogs and 27% in small sized dogs.

Prasad *et al.* (2012) in their study revealed that among 159 cases of osteoarthritis, German shepherd has high incidence of osteoarthritis about 30 percent (47) followed by Labrador retriever 25 percent (39), Great Dane percent (4) and Spitz 19 percent (30), while the Rottweiler, Doberman, Boxer, Pug, Dalmatian, Bull mastiff, Saint Bernard, Bull dog, Dachshund, Cocker Spaniel, Lhasa apso and Iris setter breeds contributed less than 2 percent.

Bland (2015) in his study revealed that large breed dogs such as German Shepherds, Labrador Retrievers, Siberian Huskies and Rottweilers are more likely to develop osteoarthritis than small breed dogs and in dogs older than a year.

Anderson *et al.* (2018) reported higher incidence of osteoarthritis in Labrador Retriever due to their higher body weights.

Lamani *et al.* (2019) reported that Labrador Retrievers has the highest prevalence of osteoarthritis among all the dog breeds.

Meeson *et al.* (2019) reported that the incidence of osteoarthritis is high in Labrador Retrievers among different dog breeds.

Anderson *et al.* (2020) reported that breed is a consistent finding as a common risk factor for development of joint diseases and certain breeds have predisposition and risk of joint diseases due to the body conformation and genetic factors and breeds like Mastiffs, Boxers, German Shepherd, Labrador Retriever, Golden Retriever, Italian Corso dog and Bernese Mountain dogs are shown to have higher risk for hip and elbow dysplasia, which further leads to osteoarthritis.

Johnson *et al.* (2020) reported that larger dog breeds like Labrador Retrievers has higher incidence of osteoarthritis because of their high body condition score.

O'Neill *et al.* (2020) in their study identified that the Labrador Retriever (30.68%), German Shepherd dog (6.98 percent), Staffordshire Bull Terrier (5.19 percent) and Rottweiler (3.73 percent) are the most often affected breeds with osteoarthritis, followed by crossbred dogs (16.23 percent).

2.3.3 Gender wise incidence

Fernandes *et al.* (2002) reported higher incidence of osteoarthritis in male dogs than female dogs.

Smith *et al.* (2006) in their work concluded that males are more prone to osteoarthritis, which might be due to sex hormone influence than females.

Mele (2007) reported that irrespective of breeds, among males and females, males are more frequently affected with osteoarthritis than females.

Prasad *et al.* (2013) reported that among osteoarthritis in dogs, the incidence for development of osteoarthritis is more in males (57%) than females (43%).

Jain *et al.* (2015) conducted study on diseases in geriatric dogs and revealed that the prevalence of arthritis accounts for 2.47 percent in male dogs and also reported that the musculoskeletal abnormalities like paraplegia, lameness and limping of limbs are some of the senile changes seen in geriatric dogs.

Lamani *et al.* (2019) reported that the incidence of osteoarthritis is high in males (60%) than in females (40 percent).

Anderson *et al.* (2020) reported that the sex is not a significant factor for the development of osteoarthritis.

2.3.4 Joint wise incidence

Paradis *et al.* (2003) reported that the hip, elbow and the stifle joints are most commonly affected joints in osteoarthritis and concluded that the degenerative joint disease develops due to developmental abnormalities like hip dysplasia, elbow dysplasia and acquired arthropathies like cranial cruciate ligament rupture.

Peach *et al.* (2005) in their study reported that hip joint is most commonly affected joint in osteoarthritis than other joints.

Ginja (2009) reported that among all the joints, hip joint is most commonly involved in osteoarthritis in dogs.

Sanghi *et al.* (2009) informed that that hip joint is most commonly affected in osteoarthritis in dogs than elbow and stifle joints.

Lamani *et al.* (2019) reported that hip joint osteoarthritis is higher in dogs followed by elbow joint and stifle arthritis.

Stabile *et al.* (2019) reported that the incidence of hip joint osteoarthritis is very high in dogs, which accounts for 82.57 percent.

2.4 RISK FACTORS

2.4.1 Obesity

McLaughlin and Roush (2002) reported that the obese animals that developed osteoarthritis are most likely to exhibit the clinical signs with more severity than non-obese OA dogs.

Lund *et al.* (2006) reported obesity as an important risk factor for development of osteoarthritis in dogs.

Marshall *et al.* (2009) reported that obesity is a major and consistent risk factor for the development of osteoarthritis and other joint related problems in dogs.

Runge *et al.* (2010) reported that the overweight and age are the common risk factors for the development of osteoarthritis in dogs.

Sanderson (2012) in their study concluded that obesity is connected with chronic inflammation, which plays a significant role in the development of some related illnesses such as osteoarthritis and controlling obesity plays an important element in reducing the prevalence of obesity-related disorders like osteoarthritis as well as the accompanying treatment costs.

Anderson *et al.* (2020) reported that overweight is an important risk factor for the development of joint related issues especially osteoarthritis.

2.4.2 Neutering

German (2006) reported that neutering is an important factor for the development of obesity in dogs, which in turn predisposes for development of osteoarthritis in dogs.

Meeson *et al.* (2019) conducted studies on effect of neutering in dogs and reported that neutering is an important factor for the development of obesity in dogs, which inturn predisposes for development of osteoarthritis in dogs.

Anderson *et al.* (2020) reported that neutering predisposes for the development of osteoarthritis in dogs which might be due to the effect of the gonadal hormones on the growth rate and development of the bones. Further, they also reported that neutered old aged dogs are more predisposed to osteoarthritis than intact dogs.

O'Neill *et al.* (2020) in their study concluded that neutering was a major factor for musculoskeletal disorders like osteoarthritis in dogs.

Preet *et al.* (2021) reported that neutering predisposes for the development of osteoarthritis in dogs, as the age advances.

2.4.3 Vigorous exercise

Mele (2007) reported that vigorous exercise, especially during the growth stages may predispose for the development of osteoarthritis and also opined that the joint surgeries have elevated the occurrence of arthritis in dogs.

Bland *et al.* (2015) reported that osteoarthritis is commonly encountered in dogs because of excessive running or heavy exercise.

Anderson *et al.* (2020) reported that because of the overuse and damage to growing joints caused by various types and intensities of exercise such as chasing balls, toys and playing frequently with other dogs during growing age may predispose to joint abnormalities like osteoarthritis.

2.4.4 Over use of calcium

Richardson and Toll (1997) on their study on relationship of nutrition to development of skeletal diseases in young dogs, reported that nutritional excess like calcium as one of the risk factors for the development of osteoarthritis in dogs.

Lauten (2006) reported that excess calcium at the young age may have high risk for the development of orthopaedic diseases in dogs.

Raditic and Athens (2019) reported that excess feeding of calcium during young age in dogs may leads to developmental joint disorders like osteoarthritis.

2.4.5 Managemental factors

Alsaleem *et al.* (2013) reported that constant usage of high stairs by the dog is an important risk factor for development of knee osteoarthritis

Witte (2019) reported that dogs kept on slippery flooring like tarpaulin and newspapers are more likely to develop hip dysplasia, which may lead to osteoarthritis in later stages of life.

Capon (2021) reported that providing non-slippery surfaces to dogs suffering from osteoarthritis will help to slow down the advancement of the disease.

Goldberg (2022) reported that making dog walk on yoga mats can assist a dog with osteoarthritis of the joints by preventing slippage on slippery surfaces, which is a potent risk factor for the development of osteoarthritis.

2.4.6 Underlying joint diseases

Sandell (2012) reported hip dysplasia as one of the most common orthopaedic issues in dogs, which leads to osteoarthritis as the age advances.

Alsaleem *et al.* (2013) reported that trauma to the joint in young stages of dog is a risk factor for the development of osteoarthritis in the later stages of life.

Ramirez-Flores *et al.* (2017) revealed that underlying joint problems such instability, joint incongruency and inappropriate loading through the joint are the secondary causes of osteoarthritis.

Meeson *et al.* (2019) reported hip dysplasia as the most common risk factor for osteoarthritis in both humans and dogs.

2.4.7 Hypothyroidism

German (2006) reported hypothyroidism as one of the several factors which cause obesity in dogs, which in turn leads to osteoarthritis.

Juge *et al.* (2017) in their study identified that hypothyroidism accounts for 3.4% of the osteoarthritic cases.

Kutzler (2020) reported that gonadectomy is an important factor for the development of hypothyroidism in dogs, which leads to osteoarthritis as the age advances.

2.5 Clinical signs

Henrotin *et al.* (2005) reported that osteoarthritis is associated with chronic pain, lameness, functional disability, which leads to loss of joint function and thereby decrease the quality of life.

Beale (2004) noted that the symptoms of osteoarthritis include soreness, reluctance to get into the car or go upstairs, lagging behind in walks and sluggish rising and in addition, there are other symptoms like stiff gait, lameness, joint thickness, joint edoema and crepitus and further opined that all osteoarthritis dogs show pronounced pain.

Goldring and Goldring (2006) reported that joint pain, which is a hallmark of osteoarthritis and the main factor contributing to the condition's lameness and as the illness worsens, further symptoms such as stiffness and loss of motion become apparent.

Clouet *et al.* (2009) reported that osteoarthritis is characterised by decrease in articular cartilage thickness, subchondral bone sclerosis, formation of osteophyte at the joint margins and alterations can be seen in the synovial fluid composition.

Lindey and Taylor (2010) reported that pain is consistent finding seen in osteoarthritis dogs and if left untreated may leads to severe debilitation and lameness.

Ranganath (2012) reported that reduced activity, stiffness, limping, reluctance to move, walk, run, climb upstairs, find difficulty in rising from rest position, soreness of muscles when touched, pain on palpation of the joint, chewing and licks of affected joint and reduced appetite are some of the signs exhibited by osteoarthritis affected dog.

Prasad *et al.* (2012) reported that reluctant to move, walk or climb upstairs, gait and posture abnormalities and decrease in thigh muscle mass are some of the signs shown by osteoarthritis dog.

Clark (2015) informed that osteoarthritis, commonly known as degenerative joint disease, a slowly progressing inflammatory disease characterised by cartilage deterioration, hypertrophy of bone at the borders and changes in the synovial membrane, which finally results in joint pain and stiffness and due to dehydration and inflammation, joint structures may change and results in diminished flexibility and significant discomfort.

Pettit and German (2015) reported that inactivity, stiffness of the joint, reluctance to exercise or move, difficulty in jumping and exercise intolerance are some of the signs noticed in osteoarthritis dog.

Meeson *et al.* (2019) reported pain as the cardinal sign of osteoarthritis.

Alves *et al.* (2020) reported that animals with osteoarthritis may not display overt lameness during a walk or trot, but instead display subtle changes in body weight distribution at a stance because of discomfort or instability and also informed that animals with osteoarthritis display a variety of clinical symptoms that can differ greatly within a few weeks of the start of osteoarthritis and opined that muscular atrophy was a consistent finding.

Anderson *et al.* (2020) reported that osteoarthritis is a common clinical and pathological outcome from a variety of joint problems that eventually result in the structural and functional deterioration of the joint with accompanying lameness and discomfort.

Barker *et al.* (2021) reported that osteoarthritis is characterised by the deterioration of articular cartilage, remodelling of the underlying bone, the production of osteophytes and varying degrees of discomfort, pain and lameness brought by synovitis.

Lawrence *et al.* (2022) reiterated that avoidance of touch, decreased mobility, disturbed sleep patterns and pain on probing and extension of the afflicted joint, additionally, they noted that inactivity of the affected joint may result in muscle atrophy and excruciating discomfort.

2.6 Diagnosis

2.6.1 Physical examination

McLaughlin and Roush (2002) reported that a complete physical examination should be performed on osteoarthritis dog to rule out neurological, metabolic and other abnormalities and then complete orthopaedic examination should be done to identify the musculoskeletal abnormalities.

Rychel (2010) reported that proper and early diagnosis is very important in arthritic cases to assess the pain, which can be done by thorough physical examination like muscle palpation involving cervical muscles, paraspinal muscles and major muscles associated with each limb.

Pers and Jorgensen (2013) reported that complete history of the osteoarthritis patient and thorough clinical examination is very essential for the diagnosis of osteoarthritis.

Pettitt and German (2015) reported that osteoarthritis usually occurs secondary to primary cause and complete physical examination should be carried out to know the exact

cause. Physical examination will be helpful to know the normal limb motion, soft tissue damage and muscle atrophy and can be compared with the normal joint.

Syrcle (2017) reported that complete physical examination should be done to know the gait abnormalities in osteoarthritis dogs; on physical examination crepitus is evident.

Kogan *et al.* (2020) reported that an initial physical examination of osteoarthritis dogs included systematic pain palpation, mapping of pain patterns, a casual gait study, a metabolic profile and a conversation with the owner should be done at the time of visit and on weekly intervals to know the improvement and drug dose adjustments.

2.6.2 Clinical parameters

2.6.2.1 Weight bearing

Hyytiainen *et al.* (2012) reported that dynamic weight bearing is tested when walking or running, whereas static weight bearing measures the weight bearing of limbs while standing and both are critical for assessing canine patients receiving physiotherapy and orthopaedic care.

Piel *et al*. (2014) reported that weight bearing assessment should be done to know the intensity of pain associated with osteoarthritis and can be done by direct and indirect methods. Indirect measures include static or dynamic weight-bearing, foot posture, gait analysis, spontaneous movement and mechanical or thermal or cold sensitivity whereas direct assessments include hind limb withdrawal test, knee compression force, effort threshold angle of knee extension, knee tissue edoema and vocalisations after stimulation of the affected knee.

Alves *et al.* (2022) reported that weight bearing assessment should be done in dogs on the initial day of visit and during the course of treatment to know the efficacy of the treatment and also opined that dogs with osteoarthritis show minor changes in weight bearing, which might be due to the pain associated with the disease.

2.6.2.2 Joint mobility

McCarthy *et al.* (2007) reported that veterinarians should examine joint mobility scores in osteoarthritis dogs, which range from 1 to 5.

Bland (2015) reported that measuring the joint mobility can help to identify the conditions of articular surfaces, joint capsule, ligaments and muscles.

Johnson *et al.* (2020) reported that joint mobility should be checked in osteoarthritis dog to know the progression of disease.

2.6.2.3 Pain scoring

Muller *et al.* (2016) in their work revealed that pain can be quantified using a pain scoring system, with higher scores indicating more pain that interferes with daily activities

Johnson *et al.* (2020) reported that pain is an important sign in osteoarthritis and can be assessed by using pain scoring system.

Corral *et al.* (2021) in their study revealed that the pain shown by the osteoarthritis dog can be quantified using a pain scoring method with a scale of 0-10.

2.6.2.4 Lameness grading

Catarino *et al.* (2020) reported that lameness grading, which varied from grade 0 to 5, should be evaluated in dogs to determine the degree of lameness, as part of the orthopaedic evaluation with osteoarthritis.

Aulakh *et al.* (2021) revealed that gait evaluation is most important part in any orthopaedic examination in dog and can be done by using lameness scoring, which also helps as consistent and unambiguous communication between veterinary practioners. Further opined that lameness scores can be used in conjugation with other measures for effective evaluation of lameness.

2.6.3 Laboratory Diagnosis

2.6.3.1 Haematology

Hurter *et al.* (2005) reported C-reactive Protein (CRP) as a marker in human clinical practise to identify and assess the efficacy of disease-modifying antirheumatic medication therapy in rheumatoid arthritis, whereas in veterinary medicine, CRP has been shown to reliably increase after induced inflammation and reliably decrease after the situation heals and

the normal CRP levels may range from 0.8–30 μg/mL. The CRP levels remain normal in the dogs affected with OA and cannot be used as a marker for diagnosis of OA.

Alam *et al.* (2006) reported that there are no significant changes in TLC, MCV, MCH, MCHC, MPV but slight decrease in TEC and Hb in dogs suffering with osteoarthritis.

Fujiki *et al.* (2007) reported that serum CRP values in osteoarthritis dogs are not substantially different from those in control dogs and there is no statistically significant difference when compared to healthy dogs.

McCarthy *et al.* (2007) reported that the dogs involved in their study showed positive radiological evidence of OA but no alterations in the hemogram.

Edamura *et al.* (2012) reported that there are no significant changes in haematological parameters in dogs affected with osteoarthritis.

Molina *et al.* (2014) in their study reported that between protocols and treatment days, the hemogram did not reveal any differences. Mean cell haemoglobin (MCH) varied according to protocols and treatment days, but the results are still within the normal range. The leukogram did not reveal any variations between celecoxib and meloxicam or between different treatment days.

Pettitt and German (2015) reported that haematological tests are not necessary for OA dogs but can be done for geriatric dogs in whom long term therapy would be recommended.

Hillstrom *et al.* (2016) reported that C-reactive protein (CRP) is an acute phase protein generated in the liver in response to increasing levels of interleukin (IL) 6 during inflammation and the concentrations of serum CRP is high in dogs with suppurative arthritis compared to dogs with OA.

Musco *et al.* (2019) reported that in OA affected dogs, haematology and serum biochemistry are within the normal range.

Daems *et al.* (2019) reported that through clinical examination and blood analysis should be evaluated before and after treatment to know the efficacy of treatment and in his

findings, he reported that there is no apparent increase in the number of white blood cells or globulins in OA dogs.

2.6.3.2 Serum biochemistry

Alam *et al.* (2006) reported that there are no significant changes in serum biochemistry parameters like ALT, ALP, BUN and creatinine in OA affected dogs.

McCarthy *et al.* (2007) reported that the dogs which had radiological evidence of OA had no alterations in biochemical profile.

Edamura *et al.* (2012) reported that there are no significant changes in serum biochemistry parameters in OA affected dogs except for a slight increase in ALP and creatinine levels.

Molina *et al.* (2014) in their study reported that renal and hepatic assessment revealed no differences between parameters except the levels of ALT and ALP differed across treatment days, but these measurements are also within the normal ranges.

Musco *et al.* (2019) reported that in OA affected dogs, serum biochemistry is within the normal range.

2.6.4 Synovial Analysis

2.6.4.1 Physical parameters

Fernandes *et al.* (2002) reported that the synovial fluid volume may remain within the normal range or may increase in dogs affected with osteoarthritis and the appearance of normal synovial fluid as clear, colourless to straw coloured fluid. In case of any inflammatory conditions, the synovial fluid appears as cloudy or turbid.

MacWilliams and Friedrichs (2003) reported that in osteoarthritis, the amount of synovial fluid collected may be within the normal range or slightly elevated than normal volume.

Gossec and Dougados (2004) reported that in osteoarthritis, synovial fluid had a low viscosity, which might be due to decreased concentration of hyaluronic acid.

May (2005) reported that gross changes in osteoarthritis synovial fluid include poor viscosity, increased volume with blood tinged in some cases and high levels of fibrinogen.

Herrero- Beaumont *et al.* (2007) reported that in OA, the synovial fluid appears as low viscous because of decreased concentration of hyaluronic acid concentration as well as decrease in chain length and molecular weight.

Anirudh and Ranganath (2015) reported that the synovial fluid volume increases in inflammatory arthropathies, whereas in osteoarthritis, the volume may be within the normal range.

Wood and Gibson (2020) reported that the physical parameters of synovial fluid should be noted immediately after collection and the volume of fluid collected in osteoarthritis was within the normal range.

2.6.4.2 Synovial staining

MacWilliams and Friedrichs (2003) reported that synovial fluid staining with Giemsa in osteoarthritis revealed the presence of macrophages, neutrophils and monocytes in the smear stained with Giemsa.

Mahaffey (2002) reported that synovial fluid staining in OA horse revealed the presence of synovial macrophages with metachromatic granules and normal neutrophils and monocytes.

Steel (2008) reported that the stained smear of septic arthritis revealed the presence of numerous neutrophils, whereas in non-inflammatory arthropathies like Degenerative Joint Diseases (DJD), the smears revealed normal macrophages, neutrophils and monocytes.

2.6.4.3 Biochemical parameters

2.6.4.3.1 Total protein

MacWilliams and Friedrichs (2003) reported that normal joint has Total Protein (T.P) of 1.5-3.0 g/dl. In OA cases, the synovial analysis might be normal or may show minor alterations and synovial fluid total protein may increase or remain normal in dogs with OA.

Xu *et al.* (2009) reported that in OA patients, the synovial fluid analysis showed an increased level of total protein.

Anirudh and Ranganath (2015) reported that synovial fluid analysis should be an integral part of any diagnostic evaluation of an animal with lameness, especially the animal with joint effusion. In Degenerative Joint Diseases (DJD) conditions, the total protein may be within the normal range in dogs.

Olsen *et al.* (2019) in their study reported that the total protein concentration may show minor alterations in osteoarthritis dogs.

2.6.4.4 Cell study

2.6.4.4.1 Total and differential cell count

Fernandes *et al.* (2002) reported that the normal WBC in synovial fluid ranges from 0-2900/mm^3 and has very few red blood cells. Monocytes are frequently seen cells in the synovial fluid and less commonly seen cells are polymorphonuclear cells (<10%) and indicated the contamination by the blood. In Degenerative Joint Diseases (DJD) conditions, the monocyte-macrophage, phagocytic count increases in synovial fluid.

MacWilliams and Friedrichs (2003) reported that total nucleated and differential cell counts might be normal to mildly elevated (>5000/ ml) in dogs with OA.

Olsen *et al.* (2019) in their study reported analysis of synovial fluid in OA dog revealed only minor impacts and some of these effects could be attributable to typical change in cell counts in joints undergoing recurrent arthrocentesis.

Wood and Gibson (2020) informed that normal synovial fluid in cats and dogs has a cellularity of 3.0 10^9 /L and 1.0 10^9 /L. Lymphocytes, macrophages and synovial cells make up the majority of the mononuclear cells. Less than 5–10% of cells are neutrophils in most cases and osteoarthritic lesions are common ailments in dogs and cats, which can be detected by examining synovial fluid.

2.6.5 Radiography

Adams *et al.* (2000) reported that ventro-dorsal (VD) view radiographs of the pelvis would be the most commonly used radiographs for evaluation of the canine hip joint osteoarthritis.

Reed (2002) in his diagnostic study reported that radiographic signs of osteoarthritis include erosion of subchondral bone, joint capsule thickening, formation of joint spurs and joint mice and in extreme cases of osteoarthritis, the bone may be misshaped but all these signs can be seen only in the advanced stages of the disease. Further opined that, early osteoarthritis does not produce any visible radiographic changes.

Peach *et al.* (2005) reported that radiographic evaluation as the gold standard method for the evaluation of osteoarthritis in dogs and stated that the common sign is the loss of joint space and the two bones appear close to each other because of loss of the cartilage.

Lorenz and Richter (2006) reported that radiographically observed signs in osteoarthritis include narrowing of joint space, osteophyte formation, subchondral bone sclerosis like thickening, fibrillation and cyst formation.

Lascelles and Robertson (2010) reported that ventro-dorsal view of pelvis radiography in osteoarthritis dog revealed new bone formation on the cranial acetabular rim, formation of new bone at the joint capsule attachment area on the femur neck.

Runge *et al.* (2010) selected the extended ventro-dorsal view of pelvis to know the presence of hip osteoarthritis and reported that evaluation can be done based on the presence of osteophytes, subchondral bone sclerosis and joint remodelling.

Rychel (2010) stated that radiographs play an important role in the diagnosis of osteoarthritis in dogs and helps to find bony changes in and around the joint and also to rule out certain conditions like bone lysis, proliferation and other abnormalities.

Pettitt and German (2015) concluded that radiography as one of the main tools for diagnosis of osteoarthritis in dogs and it gives information on osseous changes like subchondral sclerosis and osteophyte formation at the bone margins.

Meeson *et al.* (2019) reported that osteoarthritic hips in both humans and dogs exhibit signs of advanced new bone development as well as sclerosis in the acetabulae and the area around the femoral head and neck.

Alves *et al.* (2021) conducted study on the correlation between the appearance of clinical signs and radiographic signs and reported that the common radiographic signs seen in hip OA were misshaped and irregular wear of the femur head, loss of the outline of acetabular rim, loss of the rounded appearance of the femur head, formation of osteophyte on acetabulum and on femur head and subchondral bone sclerosis and the clinical and radiographic signs occur symmetrically in naturally occurring OA in dogs.

2.6.6 Ultrasonography (USG)

Rademacher *et al.* (2005) conducted the ultrasonographic diagnosis of osteoarthritis in dogs and reported that the transducer should be placed on medioventral place of the hip joint to know the changes in joints like effusions or degenerative changes.

Rocha and Torres (2007) examined a 14-day old dog by using ultrasonographic method and visualized the articular structures and opined that ultrasound could be useful for evaluation of the joint morphological variations.

Boulocher *et al.* (2008) studied the ultrasonographical findings of knee joint in the experimentally induced ACLT rabbits and found that in severe OA, there would be synovial capsule thickening, synovial effusions and bone erosions.

Goronav (2012) concluded that radiographic signs in OA can be detected lately whereas ultrasound can be used to detect early signs like effusion, cartilage defects, and capsule fibrosis and also reported that both radiography and ultrasonography when used combinedly can provide a detailed view for diagnosis of canine osteoarthritis.

Bergamino *et al.* (2015) studied ultrasonographic findings of OA in dogs and reported that on the femoral head can be seen as hyperechoic area and the joint capsule can be seen as triangular space between the femoral head and acetabular rim and articular cartilage can be visualized as anechoic band on the femur head.

Wenham *et al.* (2014) reported that ultrasound avoids radiography and helps in identifying certain OA changes like synovial hypertrophy, inflammation, osteophytes and also helps in differential diagnosis of certain bone disorders like osteoarthritis and cysts on bone.

Roemer *et al.* (2020) reported that in cases of knee OA, ultrasound offers diagnostic information above radiography, chiefly through its capacity to sensitively portray effusion and synovitis as well as directly detect structural abnormalities in cartilage and meniscus and for detecting tibiofemoral osteophytes and opined that ultrasound as more sensitive than radiography and has a high sensitivity for detecting morphologic articular cartilage deterioration in the anterior medial femoral condyle.

Singh *et al.* (2021) reported that high-frequency ultrasound imaging would be a non-invasive, widely available and reasonably priced tool for assessing periarticular and superficial intra-articular abnormalities in knee OA. Further reported that the most common imaging result on ultrasonography is osteophytosis, followed by medial meniscal extrusion, effusion, and lateral meniscal extrusion.

2.6.7 Computerised tomography (CT)

Chalmers *et al*. (2006) reported that with advanced osteoarthritis of the hip joints, trabecular thickening of the subchondral bone and femoral neck would be identified.

Kalichman and Hunter (2007) reported that in CT of osteoarthritis, the common findings recorded were sclerosis of the subchondral bone, bony erosions. apophyseal misalignment, osteophyte development and facet hypertrophy.

Wenham *et al.* (2014) reported that, for diagnosing OA in dogs, CT may provide a better view of subchondral bone cysts, and osteophytes and can also show calcified cartilage, the subchondral bone plate and trabecular subchondral bone when compared to other techniques like MRI and radiography.

Demehri *et al.* (2016) reported that cortical and medullary bone microarchitecture can be depicted very well using CT and it has proven to have a significant role in the evaluation of OA when compared to MRI and by assessing bone density and illuminating the subchondral

mineralized structures, further CT is very useful in revealing and comprehending the pathogenesis of knee OA.

Roemer *et al.* (2020) conducted CT on osteoarthritic dog and revealed bone trabecular remodelling, subchondral cysts and bone sclerosis and in addition to subchondral bone alterations and stated that CT is helpful in identifying tissue mineralization chondrocalcinosis, which is hypothesised to contribute to the onset and progression of illness.

Jone *et al.* (2022) reported that because the CT with advances in scanner technology, it is becoming possible to detect and quantify even a single osteophyte as well as examine subchondral bone alterations that was not possible with 2D plain radiography.

2.7 Treatment

2.7.1 Non- Steroidal Anti-inflammatory Drugs (NSAIDs)

Mclaughlin and Roush (2002) reported that anti-inflammatory drugs have been shown to be the cornerstone for medical therapy of OA in dogs, since they aid to relieve pain, reduce synovitis and reduce lameness and stated that NSAIDs are the most widely prescribed anti-inflammatory drugs for the treatment of OA.

Moreau *et al.* (2003) reported that meloxicam has proven to be helpful in dogs with severe OA, particularly for stifle arthritis, in terms of improved gait and capacity to live a normal life, as well as the absence of side effects.

Debbie (2006) reported that NSAIDs are group of anti-inflammatory drugs used for management of chronic pain, especially in OA cases and commonly used in veterinary practice are carprofen, meloxicam, tepoxalin, ketoprofen, tolfenic acid, phenylbutazone, cinchophen, vedoprofen and deracoxib.

McCarthy *et al.* (2007) reported that OA dogs treated with carprofen showed significant improvement in weight bearing and joint mobility by day 14 and alleviation in joint pain by day 42.

Johnston *et al.* (2008) reported that the most commonly prescribed pain relievers for OA are the nonsteroidal anti-inflammatory medications (NSAIDs) and carprofen at a dose of

2.2 mg/kg twice daily or 4.4 mg/kg once daily, depending on the weight of the patient had good result.

Malek *et al.* (2012) reported that for the treatment of canine OA, NSAIDs are frequently used.

Ranganath (2012) in his study reported that NSAIDs are frequently used to treat osteoarthritic pain in dogs resulted due to changes in the joints, bones and surrounding tissues and most commonly used ones are phenylbutazone, buffered aspirin, carprofen, etodolac, ketoprofen and meloxicam.

Alves *et al.* (2017) in their study on comparing the efficacy of carprofen and joint supplements, reported that the dogs receiving carprofen showed good improvement in alleviation of pain than joint supplements.

Taguchi *et al.* (2018) reported that NSAIDs are the standard pain management drugs used to treat OA dogs and stated that carprofen as the better choice for dogs with moderate OA.

Whittem *et al.* (2021) in their study reported carprofen as a licensed NSAID used for treating OA dogs, which can be administered at the dose rate of 2-4 mg/kg daily with a loading dose of 4mg/kg on the first day of therapy and noticed improvement in the lameness score by day 28.

2.7.2 Corticosteroids

Hazewinkel (2006) reported that glucocorticoids can help to reduce inflammation and joint discomfort in cases of synovitis as they reduce cartilage regeneration and however, long-term or recurrent use of corticosteroids, particularly noncrystalline corticosteroids intra-articular at higher doses is contraindicated.

Scott (2007) reported that glucocorticoids as the potent anti-inflammatory agents and may be used in dogs with acute OA flare-ups, severe OA unresponsive to NSAIDs or dogs that cannot tolerate NSAIDs. Further stated that long-term treatment may result in cartilage degeneration and deterioration of clinical signs by decreasing the collagen and matrix proteoglycans.

Ranganath (2012) reported that corticosteroids suppress chondrocyte mechanisms and alter matrix protein composition by lowering proteoglycan and collagen formation in OA cases. Further advised that long-term corticosteroid administration may have negative systemic effects, as well as harmful effects on cartilage, hence they should be rarely used to treat cartilage injury or DJD.

Bullock *et al.* (2018) reported that corticosteroids are more powerful anti-inflammatory drugs than NSAIDs. However due to their more negative effects they should be recommended only for a brief length of time at low dosages.

Verrico *et al.* (2020) reported that, presently most often used medicines for OA therapy in animals were NSAIDs and glucocorticoids and these two categories of medications have comparable effects as both have anti-inflammatory effects, have direct effects on cartilage metabolism.

Alves *et al.* (2021) reported that for several decades, intra articular corticosteroids have been utilised to relieve pain and inflammation associated with OA and adjacent tissues in case of dogs.

2.7.3 Nutraceuticals

Hawks (2002) reported that glucosamine is the amino monosaccharide nutrient and a precursor to the glycosaminoglycan disaccharide unit in cartilage matrix and when consumed, it was 87 percent bioavailable and apparently functions by providing a regulatory stimulus as well as raw materials for glycosaminoglycan (GAG) production. It also increased the production of GAGs, prostaglandins and collagen by chondrocytes and fibroblasts and promotes the production of hyaluronic acid by synoviocytes and synovial fibroblasts.

Goggs *et al.* (2005) investigated nutraceutical therapies for degenerative joint diseases and concluded that dietary supplementation programmes and nutraceuticals used in conjunction with nonsteroidal anti-inflammatory drugs (NSAIDs) may provide significant benefit to patients suffering from joint disorders such as osteoarthritis (OA) and osteochondritis dissecans (OCD).

Clegg *et al.* (2006) reported that chondroitin sulphate has been demonstrated to relieve pain, improve joint mobility and promote joint healing in persons with OA.

McCarthy *et al.* (2007) discovered that dogs given glucosamine and chondroitin Sulfate saw statistically significant improvements in discomfort, weight-bearing and severity of the disease by day 70 post treatment. They also reported that glucosamine and chondroitin sulphate had a beneficial therapeutic impact in dogs with osteoarthritis.

Ranganath (2012) reported that glucosamine is the primary sugar present in glycosaminoglycans and hyaluronate, both of which were essential building blocks in the synthesis and maintenance of articular cartilage. Chondroitin promotes glycosaminoglycan production and inhibits joint-damaging enzymes. He further informed that glucosamine and chondroitin provide the building blocks for cartilage-forming cells to create new cartilage and repair damaged cartilage in dogs suffering from osteoarthritis.

Lamani *et al.* (2019) reported that nutraceuticals are found to be useful in the treatment of osteoarthritis. Furthermore, nutraceuticals formulations supplied to Group A dogs for 90 days are more successful than steroidal therapy given to Group B dogs in relieving weight bearing, discomfort and lameness in osteoarthritis.

Johnson *et al.* (2020) reported human medical studies have shown that glucosamine promotes to cartilage growth and repair, lowers inflammation and slows cartilage degradation and chondroitin sulphate enhances cartilage elastic properties and minimises painful joint swelling and reduces pain symptoms linked with osteoarthritis in dogs.

2.7.4 Ashwagandha

According to Dragos *et al.* (2017), *Withania somnifera*, commonly known as Ashwagandha, which belongs to the steroid class of phytochemicals, a strong anti-osteoarthritic and anti-inflammatory herb in Ayurveda. It inhibits liposaccharide S-induced synthesis of pro-inflammatory cytokines (TNF-α, IL-1, IL-12) in peripheral and synovial fluid mononuclear cells and also has been proven in in-vivo experiments to block collagenase activity

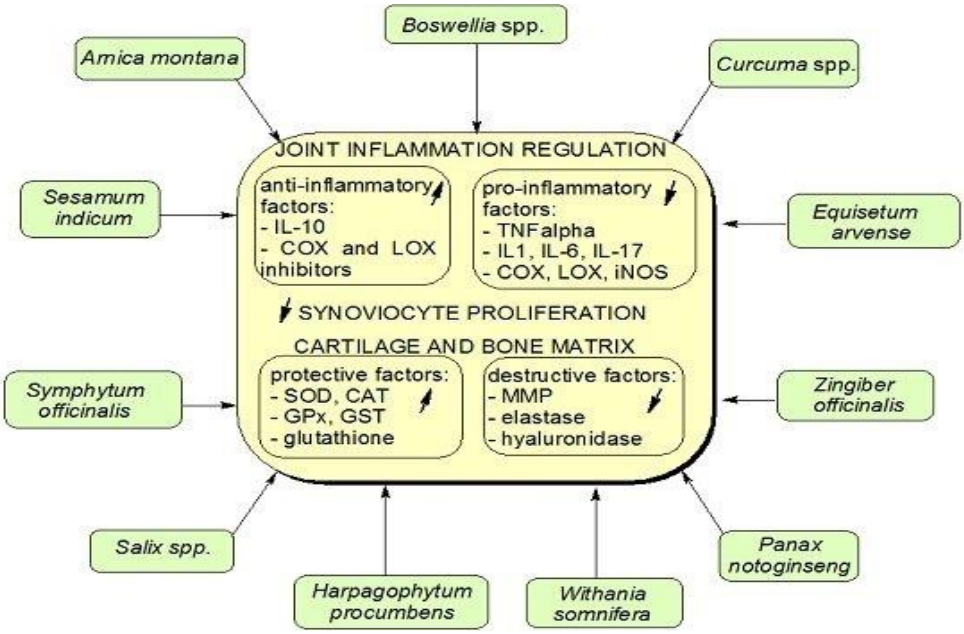

Fig. 2.4: Effect of various phytochemicals on Joint disorders (Dragos *et al.* (2017)

against the degradation of bovine achilles tendon type 1 collagen, which aids in the treatment of joint diseases (Fig. 2.4). In in-vivo investigations, animals given root powder had a protective impact on bone collagen in arthritic rats.

According to Sharma *et al*. (2020), *Withania somnifera*, a well-known anti-inflammatory plant used to treat arthritis and works successfully by lowering the rapdity of pain and disability scores in osteoarthritis patients while having no hazardous side effects. In comparison to NSAIDs, the Aswagandha is a potential anti-inflammatory and anti-arthritic medicine because they modulate the levels of inflammatory drugs and enzymes effectively without any adverse effects.

Sachin *et al.* (2017) conducted studies on Ashwagandha benefits in atheletic humans and reported that at the dose of 1000 mg/kg showed improvement in pain than other dose recommendations. They also reported that this herbal preparation significantly reduced the severity of pain and disability in 42 humans affected with arthritis.

Paul *et al.* (2021) reported that *Withania somifera*, also known as Ashwagandha in Sanskrit, has been widely utilised as an herbal therapy. The roots have a distinct wet horse odour and fruits seeds and leaves can also be used as medicines. (Fig. 2.5 and 2.6). Ashwagandha has been used to treat stress, sleeplessness and depression. According to reports, the roots include 0.13-0.31 percent alkaloids, volatile oils, starch, amino acids, reducing sugars and steroids. Fruits, in addition to roots, are used to cure skin diseases. In female balb mice, oral treatment of 500-1000 mg/kg body weights is found to be efficient in decreasing proinflammatory mediators such as cytokines, IL-6, TNF- (tumour necrosis factor), and ROS (reactive oxygen species).

Fig. 2.5: *Withania somifera* fruits and seeds (Paul *et al.*, 2021)

Fig. 2.6: *Withania somifera* roots and leaves (Paul *et al.*, 2021)

2.7.5 Physical rehabilitation techniques

2.7.5.1 TENS (Transcutaneous electrical nerve stimulation)

Levine and Bockstahler (2014) reported that the high frequency and short pulse duration TENS (50-150Hz, 2-50 microsecond pulse length) is extensively utilised in canine practise. Burst mode TENS is a series of pulses that function at 1-4 Hz and 40-150 Hz frequencies and there are both conventional and acupuncture-like TENS. A frequency range of 50-150 Hz is advised for acute pain and 1-10 Hz for chronic pain. In acute cases of OA, conventional TENS can be used 1-2 times daily for 15 minutes.

Hanks *et al.* (2015) reported that there are many orthopaedic and neurological conditions where TENS can be utilised to treat pain. It has been used to treat osteoarthritis, musculoskeletal discomfort and acute pain in humans. They also reported that high-frequency TENS and TENS with frequencies lower than 10 Hz had a substantial impact on the treatment of musculoskeletal pain. The combined efficacies of conventional TENS and acupuncture-like TENS were demonstrated to reduce pain and stiffness in their study on osteoarthritis of the knee.

Millis (2015) reported that TENS is an inexpensive, safe, non-invasive modality with few side effects that can be used to treat a variety of painful conditions. In humans, a high frequency i.e..40-50 Hz to 50-100 microsecond pulse width were available, where as in veterinary practice, conventional TENS is most commonly used as they cause less anxiety and more comfortable to the dogs.

King (2018) reported that Transcutaneous Electrical Nerve Stimulation (TENS) is used to relieve pain through the stimulation of sensory nerve fibres and in dogs with osteoarthritis TENS resulted in a considerable increase in weight bearing and ground reaction forces immediately following treatment, as well as the TENS has the advantage of being able to be used on a regular basis, numerous times each day. Chronic pain conditions such as osteoarthritis can also benefit from the use of TENS for pain management.

Flaherty (2019) reported that TENS is the administration of low-voltage electrical impulses using a range of waveforms, frequencies, and amplitudes through electrodes placed on the skin. TENS has been utilised to treat both acute and reoccurring painful disorders.

2.7.5.2 Diathermy

Linchitz and Sorell (2003) stated that superficial heat using hot packs, heating pads, paraffin wax, hydrotherapy and radiant therapy produces heat only for the superficial tissues up to 0.5 cm from the skin, whereas in diathermy the heat may penetrate up to 3-5 cms and helps in relieving pain associated with OA in dogs.

Masiero *et al.* (2021) reported heat therapy as frequent treatment and classified as superficial or deep therapy. Further they also reported that Diathermy can make use of a variety of waves and the most prevalent were long-wave diathermy (LWD), short-wave diathermy (SWD) and microwave diathermy (MWD). Long-wave (LW) frequencies range between 3 and 300 kHz, short-wave (SW) frequencies range between 3 and 30 MHz, and microwave (MW) frequencies range between 300 and 3000 GHz and when correctly administered, high and deep temperatures are reached rapidly and for long time, with low risk of burns. Continuous SWD of frequency 8 MHz and power 200 W for 20 min showed an increase of intraarticular temperature from 34.4 to 39.4 °C and helps in relieving pain associated with osteoarthritis.

CHAPTER III

MATERIALS AND METHODS

The following materials and methods were used for the present investigation entitled "**Clinico-Diagnostic and Therapeutic Studies on Osteoarthritis in Geriatric Dogs**"

3.1 Materials

Materials for this present study consists of geriatric dogs, clinical samples (blood and synovial fluid), laboratory equipment and therapeutic agents.

3.1.1 Geriatric dogs

The present study was conducted among geriatric dogs (>6 years) that were presented with the history and clinical signs of lameness, inability to bear weight and exercise intolerance to Veterinary Clinical Complex (VCC), Campus Veterinary Hospital, College of Veterinary Science, Rajendranagar; Veterinary Hospital Bhoiguda and also dogs from the peripheral hospitals over a period of six months from January 2022-June 2022. In all the suspected geriatric dogs, detailed history was taken, followed by thorough physical and clinical examination was done and were subjected to different tests for confirmatory diagnosis of osteoarthritis. The dogs diagnosed positive for OA were further divided into 2 groups (Group A and B, consisting 10 animals in each) and treated with different therapeutic regimens involving the conventional therapy, nutraceuticals and physical rehabilitation techniques like diathermy, TENS and compared with the control group.

3.1.2 Clinical samples

Whole blood, serum samples and synovial fluid were collected from the osteoarthritis suspected dogs and subjected to suitable diagnostic protocols.

3.1.2.1 Blood

Whole blood was collected from cephalic or saphenous vein with the help of sterile disposable syringe and carried into heparin/EDTA coated vials for whole blood.

3.1.2.2 Serum

Whole blood was transferred into clot activator coated sterile serum vials. Serum vacutainers were kept undisturbed till serum separation and the serum was transferred to eppendorf tubes and labelled accordingly and stored in refrigerator at 4^0C for biochemical analysis.

3.1.2.3 Synovial fluid

About 0.2 ml of synovial fluid was collected following aseptic conditions and sample was transferred into EDTA coated vials and stored at 4^0C for synovial analysis.

3.1.3 Laboratory Equipment

3.1.3.1. Haematological analyzer

Blood samples were estimated on day 0, 15 and 30 with the help of automated haematology analyser IDEXX VetAutoread^{TM} supplied by Sowar Private Limited, New Delhi.

3.1.3.2 Biochemical analyzer

Serum samples were analyzed on day the of collection and on 15 and 30 days by using IDEXX Catalyst one serum analyser, supplied by Sowar Private Limited, New Delhi.

3.1.3.3 X-ray machine

X-ray of ventrodorsal projections of joints were obtained to visualize the affected joint using Heliophos-D 500 mA X-ray machine.

3.1.3.4. Ultrasound scanner

Ultrasonography of joints were performed using Z5 Vet Mindray Diagnostic Ultrasound, supplied by Shenzhen Mindray Bio-medical Electronics Co., ltd, China.

3.1.3.5 Diathermy unit

Diathermy was performed by using BIOTECH diathermy unit, supplied by Physico Future International, New Delhi.

3.1.3.6 Trans cutaneous electrical nerve stimulation (TENS) unit

TENS was performed by using BRIO TENS unit, model no: 02007 supplied by Biotech India, Mumbai.

3.1.3.7 CT scanner

CT was performed by using Siemens Healthineers CT scanner, SOMATOM Force, supplied by Edge Medical Solutions, New Delhi.

3.1.3.8 Other materials

Other instruments like EDTA vials, Clot activator serum vials, Eppendorf tubes, Giemsa stain, Syringes of 22 and 23 gauze were also used in the present study.

3.2. Methods

3.2.1. Physiological parameters

Physiological parameters like rectal temperature (^{0}F), heart rate (beats/min) and respiratory rate (breaths/min) were recorded from the geriatric dogs on the day of visit and also 15 and 30 days during treatment period.

3.2.2 Clinical parameters

Clinical parameters like weight bearing on joints, joint mobility and pain scores and lameness scores were recorded on the day of visit and on 15 and 30 days of treatment.

3.2.2.1 Weight bearing

Weight bearing assessment in OA dog was done as per Nganvongpanit *et al.* (2013) and graded as follows:

1.Equal on all limbs while walking and standing

2. Normal standing, favours affected limb when walking

3.Partial weight bearing on standing and while walking

4. Partial weight bearing on standing and non-weight bearing while walking

3.2.2.2 Joint mobility

Joint mobility assessment in OA dog was done as per McCarthy *et al.* (2007) and recorded as follows:

1. Full range of mobility.
2. 10-20% range of motion with no crepitus.
3. 10-20% range of motion with mild crepitus.
4. 20-50% limitation range of motion with more crepitus.
5. >50% limitation in range of motion with high crepitus sounds.

3.2.2.3 Pain score

Pain score assessment in OA dog was done as per Nganvongpanit *et al.* (2013) and graded as follows:

1. No much abnormality.
2. Mild signs, dog turns head on recognition.
3. Moderate signs, dog pulls limb away.
4. Severe, dog becomes aggressive.
5. Dog won't allow for palpation.

3.2.2.4 Lameness score

Lameness score assessment in OA dog was done as Nganvongpanit *et al.* (2013) and graded as follows:

1. Walks normally.
2. Slightly lame when walking.
3. Moderately lame while walking.
4. Severely lame when walking.
5. Reluctant to rise and will not walk more than 5 paces.

3.2.3 Haemato-biochemical analysis

3.2.3.1 Hematology study

Whole blood of about 2 ml was collected from cephalic and saphenous vein with the help of sterile disposable syringe. Blood samples were estimated on day 0, 15 and 30 with the help of automated IDEXX VetAutoread^{TM} supplied by Sowar Private Limited, New Delhi (Fig. 3.1).

Hematological parameters analysed were Total erythrocyte count (10^6 cells/mm^3), Hemoglobin (g%), Total leucocyte count (10^3 cells/mm^3) and Differential leukocyte count (%) were estimated before starting therapy and also on 15 and 30 days of treatment.

Test procedure of complete blood picture:

- After collection of samples, fill the sample in micro-capillary tube and plug one end of micro-capillary tube.
- Place the micro-capillary tube in micro-capillary centrifuge and run @10,000 rpm for 5 minutes.
- Remove micro-capillary tube with the help of thumb forceps and place it in IDEXX VetAutoread^{TM} machine.
- Instrument starts analysing the sample and the results will be shown on the display screen.

3.2.3.2 Biochemical study

About 3 ml of blood was collected for serum in sterile vials and kept undisturbed for about 20 minutes and serum was separated and collected in eppendorf tubes for estimation of the biochemical parameters like serum creatinine (mg/dL), Alanine aminotransferase (IU/L), Alanine aminotransferase (IU/L) and C- reactive protein (g/dL) by using IDEXX Catalyst one serum analyser, supplied by Sowar Private Limited, New Delhi (Fig. 3.2).

Test Procedure of biochemical parameters:

- After collection of sample, Cenrtifuge it for 5 minutes.
- Collect the serum in small eppendorf tubes.
- Place the Eppendorf tube in serum slot and place the kit clips into the analyser.
- Run the machine and the results will be displayed on the computer attached to it.

3.2.3.3.1 Serum creatinine (mg/dL)

The estimation of serum creatinine was done by using IDEXX Catalyst one serum analyser, supplied by Sowar Private Limited, New Delhi with the help of CHEM 10 kit (Fig. 3.2. (b) supplied by Sowar Private Limited, New Delhi and the results were expressed in mg/dl.

3.2.3.3.2 Alanine aminotransferase (IU/L)

The estimation of serum alanine aminotransferase was done by using IDEXX Catalyst one, supplied by Sowar Private Limited, New Delhi with the help of CHEM 10 kit (Fig. 3.2. (b)) supplied by Sowar Private Limited, New Delhi and the results were expressed in IU/L.

3.2.3.3.3 Aspartate aminotransferase (IU/L)

The estimation of serum aspartate aminotransferase was done by using IDEXX Catalyst one, supplied by Sowar Private Limited, New Delhi with the help of CHEM 10 kit (Fig. 3.2. (b) supplied by Sowar Private Limited, New Delhi and the results were expressed in IU/L.

3.2.3.3.4 C- reactive protein (g/dL)

The estimation of C-reactive protein was done by using IDEXX Catalyst ones, supplied by Sowar Private Limited, New Delhi with the help of CHEM 10 kit (Fig. 3.2. (b)) supplied by Sowar Private Limited, New Delhi and the results were expressed in g/dl.

3.2.4 Synovial fluid Examination

Synovial fluid samples were collected from all the OA dogs by arthrocentesis after following strict aseptic conditions by using arthrocentesis needles. Length of the needle varied with the joint and size of the animal (22 or 23 G in for large dogs, 25 G for smaller dog). After collection, the sample were analyzed for various parameters like colour, viscosity, transparency, total and differential nucleated cell count and total protein by using IDEXX Catalyst one, supplied by Sowar Private Limited, New Delhi the help of CHEM 10 kit supplied by Sowar Private Limited, New Delhi. The detailed procedure was explained in Appendix-B.

Fig 3.1: a) Heamatological analyser b) micro-capillaryCentrifuge

Fig. 3.2: Automated serum analyser a) computer screen b) chem 10 kit

3.2.4.1 Procedure for synovial fluid collection

Osteoarthritis dogs were kept in lateral recumbency with the affected joints on top after shaving the arthrocentesis sites and prepared aseptically with a chlorhexidine/ povidone-iodine scrub and alcohol. In small and medium-sized dogs, a 40-mm needle was used for aspirating from all joints, in big and gigantic breeds, a larger needle is used for the hip and shoulder joints. Needle and syringe were linked before inserting into the joint (Fig. 3.3). A little amount of negative pressure was applied to aspirate fluid from a joint. Once a sample of synovial fluid was taken, the syringe's negative pressure was released and the needle was gradually removed and fluid collected was placed in EDTA vials (Clements, 2006).

3.2.4.2 Synovial fluid analysis

3.2.4.2.1 Physical examination

Physical characteristics such as colour, viscosity and transparency were noted as soon as the fluid was in the syringe or tube (Fig. 3.4) (Wood and Gibson, 2020).

3.2.4.2.2 Total and Differential Cell Count

In synovial fluid, total and differential cell count in the synovial fluid were carried out by IDEXX Catalyst one automated serum analyser as per the procedure described by Sugiuchi et al. (2015) and were expressed in cells/ml. The detailed procedure was described in Appendix-C.

3.2.4.2.3 Total protein

Total Protein in synovial fluid was determined by IDEXX Catalyst one automated serum analyser (Clements, 2006) and was expressed in g/dL. The detailed procedure was described in Appendix-C.

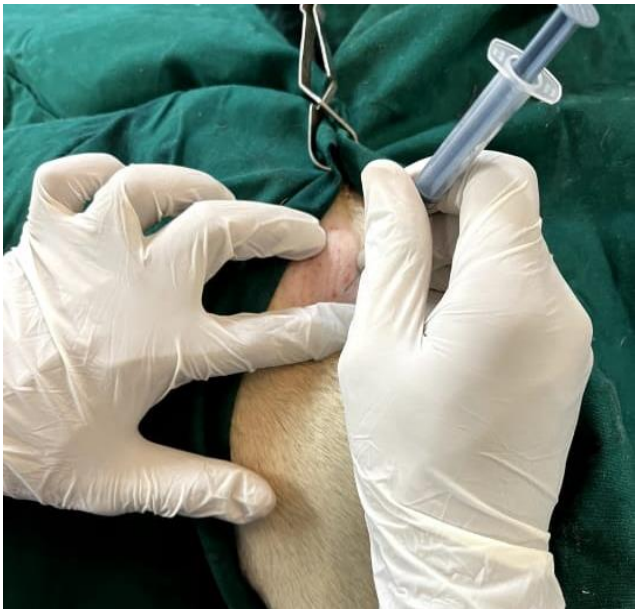

Fig. 3.3: Collection of synovial Fluid

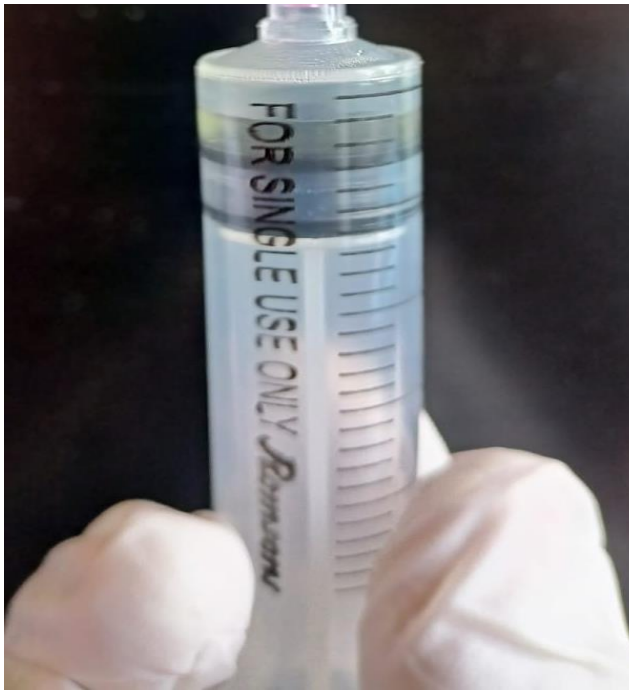

Fig. 3.4: Synovial fluid collected

3.2.4.2.4 Synovial fluid staining

Synovial fluid staining was done immediately after collection of sample and results were noted (MacWilliams and Friedrichs, 2003). The procedure was explained in detail in Appendix-B.

3.2.5 Radiographic evaluation

Radiological studies were carried out in the dogs before treatment and on the 15 and 30 days by using X-ray machine supplied by Heliophos-D 500 mA X-ray machine (Fig. 3.5). Ventro-dorsal views were taken to evaluate hip joint disorders and also to access the extent of the damage of joint in OA dogs. (Impellizeri *et al.*, 2000).

3.2.6 Ultrasonographic evaluation

Ultrasonographic evaluation of the OA joints was performed by using Shenzhen Mindray machine supplied by Bio-medical Electronics Co., ltd, China with a 7.5 to 12 MHz linear transducer (Fig. 3.6).

Procedure of ultrasonography:

Before conducting ultrasonography, the joint area should be properly shaved and dogs were placed in lateral recumbency with affected joint placed upwards. Probe was placed longitudinal to the affected hip joint and findings were recorded.

Ultrasonographic evaluation was done to know the soft tissue damage associated with OA, which cannot be visualized in radiography like joint effusions, thickening of joint capsule and cartilage, muscle and tendon tear. When imaging joints, sound waves move faster through bone and slower through joint fluid, making ultrasonography more beneficial for soft tissue structures. OA alterations like hyperreflective with uneven boundaries on the bone surface were demonstrated (Marino and Loughin, 2010).

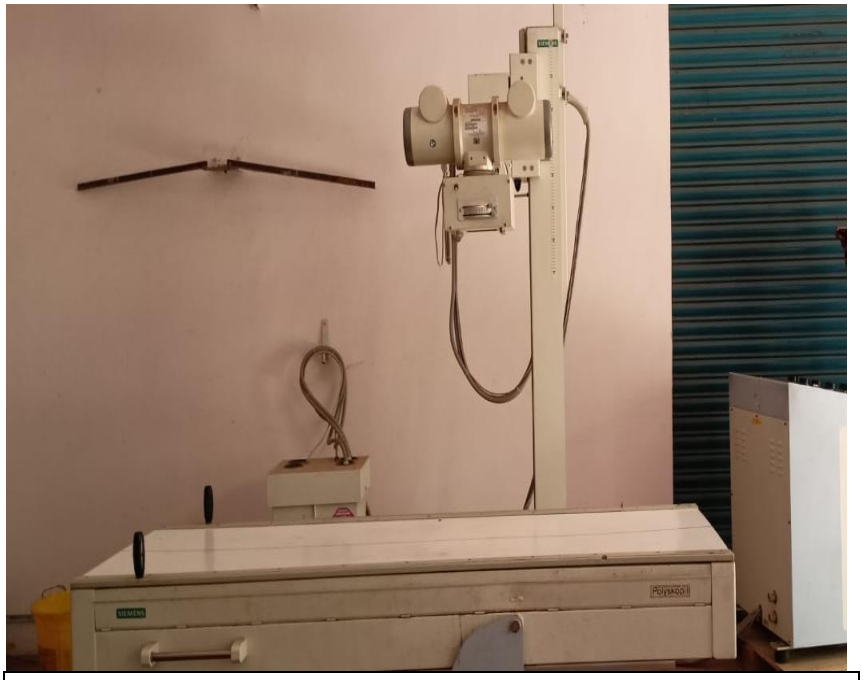

Fig 3.5: X-ray Machine

Fig. 3.6: Ultrasonography machine

Fig. 3.7: CT scanning in OA dog

3.2.7 Computerised Tomography (CT) Scanning

CT was performed by using Siemens Healthineers CT scanner SOMATOM Force, supplied by Edge Medical Solutions, New Delhi (Fig 3.7). The detailed procedure of CT scanning was explained in Appendix-C.

3.2.8 Therapeutic trials

Group A: Ten dogs were administered with Inj. Prednisolone @0.5-1.0 mg/kg for 5 days followed by diathermy @ twice a week for 4 weeks (detailed procedure was explained in Appendix-E) and commercially available nutraceuticals like Lubrihans @1tablet/10 kg body weight for a period of 30 days. Group B: Ten dogs were administered with Carprofen @ 2.2-4.4 mg/kg for 5 days followed by TENS @ twice a week for 4 weeks (detailed procedure was explained in Appendix-F) and Ashwagandha @ 500-1000 mg/kg for a period of 30 days.

3.2.9 Statistical Analysis

The results of clinical, haematological, biochemical and synovial parameters are presented as mean ± SE. Differences between groups and period means were calculated by a two-way analysis of variance (ANOVA) using computer based statistical programme (Graph pad prism).

Fig. 3.8: Long wave diathermy a) earth probe b) treatment probe

Fig. 3.9: TENS (Trans cutaneous Electrical Nerve Stimulator)

a. a product from MSD Animal Health company, Pune

contents: Prednisolone Acetate IP- 10mg and Benzoyl Alcohol IP- 0.945%

b. a product from Sava health care limited.

contents: Collagen Peptide type II 50 mg, Glucosamine sulphate potassium chloride 500mg, Sodium Hyaluronate 70 mg, Chondroitin Sulphate sodium 275 mg.

c product from Savavet, available in the name of CARODYL.

contents: Carprofen: 100 mg

d. a diffusible product from Herbal hills

contents: *Withania somnifera* root extract- 100 grams

CHAPTER IV

RESULTS

The present study **"Clinico-Diagnostic and Therapeutic Studies on Osteoarthritis in Geriatric Dogs"** was conducted to investigate the osteoarthritis in geriatric dogs. The incidence, risk factors, clinical signs, diagnosis and therapeutic management of osteoarthritis using nutraceuticals, herbal preparation and physical rehabilitation techniques like diathermy and TENS were studied, recorded and presented in this chapter.

4.1 Incidence

The Geriatric dogs (>6years) with the clinical signs of lameness presented at Veterinary Clinical Complex (VCC), Campus Veterinary Hospital, College of Veterinary Science, Rajendranagar; Veterinary Hospital Bhoiguda and also dogs from the peripheral hospitals over a period of six months from January 2022-June 2022 were formed basis for the study. Among the 3040 geriatric dogs presented, 350 dogs were with the signs of lameness, inability to bear weight and exercise intolerance and were diagnosed positive for osteoarthritis, indicating the incidence rate of 11.51%.

4.1.1 Age wise incidence

Out of 350 positive cases of osteoarthritis, the age wise incidence was categorized as 6-10 years, 10-15 years and more than 15 years and each group comprising of 105, 220 and 25 dogs with the incidence of 30%, 62.86% and 7.14%, respectively. The results were furnished in Table 4.1 and Fig. 4.1.

Table 4.1: Age wise incidence of Osteoarthritis in Geriatric Dogs

Sl. No.	Age	No. of. Dogs	Percentage (%)
1.	6-10 years	105	30%
2.	10-15 years	220	62.86%
3.	>15 years	25	7.14%
4.	**Total**	**350**	**100**

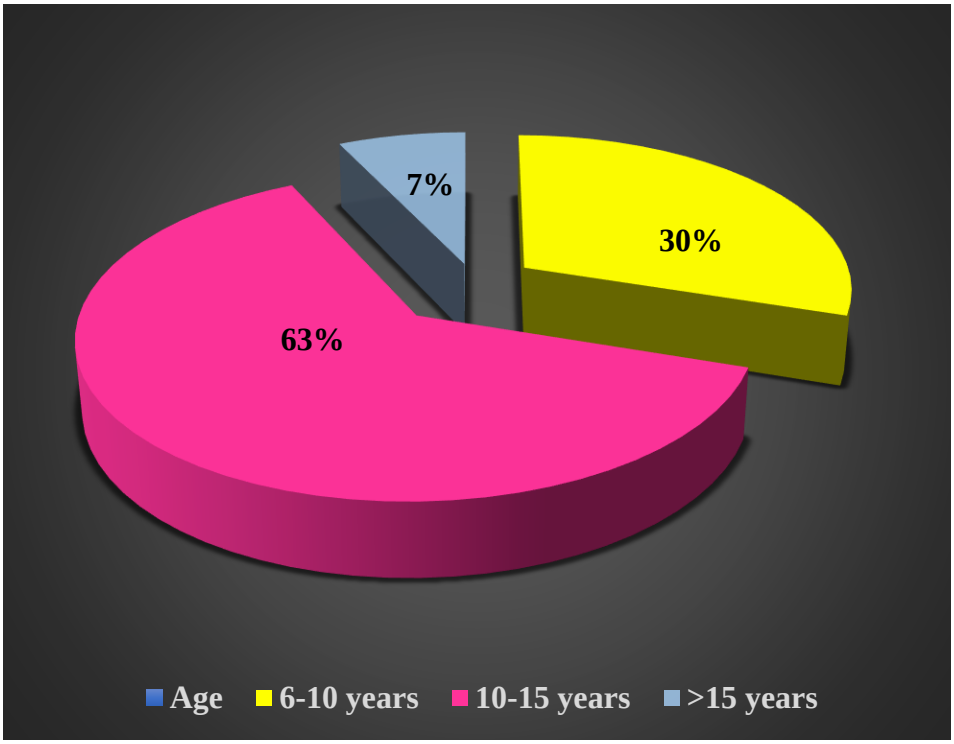

Fig. 4.1: Age wise incidence of Osteoarthritis in Geriatric dogs.

4.1.2 Breed wise incidence

In the present investigation, out of 350 positive cases, 160 Labrador Retriever, 72 German Shepherd, 38 Rottweiler, 33 non-descript, 25 Pug, 13 Spitz, 8 Golden Retriever and one Dalmatian were affected giving the breed wise incidence of 45.71%, 20.57%, 10.86%, 9.43%, 7.14%, 3.71%, 2.29% and 0.29%, respectively. The findings were furnished in Table 4.2 and Fig. 4.2.

Table 4.2: Breed wise incidence of Osteoarthritis in Geriatric Dogs

Sl. No.	Breed	No. of. Dogs	Hip joint	Elbow joint	Stifle joint	Percentage (%)
1	Labrador Retriever	160	135	20	05	45.71%
2	German shepherd	72	59	12	01	20.57%
3	Rottweiler	38	32	06	0	10.86%
4	Non-descript	33	25	07	01	9.43%
5	Pug	25	21	04	0	7.14%
6	Spitz	13	10	03	0	3.71%
7	Golden Retriever	08	06	02	0	2.29%
8	Dalmatian	01	01	0	0	0.29%
9	**Total**	**350**	**289**	**54**	**07**	**100**

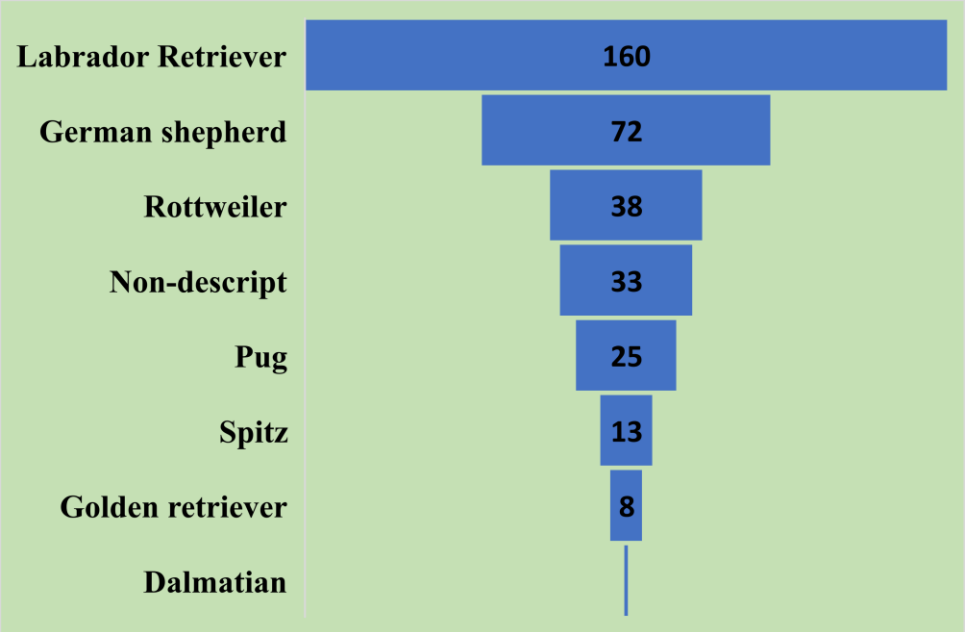

Fig. 4.2: Breed wise incidence of Osteoarthritis in Geriatric Dogs.

4.1.3 Gender wise incidence

In the present investigation, out of 350 positive cases, 210 (60%) were male dogs and 140 (40%) were females. The results were furnished in Table 4.3 and Fig. 4.3.

Table 4.3: Gender wise incidence of Osteoarthritis in Geriatric Dogs

Gender	No. of. Dogs	Percentage
Male	210	60%
Female	140	40%
Total	**350**	**100%**

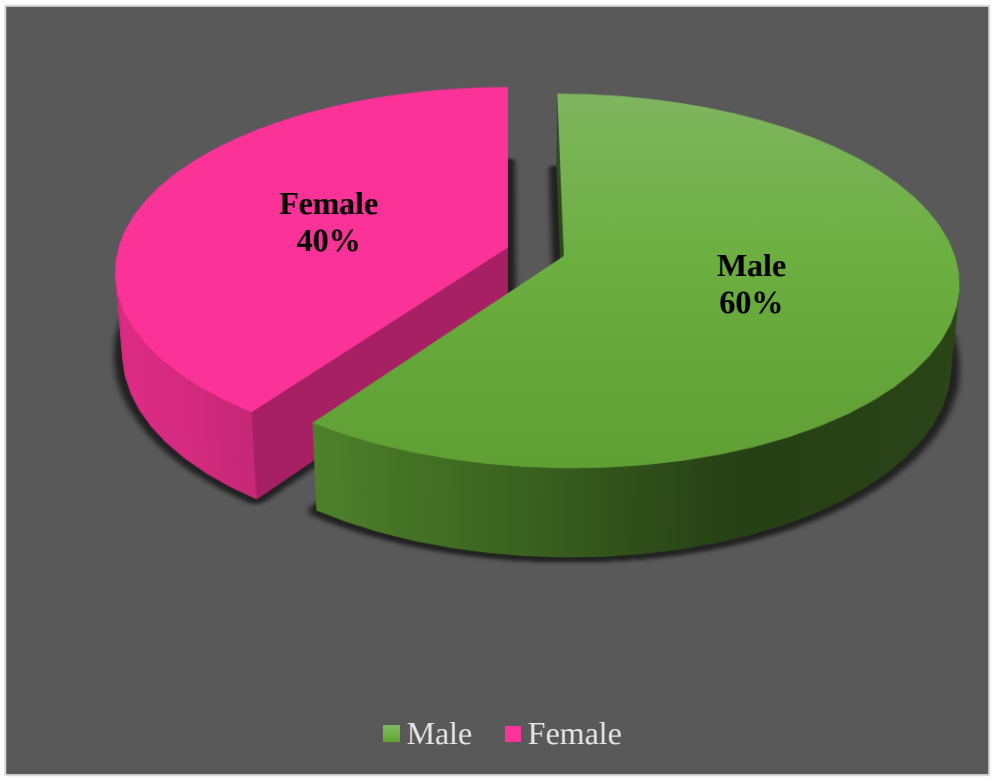

Fig. 4.3: Gender wise incidence of Osteoarthritis in Geriatric Dogs.

4.1.4 Joint wise incidence

With respect to joint wise incidence of osteoarthritis, hip joint was found to be more involved in osteoarthritis 289 (82.57%) dogs, followed by elbow joint 54 (15.43%) dogs and least involved joint was stifle 07 (2%) dogs.

Bilateral hip osteoarthritis was seen in 152 (43.43%) dogs, unilateral right hip osteoarthritis in 75 (21.43%), unilateral left hip osteoarthritis in 62 (17.71%), elbow joint osteoarthritis in 54 (15.43%) and stifle joint osteoarthritis in 07 (2%). The results were furnished in Table 4.4 and Fig. 4.4.

Table 4.4: Joint wise incidence of Osteoarthritis in Geriatric Dogs

Sl. No.	Joint affected	No. of. Dogs	Percentage (%)
1	Bilateral hip	152	43.43%
2	Unilateral hip joint (right)	75	21.43%
3	Unilateral hip joint (left)	62	17.71%
4	Elbow joint	54	15.43%
5	Stifle joint	07	2.00%
6	**TOTAL**	**350**	**100%**

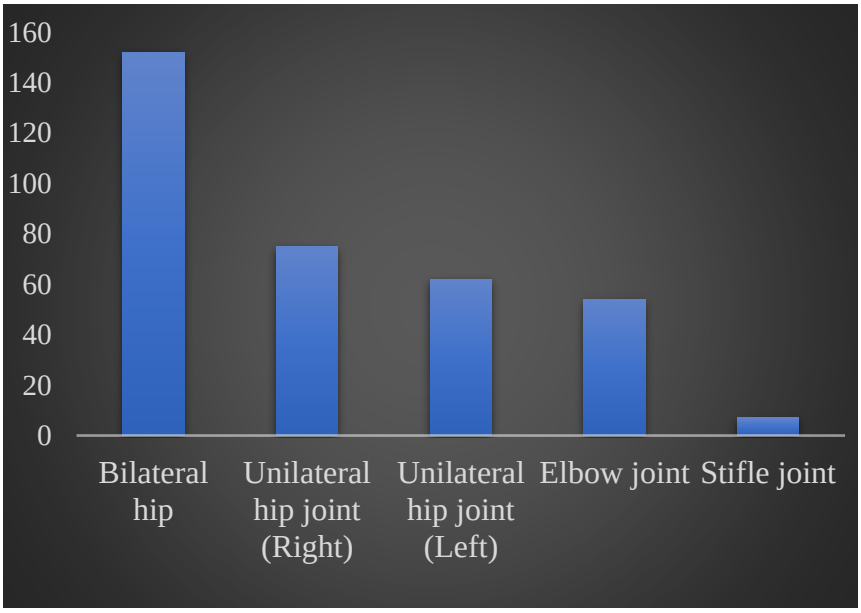

Fig. 4.4: Joint wise incidence of Osteoarthritis in Geriatric Dogs.

4.2. Risk factors

In the present investigation, the risk factors recorded for osteoarthritis were, overweight in 202 (57.71%) dogs, neutering in 45 (12.86%) dogs, slippery floor in 33 (9.43%) dogs, overuse of calcium in 30 (8.57%) dogs, heavy exercise in 20 (5.71%) dogs, underlying joint diseases in 15 (4.29%) dogs and hypothyroidism in 5 (1.43%) dogs. The results were furnished in Table 4.5 and Fig. 4.5.

Table 4.5: Risk factors associated with Osteoarthritis in Geriatric Dogs

Sl. No	Factor	No. of. Dogs	Percentage (%)
1	Over weight	202	57.71%
2	Neutering	45	12.86%
3	Slippery floor	33	9.43%
4	Over use of calcium	30	8.57%
5	Heavy exercise	20	5.71%
6	Underlying joint issues	15	4.29%
7	Hypothyroidism	05	1.43%
8	**Total**	**350**	**100**

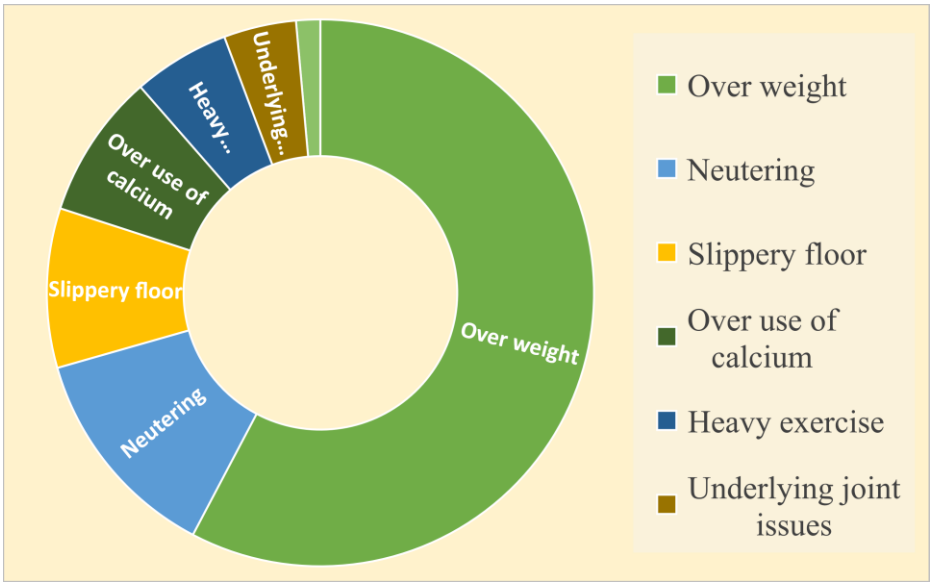

Fig. 4.5: Risk factors associated with Osteoarthritis in Geriatric Dogs.

4.3 Clinical signs

Prominent clinical signs observed in all the osteoarthritis positive cases were pain in 350 (100%), reluctant to move in 310 (88.57%), lameness in 250 (71.43%), sluggish rising in 180 (51.43%), difficulty in walking upstairs or jumping in 150 (42.86%), stiffness in 50 (14.29%) and reduced appetite in 20 (5.71%) dogs. The results were furnished in Table 4.6 and Fig. 4.6.

4.4 Physiological parameters

The mean rectal temperature (°F), respiratory rate (breaths/min) and heart rate (beats/min) of osteoarthritis dogs were presented in Tables 4.7 and 4.8 and Figures 4.9, 4.10 and 4.11.

4.4.1 Rectal temperature (°F)

The mean±SE of rectal temperature (°F) recorded in group I and group II dogs before therapy were 102.19±0.06 and 102.02±0.09, respectively and found no non-significant difference ($P>0.05$) when compared to the healthy control group (102.3±0.05). On day 15 and 30, the mean rectal temperature recorded in group I were 102.03±0.05 and 102.01±0.04 and in group II were 102.01±0.05 to 102.02±0.03. The mean rectal temperature in dogs in both the groups before and after therapy showed no significant difference ($P>0.05$) when compared to apparently healthy dogs. The results were furnished in the Table 4.7 and 4.8 and Fig. 4.9.

Table 4.6: Clinical signs exhibited by Osteoarthritis affected Geriatric Dogs

Sl. No.	Clinical signs	No. of animals	Percentage (%)
1	Pain	350	100 %
2	Reluctant to move	310	88.57 %
3	Lameness	250	71.43 %
4	Sluggish rising	180	51.43 %
5	Difficulty in walking upstairs or jumping	150	42.86 %
6	Stiffness	50	14.29 %
7	Reduced appetite	20	5.71 %

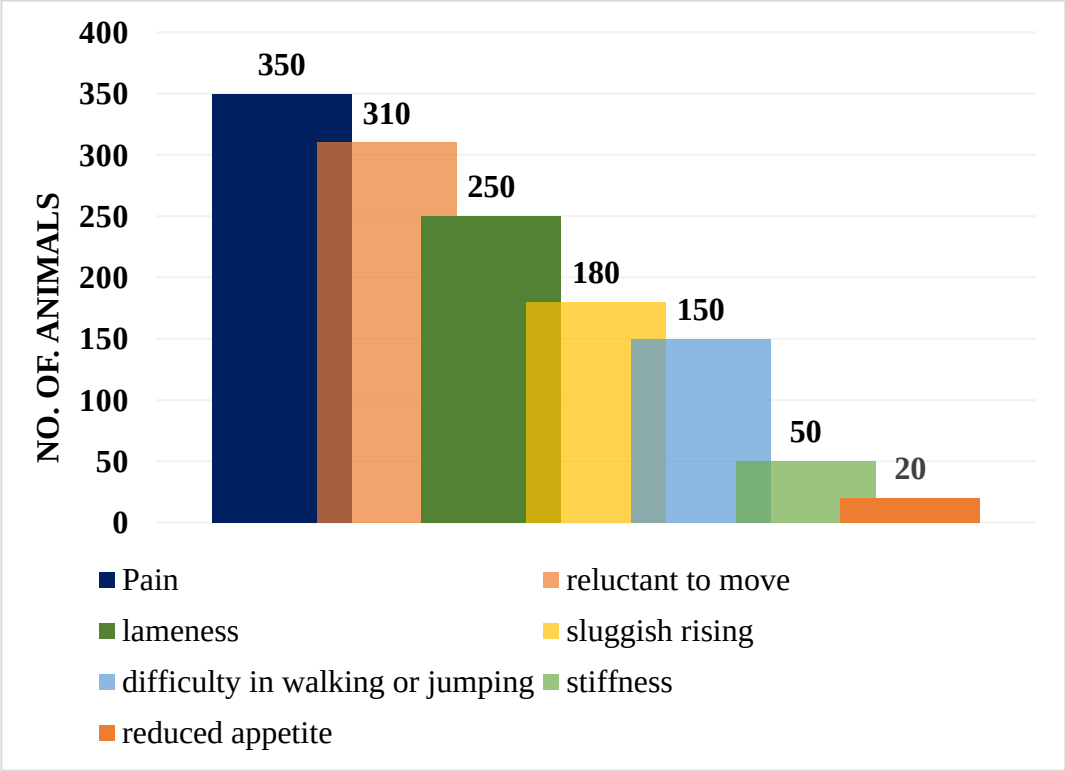

Fig. 4.6: Clinical signs exhibited by Osteoarthritis affected Geriatric Dogs

Fig. 4.7: Severely affected osteoarthritis dog showing the sign of reluctance to rise due to severe pain in the joint

Fig. 4.8: Osteoarthritis affected dog showing the sign of difficulty in walking due to pain in the joint

Table 4.7: Physiological parameters in Group I OA affected Geriatric Dogs

Sl. No	Parameter	Apparently healthy Dogs	Day 0	Day 15	Day 30
1	Temperature (°F)	102.3±0.05	102.19 ±0.06	102.03±0.05	102.01±0.04
2	Heart rate (beats/min)	99.5±0.02	98.5±0.30	99.49±0.40	97.9±0.29
3	Respiratory rate (breaths/min)	35.1±0.02	35.20±0.34	34.20±0.30	33.76±0.31

Note: Variations are non-significant.

Table 4.8: Physiological parameters in Group II OA affected Geriatric Dogs

Sl. No	Parameter	Apparently healthy Dogs	Day 0	Day 15	Day 30
1	Temperature (°F)	102.3±0.05	102.02±0.09	102.01±0.05	102.02±0.03
2	Heart rate (beats/min)	99.5±0.02	99.15±0.22	98.58±0.22	98.60±0.19
3	Respiratory rate (breaths/min)	35.1±0.02	35.05±0.26	33.98±0.23	34.68±0.25

Note: Variations are non-significant.

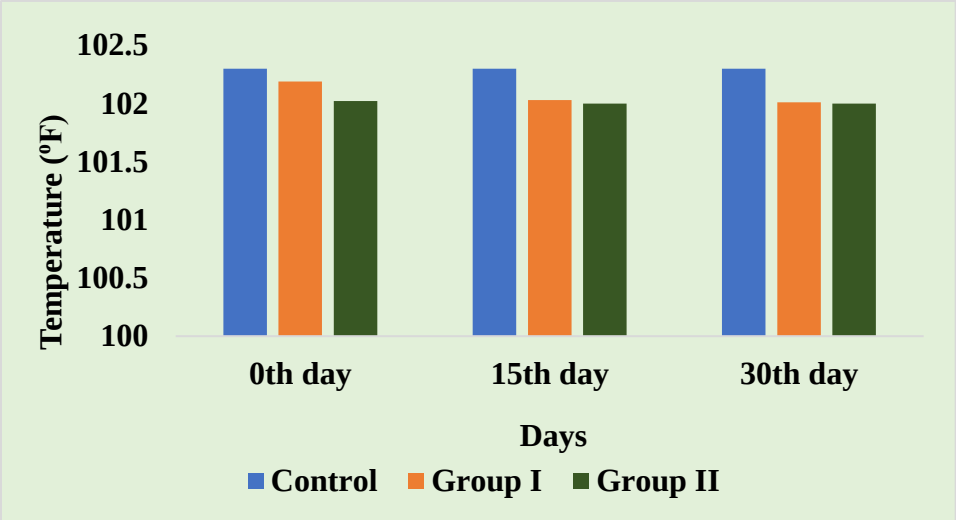

Fig. 4.9: Temperature (ºF) recorded in different groups of Geriatric dogs affected with OA.

4.4.2 Heart Rate (beats/Per min)

The mean±SE heart rate recorded in group I and group II dogs before therapy were 98.5±0.30 and 99.15±0.22, respectively and found no significant difference (P>0.05) when compared to the healthy control group (99.50±0.02). On day 15 and 30 after treatment, the mean heart rates recorded in group I were 99.49±0.40 and 97.9±0.29 and in group II were 98.58±0.22 and 98.60±0.19, respectively. The difference in mean heart rate in both the groups before and after therapy were non-significant (P>0.05) when compared to apparently healthy group. The results were furnished in the Table 4.7 and 4.8 and Fig. 4.10.

4.4.3 Respiratory Rate (breaths/min)

The mean±SE of respiratory rate recorded in group I and group II dogs before therapy were 35.20±0.34 and 35.05±0.26, respectively and found no significant difference (P>0.05) compared to the healthy control group (35.1±0.02). On day 15 and 30 after treatment, the mean respiratory rates recorded in group I were 34.20±0.30 and 33.76±0.31 and in group II were 33.98±0.23 and 34.68±0.25, respectively and reduced non-significantly (P>0.05) when compared to day 0. The results were furnished in the Table 4.7 and 4.8 and Fig. 4.11.

4.5 Clinical Parameters

4.5.1 Weight bearing

The mean of weight bearing score before therapy in group I and group II dogs were 4.2±0.26 and 4.4±0.20, respectively and was significantly high (P<0.01) when compared to apparently healthy dogs (2.3±0.51). However, after therapy, on day 15 and 30, the values in group I were 3.4±0.15 and 2.9±0.15 and in group II were 3.3±0.15 and 2.3±0.14, respectively showed significant reduction (P<0.05) when compared to day 0 and these reduced values were

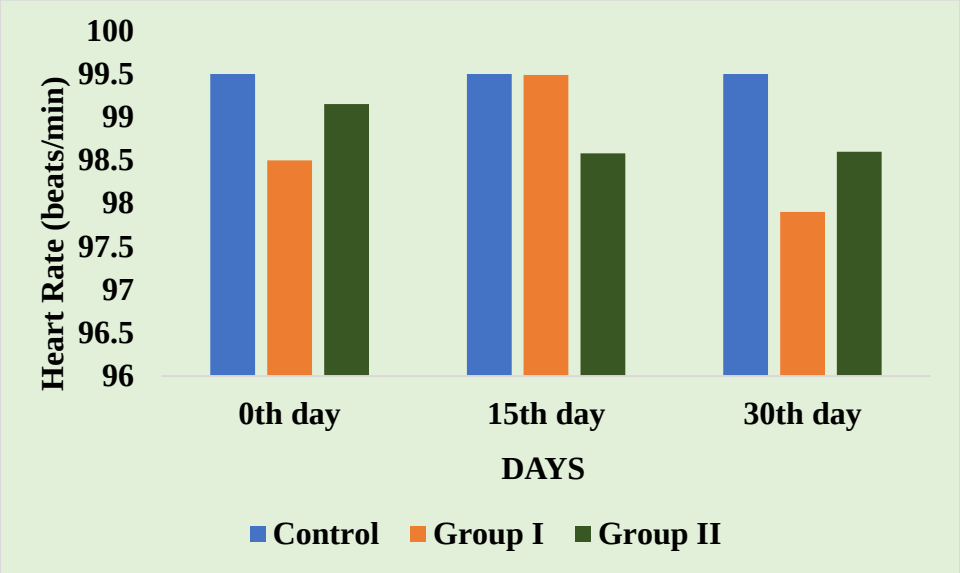

Fig. 4.10: Herat rate (beats/min) in different groups of Geriatric dogs affected with OA.

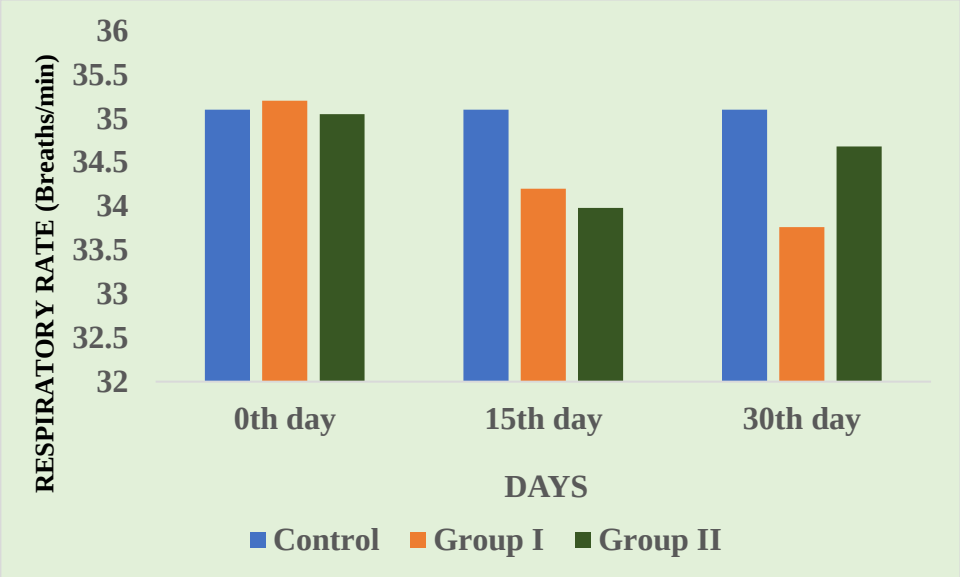

Fig. 4.11: Respiratory rates (breaths/min) in different groups of Geriatric Dogs affected with OA.

Table 4.9: Clinical parameters in Group I OA affected Geriatric Dogs

Sl. No.	Parameter	Apparently healthy Dogs	Day 0	Day 15	Day 30
1	Weight bearing	2.3±0.51	4.2±0.26**	3.4±0.15	2.9±0.15*
2	Joint motion	2.0±0.20	4.5±0.15**	3.2±0.12	2.8±0.14*
3	Pain score	1.9±0.05	4.4±0.15**	3.3±0.14	2.1±0.17*
4	Lameness score	2.5±0.04	4.5±0.15**	3.9±0.17	2.9±0.15*

Note: **Significant when compared to Apparently healthy dogs (P<0.01).

* Significant when compared to 0^{th} day (P<0.05).

Table 4.10: Clinical parameters in Group II OA affected Geriatric Dogs

Sl. No.	Parameter	Apparently healthy Dogs	Day 0	Day 15	Day 30
1	Weight bearing	2.3±0.51	4.4±0.20**	3.3±0.10	2.3±0.14*
2	Joint motion	2.0±0.20	4.18±0.12**	3.8±0.12	2.0±0.10*
3	Pain score	1.9±0.05	4.5±0.18**	3.5±0.15	1.9±0.04**
4	Lameness score	2.5±0.04	4.6±0.20**	3.3±0.17	2.4±0.14*

Note: **Significant when compared to Apparently healthy dogs (P<0.01).

* Significant when compared to 0^{th} day (P<0.05).

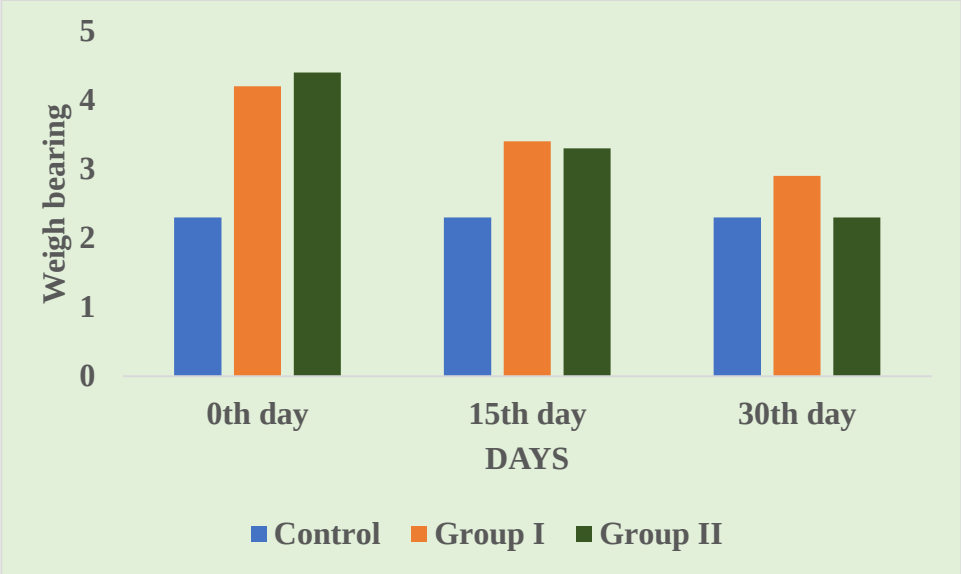

Fig. 4.12: Weight bearing in different groups of OA affected Geriatric dogs.

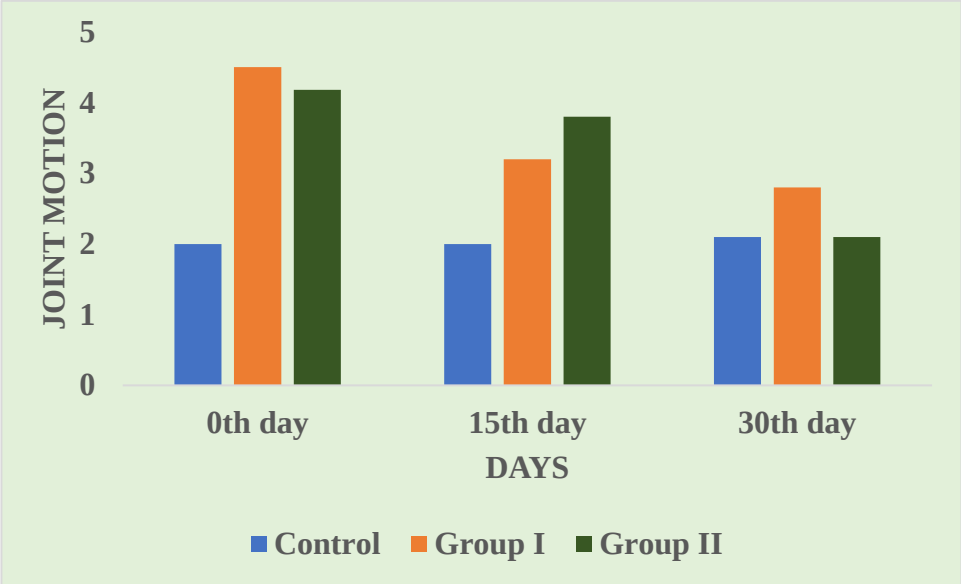

Fig. 4.13: Joint motion in different groups of OA affected Geriatric dogs.

comparable to apparently healthy dogs. The results were furnished in Tables 4.9 and 4.10 and Fig. 4.12.

4.5.2 Joint mobility

The mean of joint motion before therapy in group I and group II dogs were 4.5±0.15 and 4.18±0.10, respectively and was significantly high (P<0.01) when compared to apparently healthy dogs (2.0±0.20). However, after therapy, on day 15 and 30, the mean values in group I were 3.2±0.12 and 2.8±0.14 and in group II were 3.8±0.12 and 2.0±0.10 and showed significant reduction (P<0.05) when compared to day 0 and these reduced values were comparable to apparently healthy dogs. The results were furnished in Tables 4.9 and 4.10 and Fig. 4.13.

4.5.3 Pain Score

The mean of pain score before therapy in group I and group II dogs were 4.4±0.15 and 4.5±0.18, respectively and was significantly high (P<0.01) when compared to apparently healthy dogs (1.9±0.05). However, after therapy, on day 15 and 30, the mean values in group I were 3.3±0.14 and 2.1±0.17 and in group II were 3.5±0.15 and 1.9±0.04 and showed significant reduction (P<0.05) when compared to day 0 and these reduced values were comparable to apparently healthy dogs. The results were furnished in Tables 4.9 and 4.10 and Fig. 4.14.

4.5.4 Lameness Grading

The mean of lameness grading before therapy in group I and group II dogs were 4.5±0.15 and 4.6±0.20, respectively and was significantly high (P<0.01) when compared to apparently healthy dogs (2.5±0.04). However, after therapy, on day 15 and 30, the mean values in group I were 3.9±0.17 and 2.9±0.15 and in group II were 3.3±0.17 and 2.4±0.14 and showed significant reduction (P<0.05) when comparable to day 0 and these reduced values were comparable to apparently healthy dogs. The results were furnished in Tables 4.9 and 4.10 and Fig. 4.15.

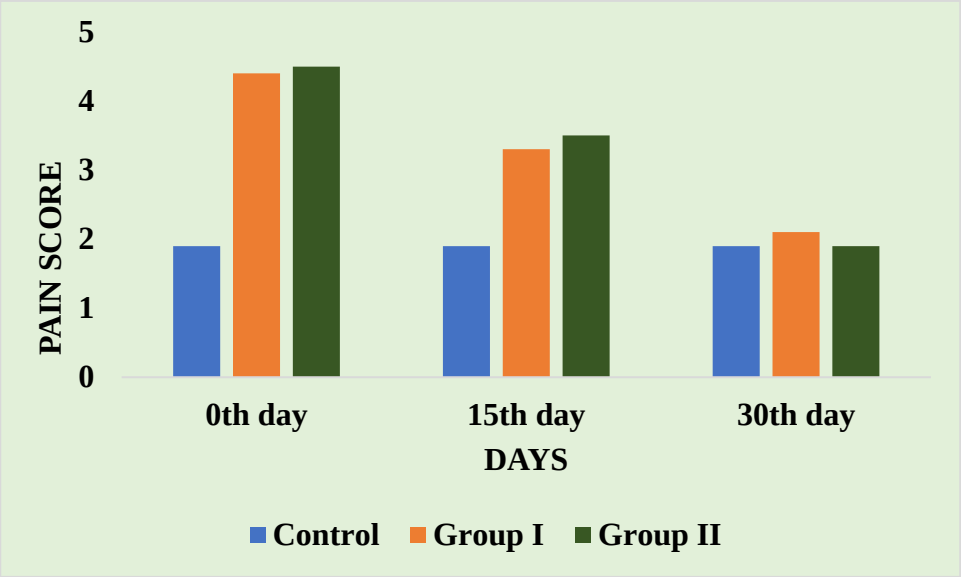

Fig. 4.14: Pain scores in different groups of OA affected Geriatric dogs.

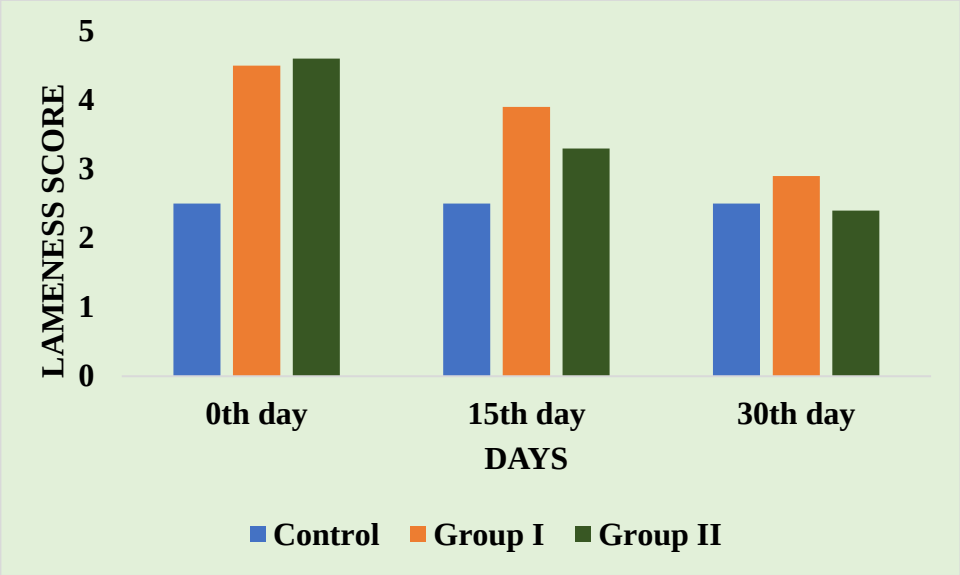

Fig. 4.15: Lameness scores in different groups of OA affected Geriatric dogs.

4.6 Haematology

4.6.1 Total Erythrocyte Count (10^6 cells/mm^3)

The mean±SE of total erythrocytic count (10^6 cells/mm^3) recorded in group I and group II dogs before therapy were 7.53±0.07 and 7.59±0.05, respectively and showed non-significant difference (P>0.05) when compared to apparently healthy control dogs (7.6±0.30). However, after therapy, on day 15 and 30, the mean±SE of total erythrocytic count recorded in group I were 7.52±0.06 and 7.60±0.07 and in group II were 7.54±0.04 to 7.6±0.05 and were comparable to apparently healthy dogs. The results were furnished in the Tables 4.11 and 4.12 and Fig. 4.16.

4.6.2 Haemoglobin (g%)

The mean±SE of haemoglobin (g%) recorded in group I and group II before therapy were 11.78±0.06 and 11.88±0.02, respectively and showed non-significant difference (P>0.05) when compared to the apparently healthy control dogs (12.0±0.21). However, on day 15 and 30 after therapy, the mean±SE of haemoglobin (g%) recorded in group I were 11.82±0.07 and 11.88±0.05 and in group II were 11.87±0.03 and 11.94±0.02 and were comparable to apparently healthy dogs. The results were furnished in Tables 4.11 and 4.12 and Fig. 4.17.

Table 4.11: Haematological findings in Group I OA affected Geriatric Dogs

S. No	Parameter	Apparently healthy Dogs	Day 0	Day 15	Day 30
1	TEC (10^6 cells/mm^3)	7.6±0.30	7.53±0.07	7.52±0.06	7.60±0.07
2	Hb (g%)	12.0±0.21	11.78±0.06	11.82±0.07	11.88±0.05
3	TLC (10^3 cells/mm^3)	11.88±0.02	11.76±0.05	11.60±0.04	11.71±0.05
4	Neutrophils (%)	72.52±0.12	72.52±0.24	72.51±0.10	72.52±0.02
5	Lymphocytes (%)	22.9±0.23	22.88±0.24	22.86±0.17	22.89±0.14
6	Eosinophils (%)	2.3±0.02	2.3±0.14	2.27±0.10	2.29±0.14
7	Monocytes (%)	2.27±0.05	2.25±0.02	2.26±0.05	2.26±0.02

Note: Variations are non-significant.

Table 4.12: Haematological findings in Group II OA affected Geriatric Dogs

S. No	Parameter	Apparently healthy Dogs	Day 0	Day 15	Day 30
1	TEC (10^6 cells/mm^3)	7.6±0.30	7.59±0.05	7.54±0.04	7.6±0.05
2	Hb (g%)	12.0±0.21	11.88±0.02	11.87±0.03	11.94±0.02
3	TLC (10^3 cells/mm^3)	11.88±0.02	11.73±0.04	11.70±0.05	11.87±0.04
4	Neutrophils (%)	72.52±0.12	72.55±0.28	72.45±0.10	72.51±0.11
5	Lymphocytes (%)	22.9±0.23	22.85±0.14	22.9±0.16	22.87±0.20
6	Eosinophils (%)	2.3±0.02	2.29±0.06	2.3±0.01	2.29±0.05
7	Monocytes (%)	2.27±0.05	2.24±0.04	2.25±0.03	2.28±0.01

Note: Variations are non-significant.

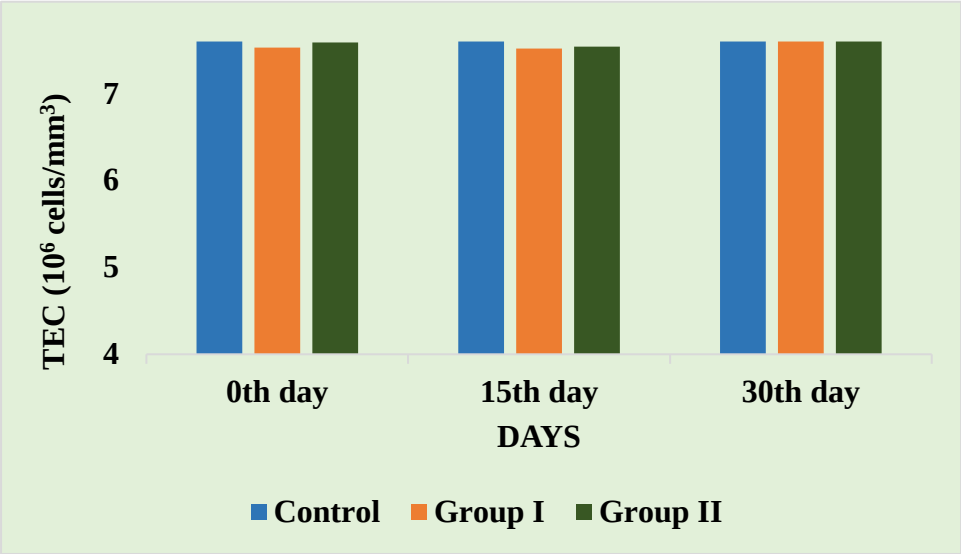

Fig. 4.16: Total erythrocytic count (10^6 cells/mm^3) in different groups of OA affected Geriatric Dogs.

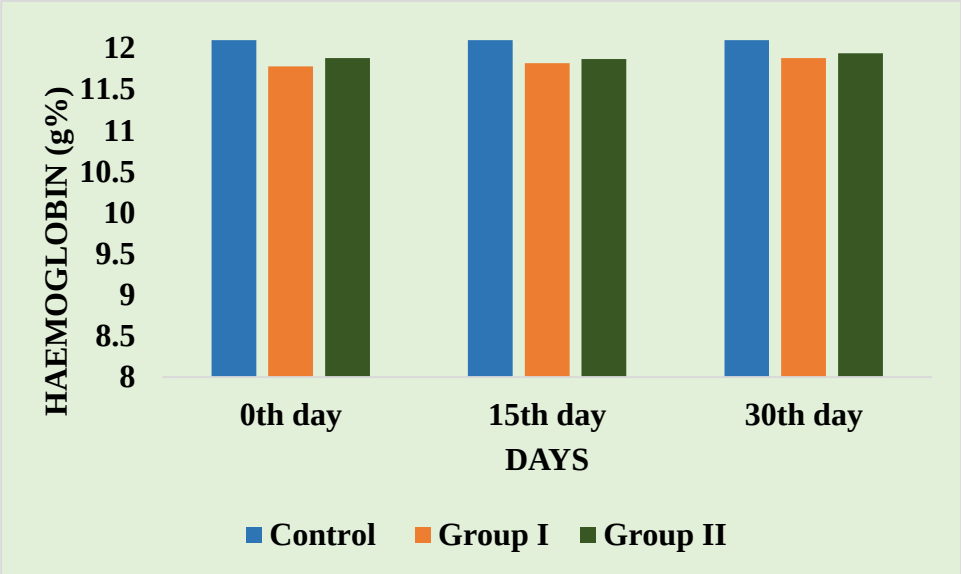

Fig. 4.17: Haemoglobin (g%) in different groups of OA affected Geriatric Dogs.

4.6.3 Total Leukocyte Count (10^3 cells/mm^3)

The mean±SE of Total Leukocyte Count (10^3 cells/mm^3) recorded in group I and group II dogs before therapy were 11.76±0.05 and 11.73±0.04, respectively and showed non-significant difference (P>0.05) when compared to the apparently healthy control dogs (11.88±0.02). However, after therapy, on day 15 and 30, the mean±SE of Total Leukocyte Count recorded in group I were 11.60±0.04 and 11.71±0.05 and in group II were 11.70±0.05 and 11.87±0.04 and were comparable to apparently healthy dogs. The results were furnished in Tables 4.11 and 4.12 and Fig. 4.18.

4.6.4 Differential Leukocyte count

4.6.4.1 Neutrophils (%)

The mean±SE of neutrophils (%) recorded in group I and group II dogs before therapy were 72.52±0.24 and 72.55±0.28, respectively and showed non-significant difference (P>0.05) when compared to apparently healthy control dogs (72.52±0.12). However, after therapy, on day 15 and 30, the mean±SE of neutrophils recorded in group I were 72.51±0.10 and 72.52±0.02 and in group II were 72.45±0.10 and 72.51±0.11 and were comparable to apparently healthy dogs. The results were furnished in Tables 4.11 and 4.12 and Fig. 4.19.

4.6.4.2 Lymphocytes (%)

The mean±SE of lymphocytes (%) recorded in group I and group II dogs before therapy were 22.88±0.24 and 22.85±0.14, respectively and showed non-significant difference (P>0.05) when compared to apparently healthy control dogs (22.9±0.23). However, after therapy, on day 15 and 30, the mean±SE of lymphocytes recorded in group I were 22.86±0.17 and 22.89±0.14 and in group II were 22.90±0.16 and 22.87±0.20 and were comparable to apparently healthy dogs in the study period. The results were furnished in Tables 4.11 and 4.12 and Fig. 4.20.

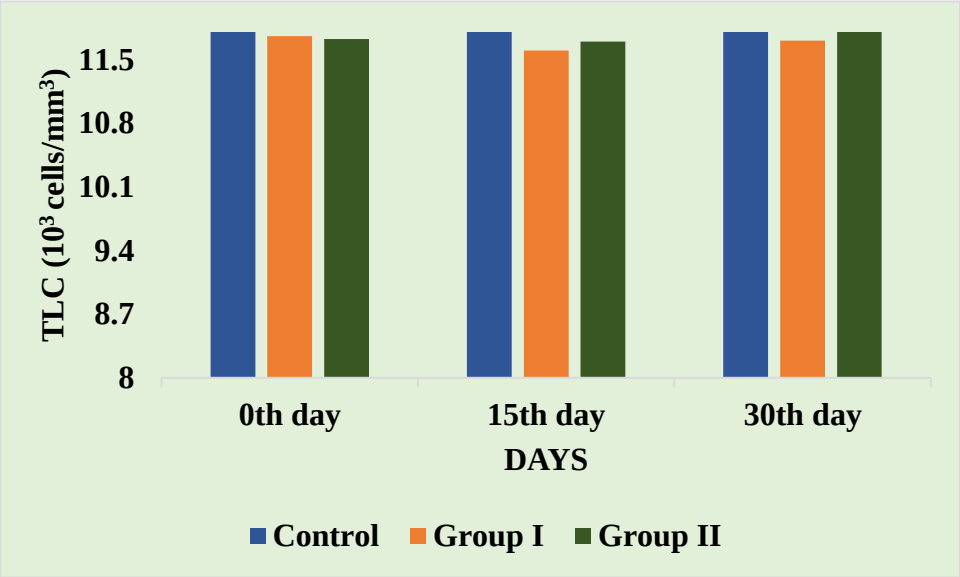

Fig. 4.18: Total Leukocyte Count (10^3 cells/mm^3) in different groups of OA affected Geriatric Dogs.

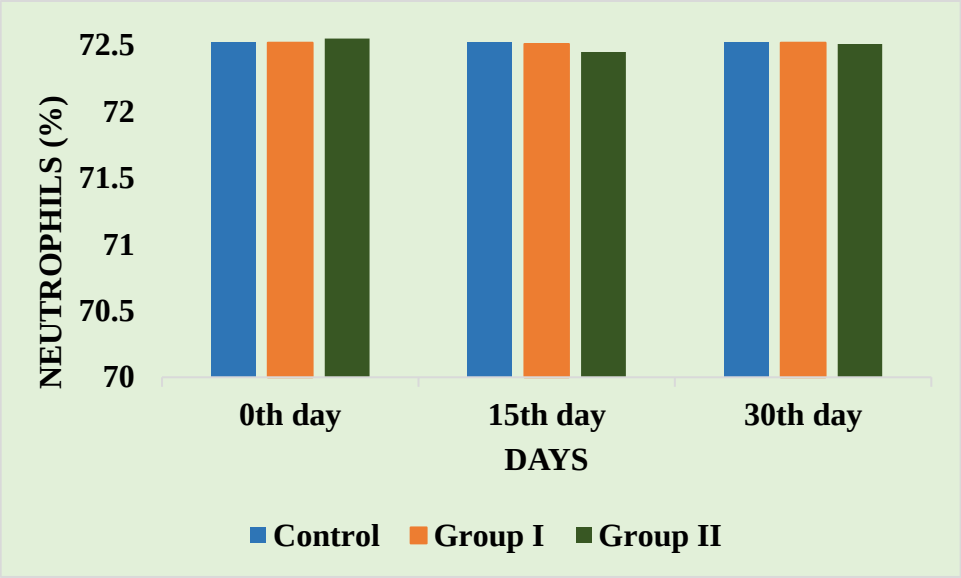

Fig. 4.19: Neutrophils (%) in different groups of OA affected Geriatric Dogs.

4.6.4.3 Eosinophils (%)

The mean±SE of eosinophils (%) recorded in group I and group II dogs before therapy were 2.3±0.14 and 2.29±0.06, respectively and were non-significant (P>0.05) when compared to apparently healthy control dogs (2.3±0.02). However, after therapy, on day 15 and 30, the mean±SE of eosinophils recorded in group I were 2.27±0.10 and 2.29±0.14 and in group II were 2.3±0.01 and 2.29±0.05 and were comparable to apparently healthy dogs in the study period. The results were furnished in Tables 4.11 and 4.12 and Fig. 4.21.

4.6.4.4. Monocytes (%)

The mean±SE of monocytes (%) recorded in group I and group II dogs before therapy were 2.25±0.02 and 2.24±0.04, respectively and showed non-significant difference (P>0.05) when compared to apparently healthy dogs (2.27±0.05). However, after therapy, on day 15 and 30, the mean±SE of lymphocytes recorded in group I were 2.26±0.05 and 2.26±0.02 and in group II were 2.25±0.03 and 2.28±0.01 and were comparable to apparently healthy dogs in the study period. The results were furnished in Tables 4.11 and 4.12 and Fig. 4.22.

4.7 Serum Biochemistry

4.7.1 Serum Creatinine (mg/dL)

The mean±SE of creatinine (mg/dL) recorded in group I and group II dogs before therapy were 0.82±0.09 and 0.79±0.09, respectively and showed non-significant difference (P>0.05) when compared to apparently healthy control dogs (0.82±0.13). However, after therapy, on day 15 and 30, the mean±SE of creatinine recorded in group I were 0.75±0.10 and 0.81±0.05 and in group II were 0.77±0.07 and 0.82±0.02 and were comparable to apparently healthy dogs in the study period. The results were furnished in Tables 4.13 and 4.14 and Fig. 4.23.

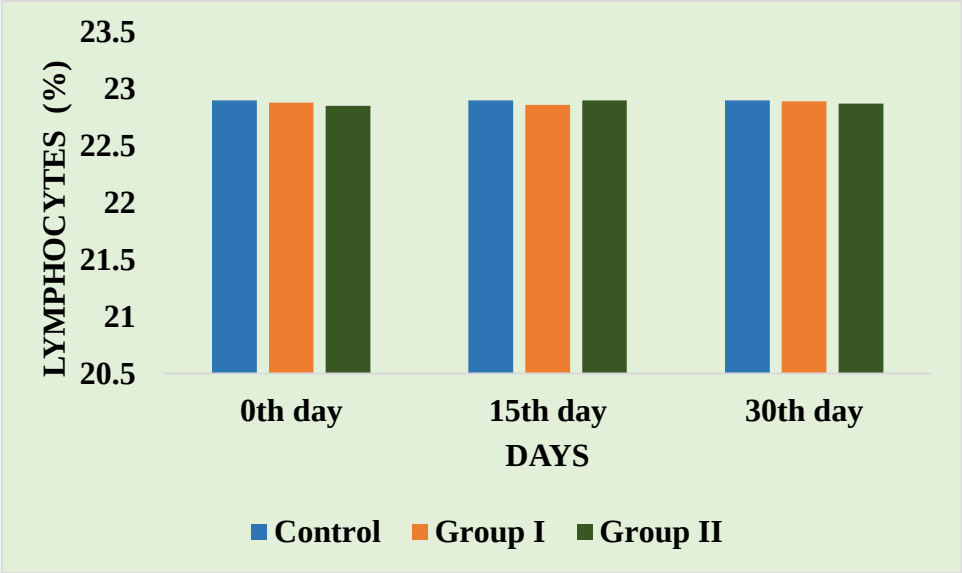

Fig. 4.20: Lymphocytes (%) in different groups of OA affected Geriatric Dogs.

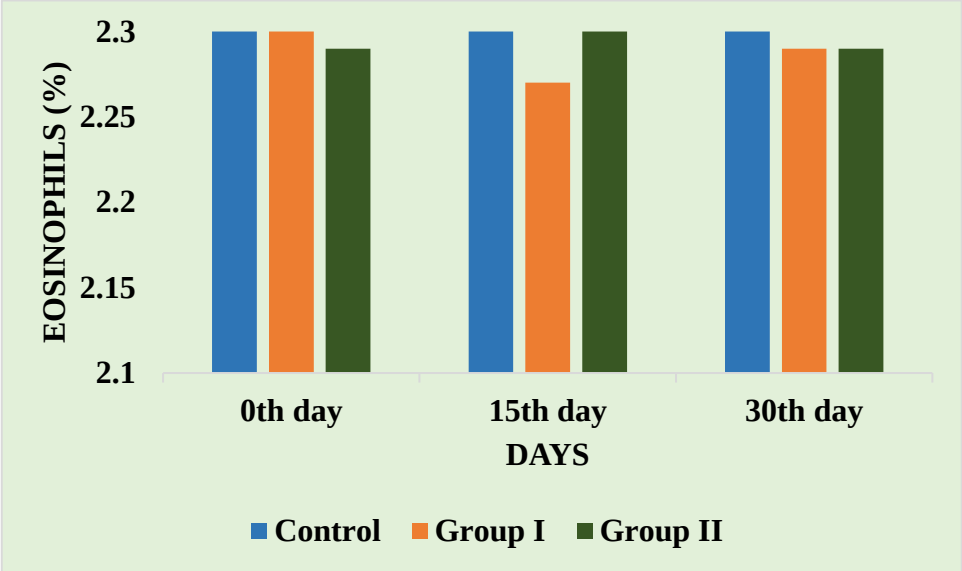

Fig. 4.21: Eosinophils (%) in different groups of OA affected Geriatric Dogs.

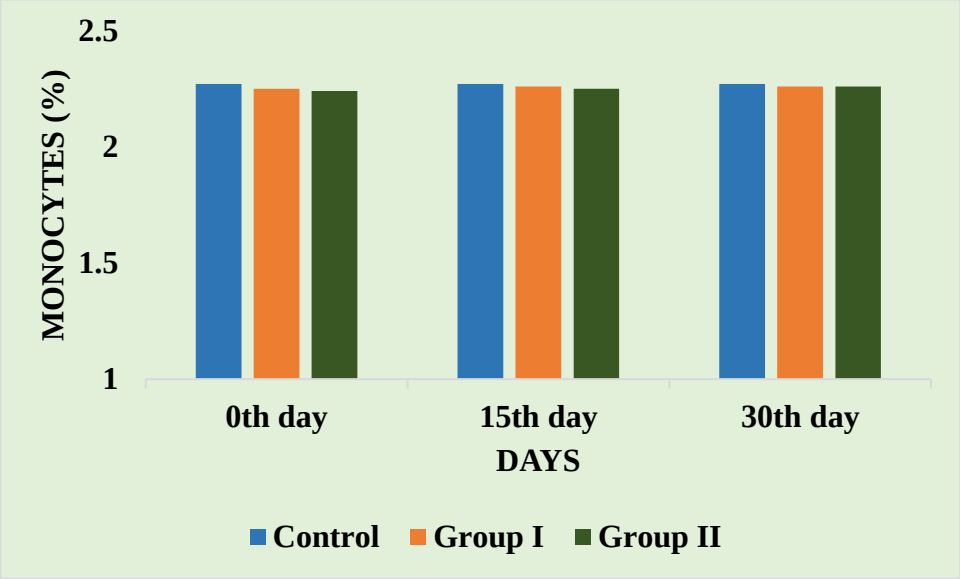

Fig. 4.22: Monocytes (%) in different groups of OA affected Geriatric Dogs.

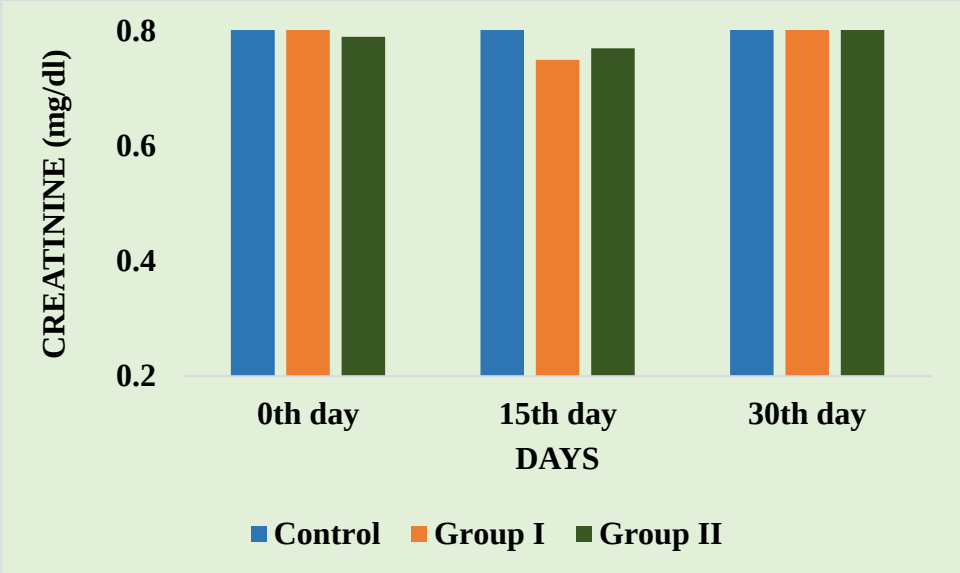

Fig. 4.23: Creatinine (mg/dl) in different groups of OA affected Geriatric Dogs.

Table 4.13: Serum Biochemical findings in Group I OA affected Geriatric Dogs

Sl. No	Parameter	Apparently healthy Dogs	Day 0	Day 15	Day 30
1	Creatinine (mg/dL)	0.82±0.13	0.82±0.09	0.75±0.10	0.81±0.05
2	ALT (IU/L)	45.89±1.11	45.69±0.75	45.55±0.66	45.81±0.60
3	AST (IU/L)	34.5±0.10	34.01±0.22	33.30±0.17	34.1±0.03
4	CRP (g/dL)	6.54±0.05	6.51±0.21	6.49±0.19	6.52±0.18

Note: Variations are non-significant

Table 4.14: Serum Biochemical parameters in Group II OA affected Geriatric Dogs

Sl. No	Parameter	Apparently healthy Dogs	Day 0	Day 15	Day 30
1	Creatinine (mg/dl)	0.82±0.13	0.79±0.09	0.77±0.07	0.82±0.02
2	ALT (IU/L)	45.89±1.11	45.71±0.60	44.61±0.40	45.85±0.40
3	AST (IU/L)	34.5±0.10	34.20±0.17	33.43±0.18	34.4±0.19
4	CRP (g/dL)	6.54±0.05	6.52±0.30	6.47±0.33	6.53±0.20

Note: Variations are non-significant

4.7.2 Serum alkaline aminotransferase (ALT) (IU/L)

The mean±SE of serum alkaline aminotransferase (IU/L) recorded in group I and group II dogs before therapy were 45.69±0.75 and 45.71±0.60, respectively and showed non-significant difference (P>0.05) when compared to apparently healthy control dogs (45.89±1.11). However, after therapy, on day 15 and 30, the mean±SE of serum alkaline aminotransferase recorded in group I were 45.55±0.66 and 45.81±0.60 and in group II were 44.61±0.40 and 45.85±0.40 and were comparable to apparently healthy dogs in the study period. The results were furnished in Tables 4.13 and 4.14 and Fig. 4.24.

4.7.3 Serum Aspartate Aminotransferase (AST) (IU/L)

The mean±SE of serum aspartate aminotransferase (IU/L) recorded in group I and group II dogs before therapy were 34.01±0.22 and 34.20±0.17, respectively and showed non-significant difference (P>0.05) when compared to apparently healthy control dogs (34.5±0.10). However, after therapy, on day 15 and 30, the mean±SE of serum alkaline aminotransferase recorded in group I were 33.30±0.17 and 34.10±0.03 and in group II were 33.43± 0.18 and 34.4±0.19 and were comparable to apparently healthy dogs in the study period. The results were furnished in Tables 4.13 and 4.14 and Fig 4.25.

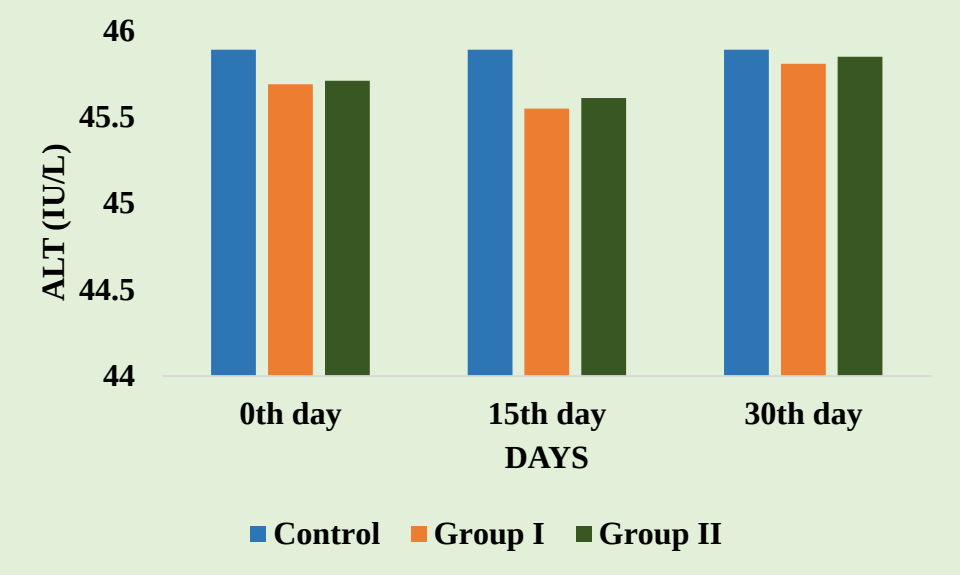

Fig. 4.24: Serum alkaline aminotransferase (IU/L) in different groups of OA affected Geriatric Dogs.

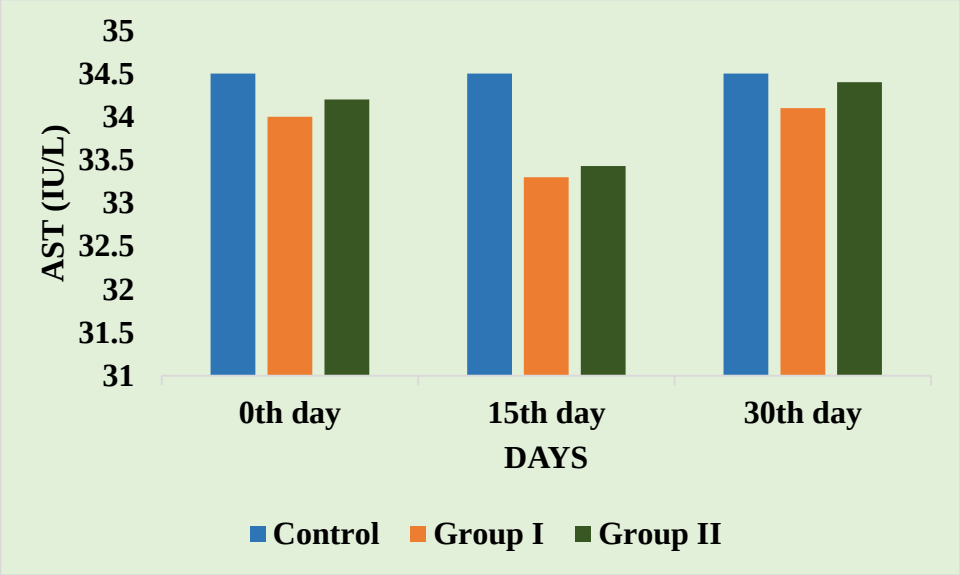

Fig 4.25: Serum aspartate aminotransferase (IU/L) in different groups of OA affected Geriatric Dogs.

4.7.4 C-reactive protein (g/dL)

The mean±SE CRP(g/dL) recorded in group I and group II dogs before therapy were 6.51±0.21 and 6.52±0.30, respectively and showed non-significant difference (P>0.05) when compared to apparently healthy control group (6.54±0.05). However, after therapy, on day 15 and 30, the mean±SE of CRP recorded in group I were 6.49±0.19 and 6.52±0.18 and in group II were 6.47± 0.33 and 6.53±0.20 and were comparable to apparently dogs in the study period. The results were furnished in Tables 4.13 and 4.14 and Fig. 4.26.

4.8 Synovial Fluid Analysis

Synovial fluid samples were collected from OA dogs in group I and group II and subjected for analysis.

4.8.1 Physical parameters

4.8.1.1 Volume (mL)

The mean volume of synovial fluid collected in group I and group II dogs before therapy were 0.20±0.01 and 0.20±0.02, respectively and found non-significant difference (P>0.05) when compared to the healthy control group (0.21±0.05). On day 15 and 30 of therapy, the mean volume collected in group I were 0.21±0.02 and 0.20±0.05 and in group II were 0.21±0.03 and 0.21±0.01. There was no significant difference (P>0.05) in the mean synovial fluid collected in both the groups. The results were furnished in Table 4.15 and 4.16 and Fig. 4.27.

4.8.1.2. Colour

The colour of the synovial fluid collected on 0^{th} day in group I dogs was noticed to be ranged colourless in (5), straw coloured in (3 cases) and red tinged in (2) dogs and in group II colourless in (4), straw coloured in (5) and red tinged in (1) dogs. On 15^{th} day, after therapy, the colour of synovial fluid in group I was noted as colourless in (6), straw coloured in (3) and red tinged in (1) dogs, whereas in group II colourless in (7) and straw coloured in (3) dogs. After 30 days of therapy, the colour of synovial fluid obtained was colourless in both the groups and was comparable with that of apparently healthy control dogs.

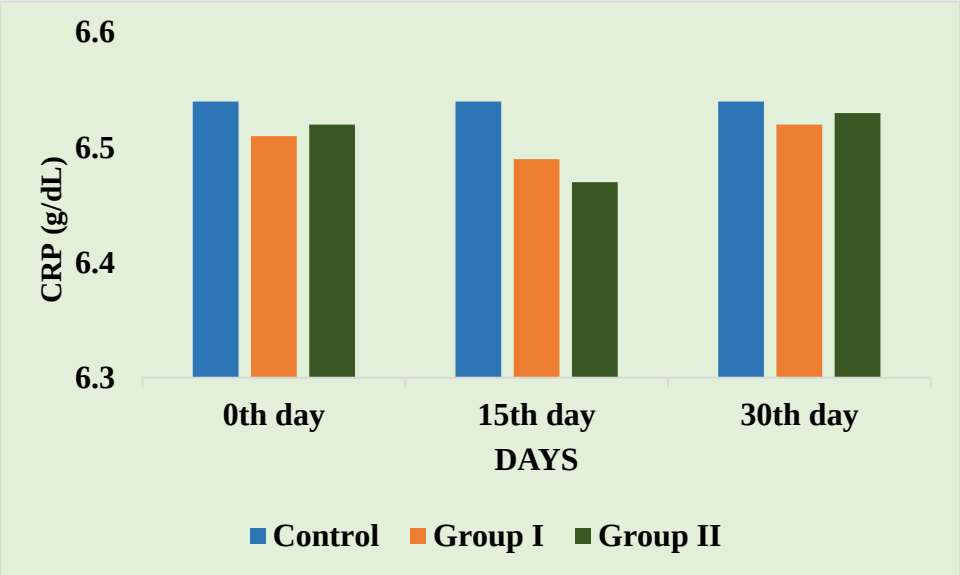

Fig. 4.26: C-reactive protein (g/dL) in different groups of OA affected Geriatric Dogs.

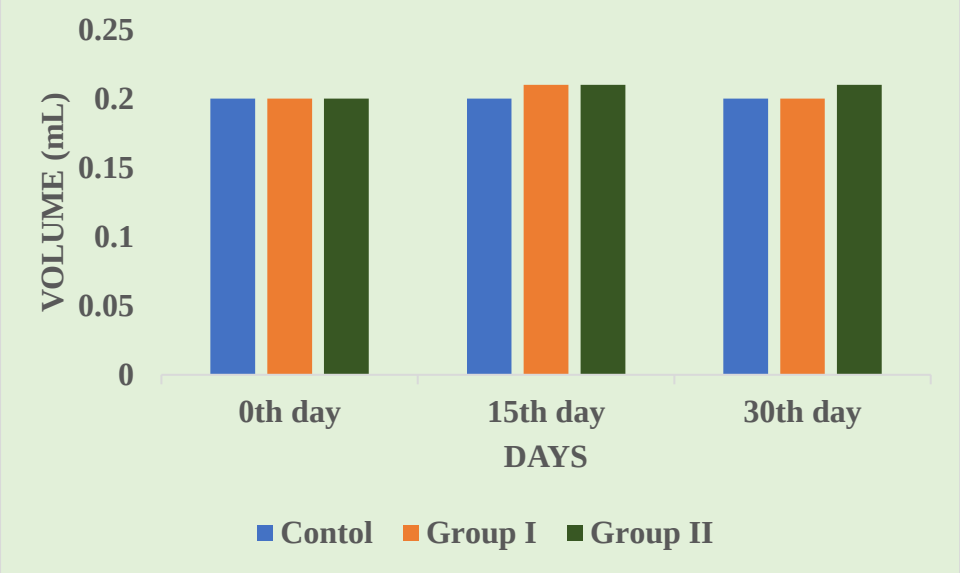

Fig. 4.27: Synovial fluid volume in different groups of OA affected Geriatric Dogs.

4.8.1.3 Viscosity

The viscosity of the synovial fluid sample on day 0 in group I dogs were reported as ++ in 2, +++ in 3, ++++ 5 dogs and in group II as ++ in 1, +++ in 5, ++++ 4 dogs. On 15th day of therapy, the viscosity in group I dogs were marked as ++ in 1, +++ in 1 dogs and ++++ in 8, whereas in group II as +++ in 2 and ++++ in 8. However, on 30th day of therapy, the viscosity in both the groups was ++++ 20. The results were furnished in Fig. 4.28-4.30.

4.8.1.4 Synovial fluid staining

Synovial fluid staining in the present study revealed the presence of synovial macrophages, neutrophils and monocytes under 100x power of magnification in microscope and were furnished in Fig. 4.31 and 4.32.

4.8.2 Synovial fluid biochemistry

4.8.2.1. Protein concentration (g/dL)

The mean of total protein in synovial fluid recorded in group I and group II osteoarthritis geriatric dogs on day 0 were 3.95±0.03 and 4.04±0.07, respectively and was significantly high ($P<0.01$) when compared to the healthy control group (2.4±0.20). On day 15 and 30, the mean concentration of total protein recorded in group I were 3.5±0.10 and 2.31±0.05 and in group II were 3.06±0.15 and 2.01±0.07. After therapy, values in both groups decreased significantly ($P<0.05$) when compared to day 0 and these decreased values were comparable apparently healthy dogs. The results were furnished in Table 4.15 and 4.16 and Fig. 4.33.

Fig. 4.28: Synovial fluid showing normal viscosity in Early OA dog

Fig. 4.29: Synovial Fluid showing decrease in viscosity in mild OA dog

Fig. 4.30: Synovial Fluid showing low viscosity in severely affected OA dog

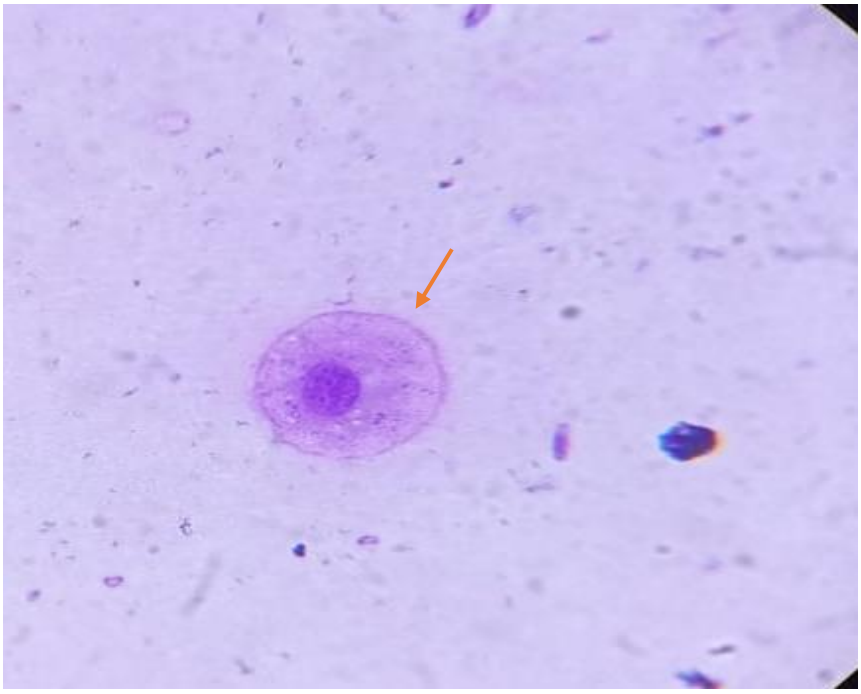

Fig. 4.31: Synovial fluid stained smear showing macrophage in OA affected dogs (100x).

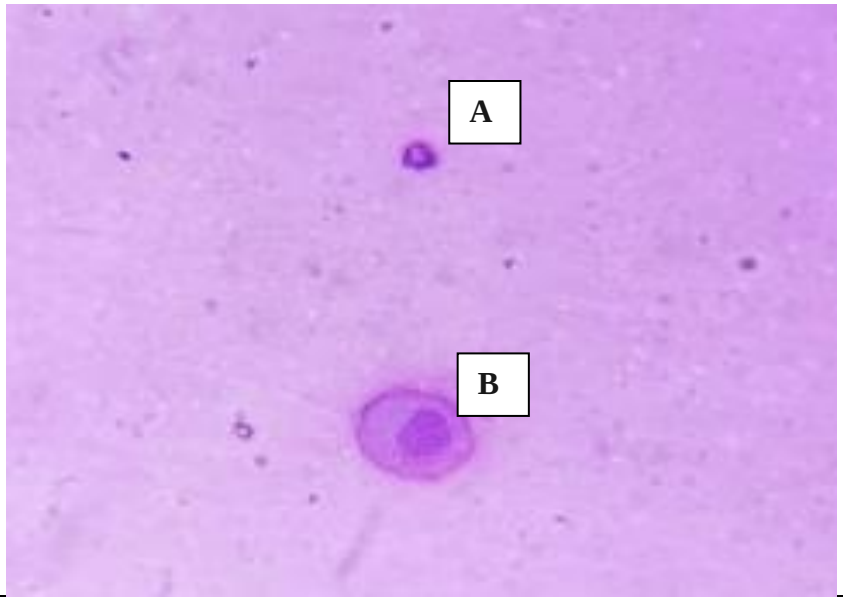

Fig. 4.32: Synovial fluid stained smear showing A)neutrophil and B) monocyte in OA dog (100x).

4.8.3 Cell study

4.8.3.1 Total nucleated cell count (cells/mL)

The mean values of total nucleated cell (cells/mL) count recorded in group I and group II osteoarthritis dogs before therapy were 2844.90±157.36 and 2866.12±150.96, respectively and found non-significant difference (P>0.05) when compared to the healthy control group (2897.12±150.14). On day 15 and 30 of the therapy, the findings in group I were 2889.81±84.63 and 2891.55±58.5 and in group II were 2866.4±41.43 and 2891.5±25.41. There was no significant difference (P>0.05) in after therapy values in both the groups. The results were furnished in Table 4.15 and 4.16 and Fig. 4.34.

4.8.3.2 Differential cell count

4.7.3.2.1. Mononuclear cells (%)

The mean mononuclear cell count (%) recorded in group I and group II osteoarthritic dogs before therapy were 83.6±1.57 and 83.24±1.10, respectively and were non-significant (P>0.05) when compared to the healthy control group (84.32±0.21). After 15 and 30 days of therapy, the values in group I were 83.7±1.74 and 83.97±1.32 and in group II were 84.2±1.95 and 84.3±0.06. After therapy, cell count in both the groups had no significant difference (P>0.05) when compared to apparently healthy dogs. The results were furnished in Table 4.15 and 4.16 and Fig. 4.35.

Table 4.15: Synovial fluid findings in Group I OA affected Geriatric Dogs

Sl. No	Parameter	Apparently healthy Dogs	Day 0	Day 15	Day 30
1	Volume (mL)	0.21±0.05	0.2±0.01	0.21±0.02	0.2±0.05
2	Total Protein (g/dL)	2.4±0.20	3.95±0.03*	3.5±0.10	2.31±0.05*
3	TNCC (cells/mL)	2897.12±150.14	2844.90±157.36	2889.81±84.63	2891.55±58.5
4	Mononuclear cells (%)	84.32±0.21	83.6±1.57	83.7±1.74	83.97±1.32
5	Neutrophils (%)	7.5±0.12	7.4±0.24	7.5±0.15	7.49±0.23

Note: Variations are non-significant except for Total Protein (**P<0.05 and * 0.01).

Table 4.16: Synovial fluid findings in Group II OA affected Geriatric Dogs

Sl. No	Parameter	Apparently healthy Dogs	Day 0	Day 15	Day 30
1	Volume (mL)	0.21±0.05	0.20±0.02	0.21±0.03	0.21±0.01
2	Total Protein (g/dL)	2.4±0.20	4.04±0.07*	3.06±0.15	2.01±0.07**
3	TNCC (cells/mL)	2897.12±150.14	2866.12±150.96	2866.4±41.43	2891.5±25.41
4	Mononuclear cells (%)	84.32±0.21	83.24±1.10	84.2±1.95	84.3±0.06
5	Neutrophils (%)	7.5±0.12	7.5±0.04	7.3±0.15	7.49±0.23

Note: Variations are non-significant except for Total Protein (**P<0.05 and * 0.01).

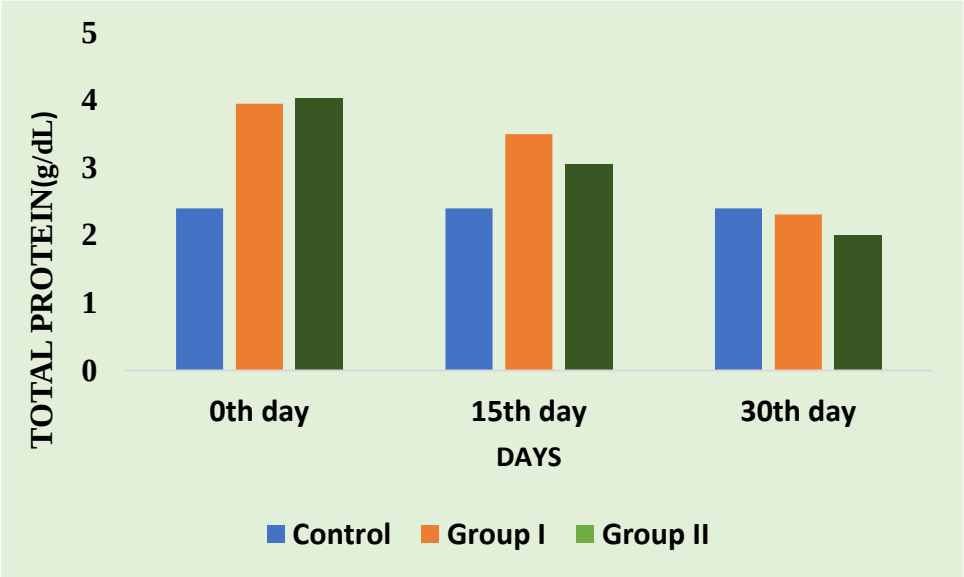

Fig. 4.33: Protein Concentration (g/dL) in different groups of OA affected Geriatric Dogs.

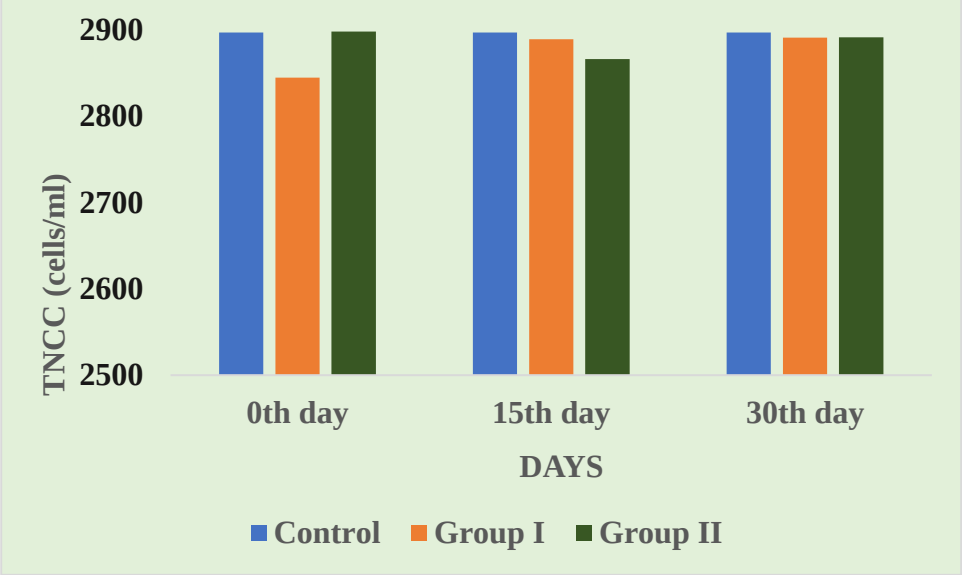

Fig. 4.34: Total nucleated cell count (cells/mL) in different groups of OA affected Geriatric Dogs.

4.8.3.2.2. Neutrophils

The mean neutrophil count (%) recorded in group I and group II osteoarthritic dogs before therapy were 7.4±0.24 and 7.5±0.04, respectively and there was non-significant difference (P>0.05) seen when compared to healthy control group, (7.5±0.12). On day 15 and 30, these findings in group I were 7.5±0.15 and 7.49±0.23 and in group II were 7.3±0.15 and 7.49±0.23, respectively. After therapy values in dogs of both the groups had no significance difference (P>0.05) when compared to apparently healthy dogs. The results were furnished in Table 4.15 and 4.16 and Fig. 4.36.

4.9 Radiographical evaluation

The affected joints were examined in the extended Ventro-dorsal view of the hip joints on day 0, 15 and 30.

On day 0, out of 20 dogs, 10 dogs showed reduction in the joint space, possible osteophyte formation at the joint margins and rough density at joint margins (Fig. 4.37 and 4.38) which indicates mild osteoarthritis. 5 dogs showed marked reduction in the joint space with clear evident of osteophyte formation and subchondral bone sclerosis at the joint margins (Fig. 4.39 and 4.40) which indicates advanced stage of osteoarthritis. 5 dogs do not show any radiographical evidence of osteoarthritis under radiographical examination.

4.10 Ultrasonographical evaluation

B-mode ultrasonography of the joints was performed with a linear probe of frequency 10-12 Hz and changes were noted. Ultrasonography of joints revealed the presence of joint effusions, loss of joint space, loss of muscle structure and tendinitis in dog affected with osteoarthritis (Fig 4.41-4.44). After therapy by day 30, decrease in tendinitis and myositis was seen in group I dogs more than group II dogs. (Fig. 4.45 and 4.46).

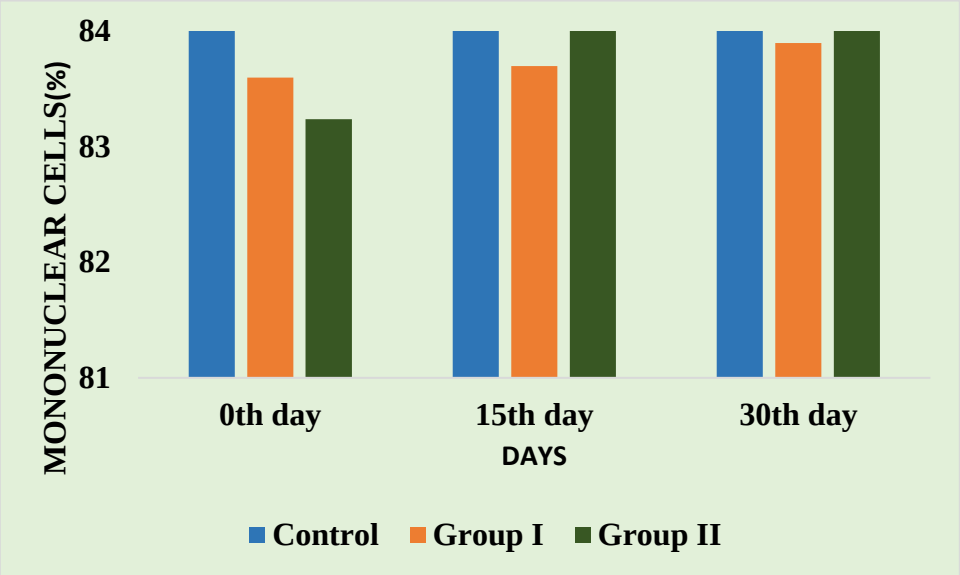

Fig. 4.35: Mononuclear cells (%) in different groups of OA affected Geriatric Dogs.

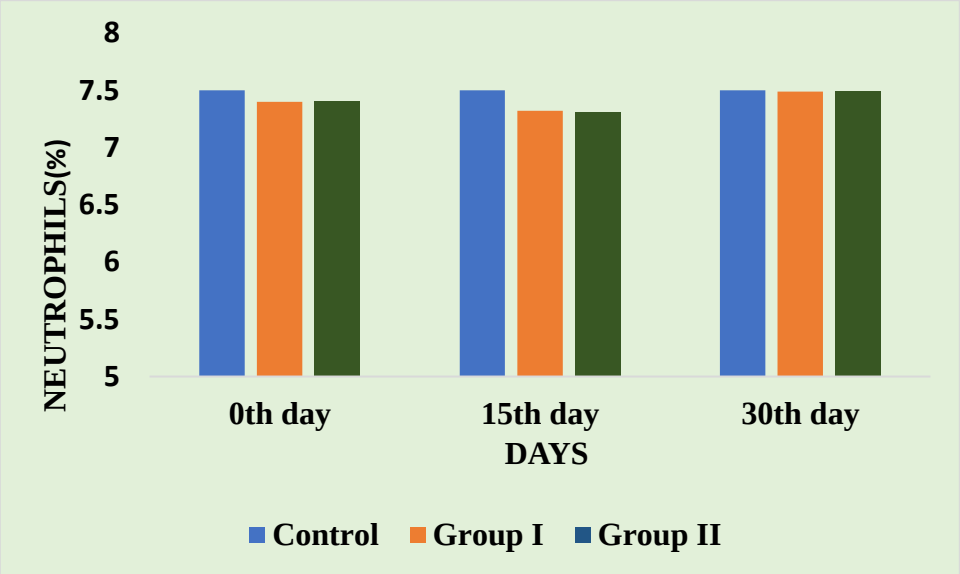

Fig 4.36: Neutrophils (%) in different groups OA affected Geriatric Dogs

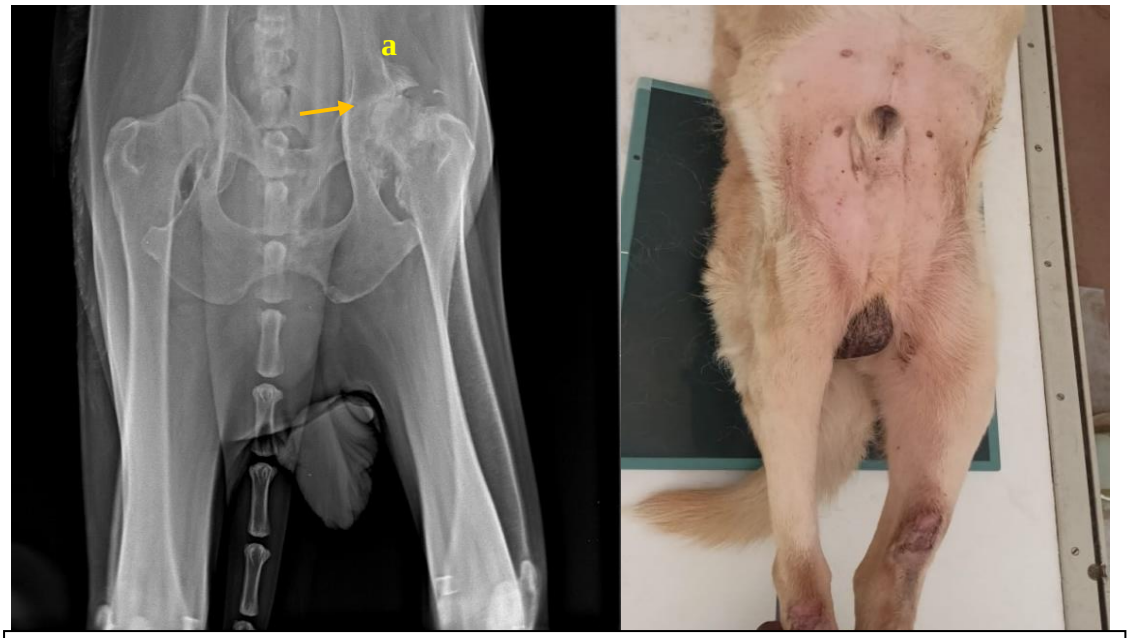

Fig. 4.37: Radiography of OA dog showing a) unilateral hip osteoarthritis, arrow indicating loss of joint space and degenerating joint.

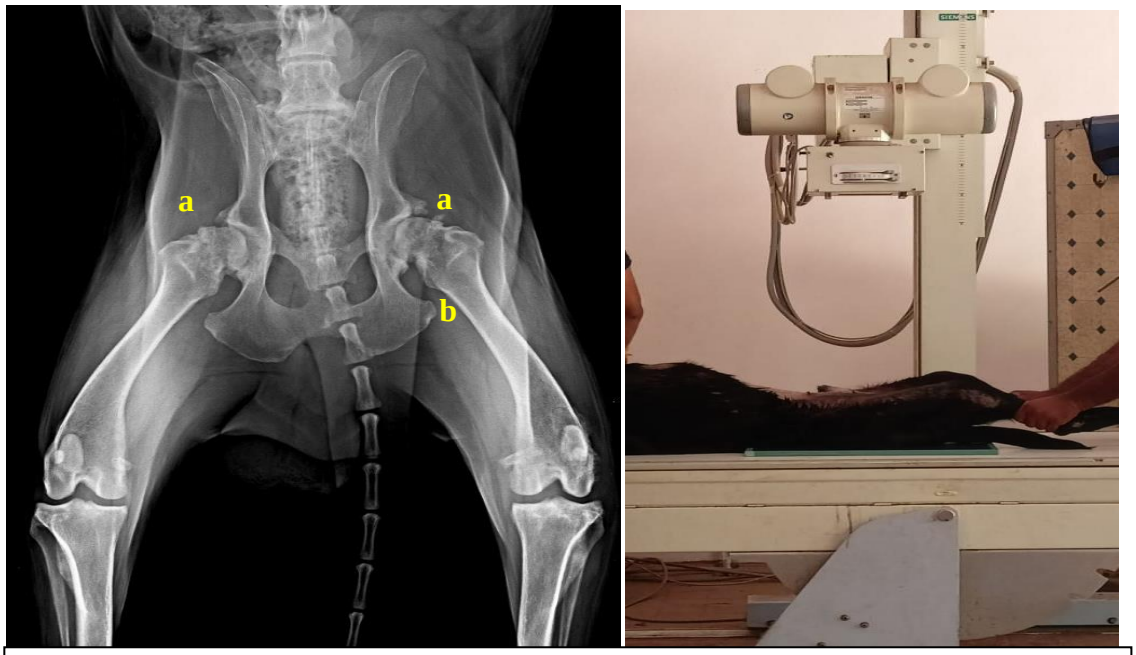

Fig. 4.38: Radiography of OA dog showing a) bilateral hip osteoarthritis, arrow indicating loss of joint space and loss of femoral head contour b) sclerosis of bone and white arrow indicating osteophyte formation. margin.

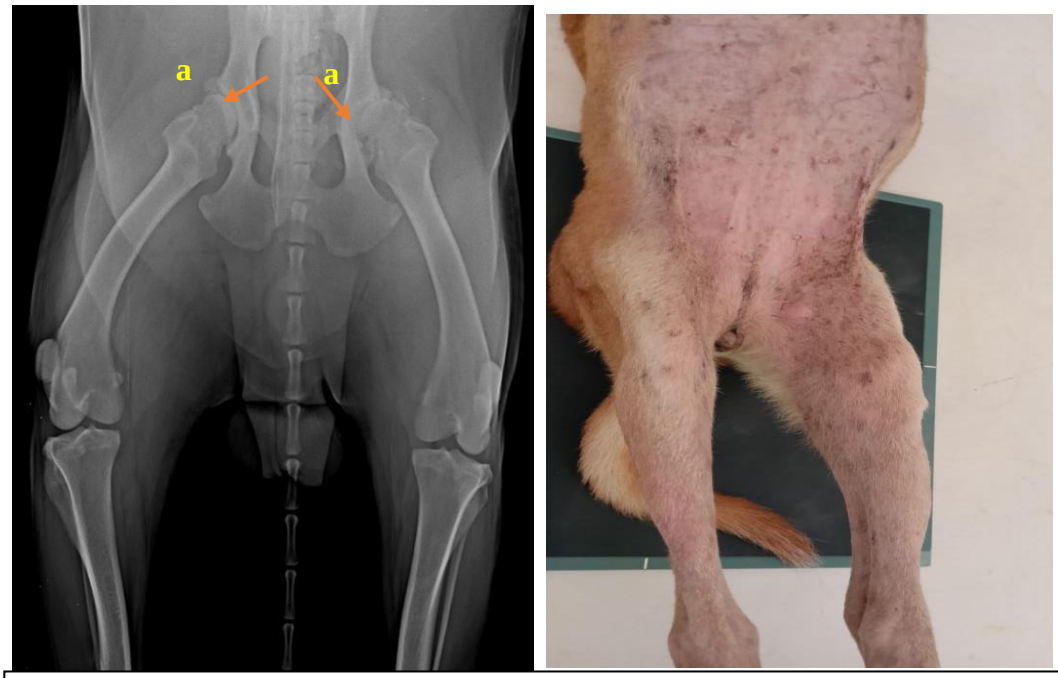

Fig. 4.39: Radiography of OA dog showing a) bilateral hip osteoarthritis, arrow indicating loss of joint space, loss of femoral head contour with complete distortion of joint.

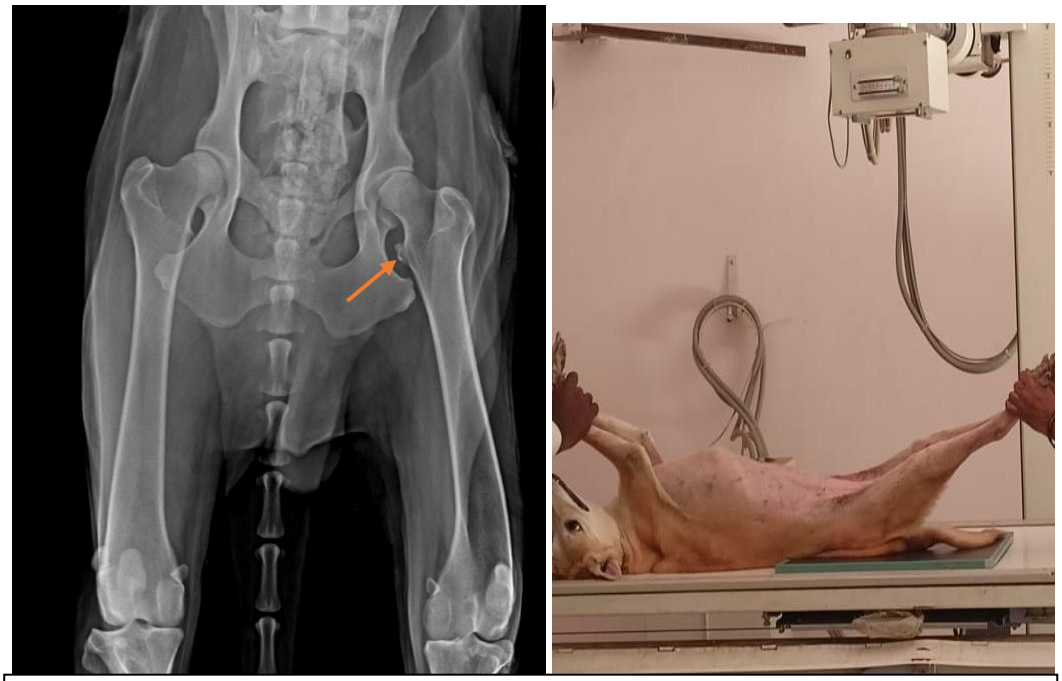

Fig. 4.40: Radiography of OA dog showing osteophyte formation at the joint margin, indication early stage of osteoarthritis.

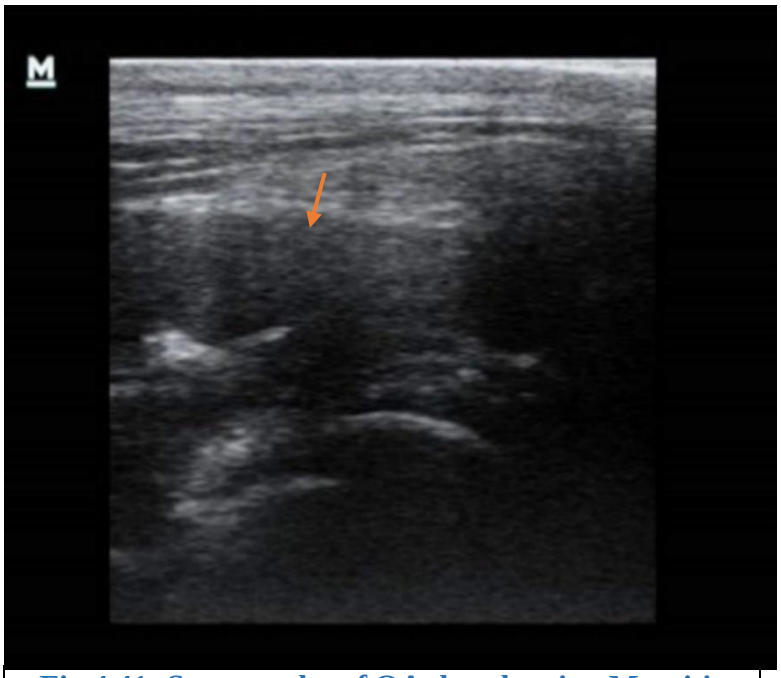

Fig 4.41: Sonography of OA dog showing Myositis

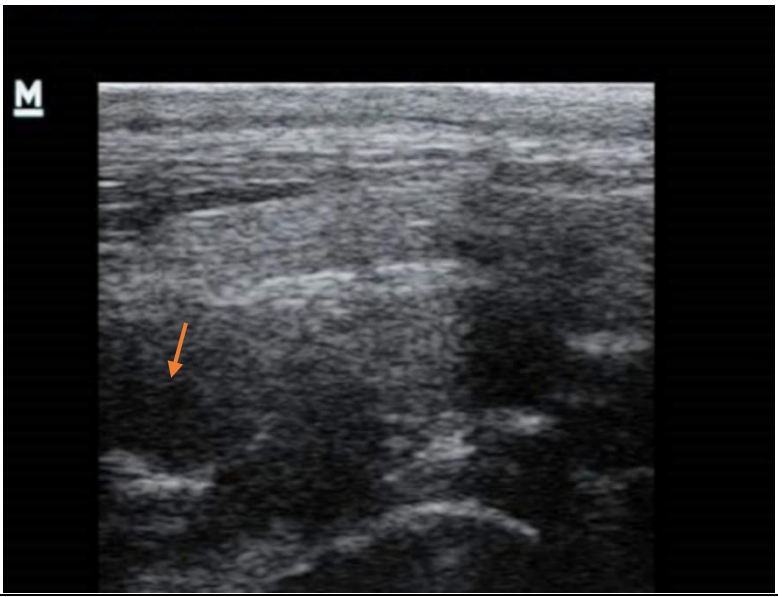

Fig. 4.42: Presence of tendinitis and severe myositis in OA affected dog

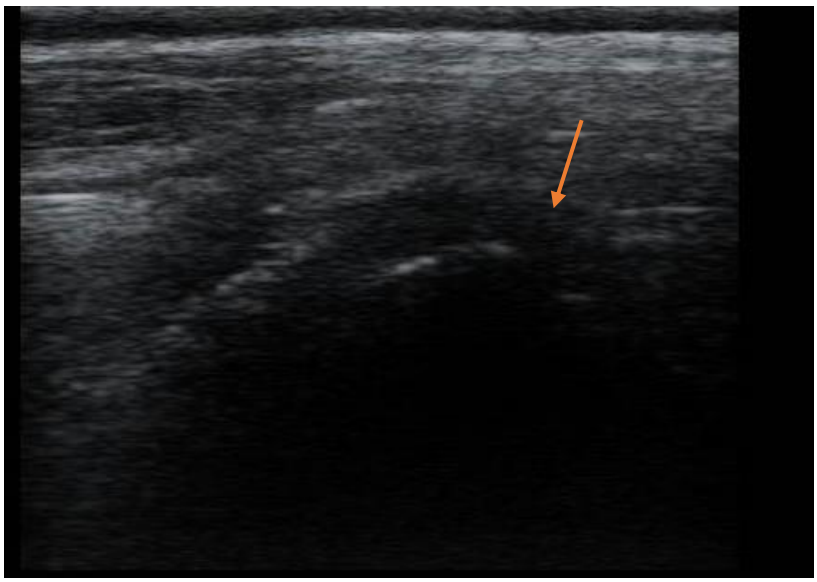

Fig 4.43: Presence of Joint effusions

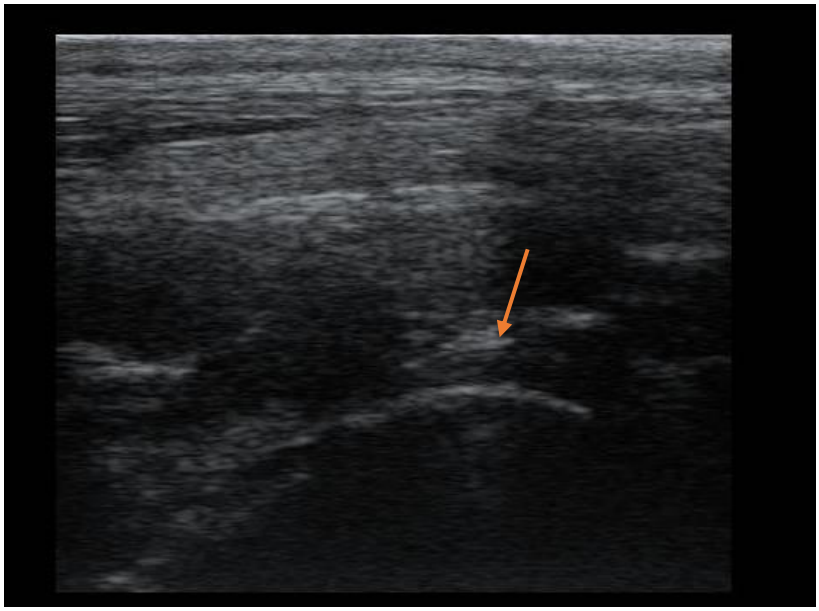

Fig 4.44: Presence of osteophyte on the head of femur

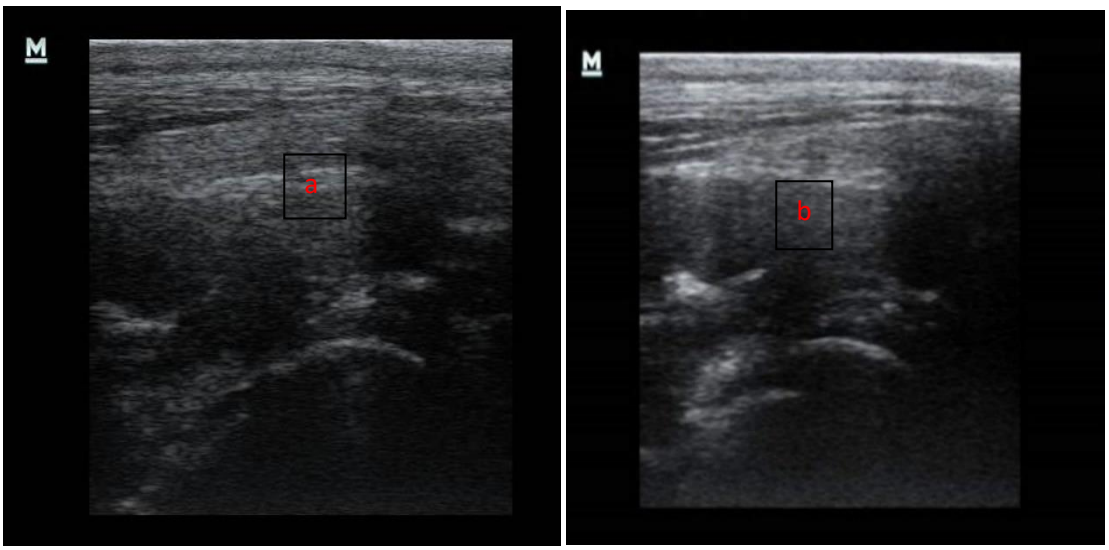

Fig. 4.45: Ultrasonography of OA affected dog in Group I showing a) myositis before therapy and b) after therapy, no complete loss of myositis is seen

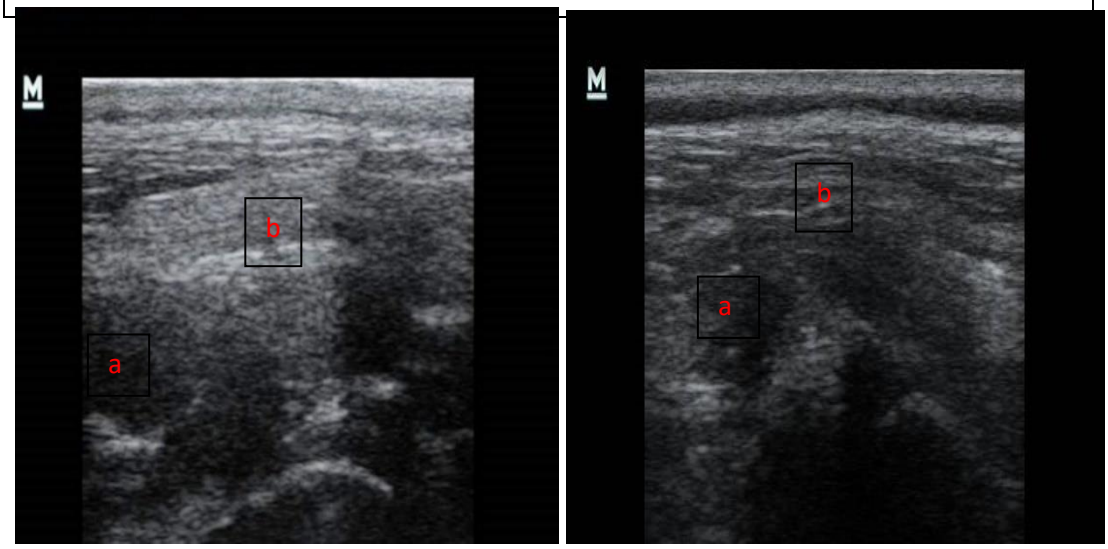

Fig. 4.46: Ultrasonography of OA affected dog in Group II showing a) myositis and tendinitis before therapy and b) after therapy, absence of tendinitis and appearance of muscle striations, indicating loss of myositis is seen

4.11 CT scanning

Out of 20 dogs receiving different therapeutic regimens, only 2 possible cases were further proceeded for CT scanning, which revealed the presence of hypertrophied femoral head and visible osteophytes at the joint margins (Fig. 4.47 and 4.48).

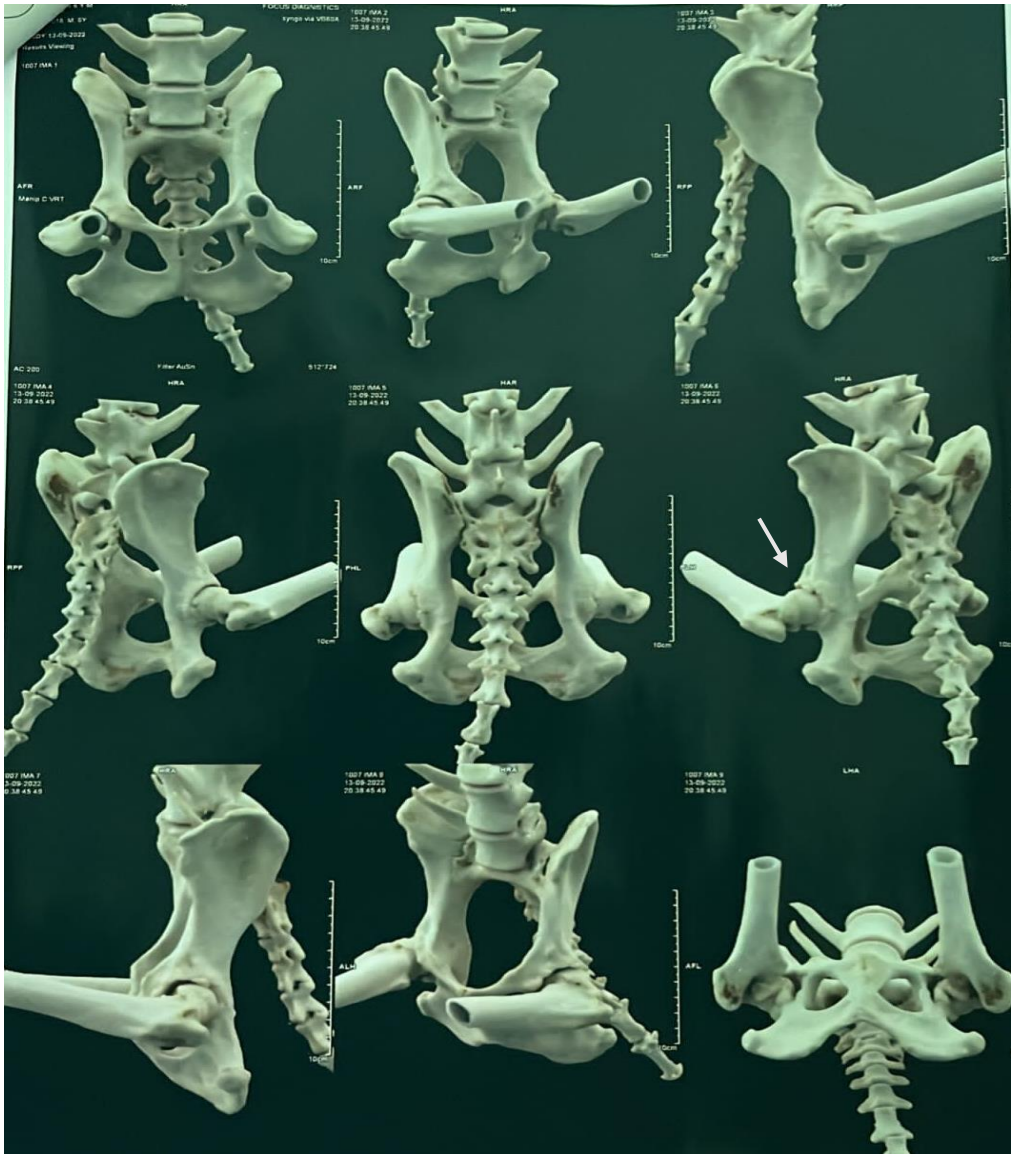

Fig. 4.47: CT in osteoarthritis dog showing normal joint formation and arrow indicating degenerating joint.

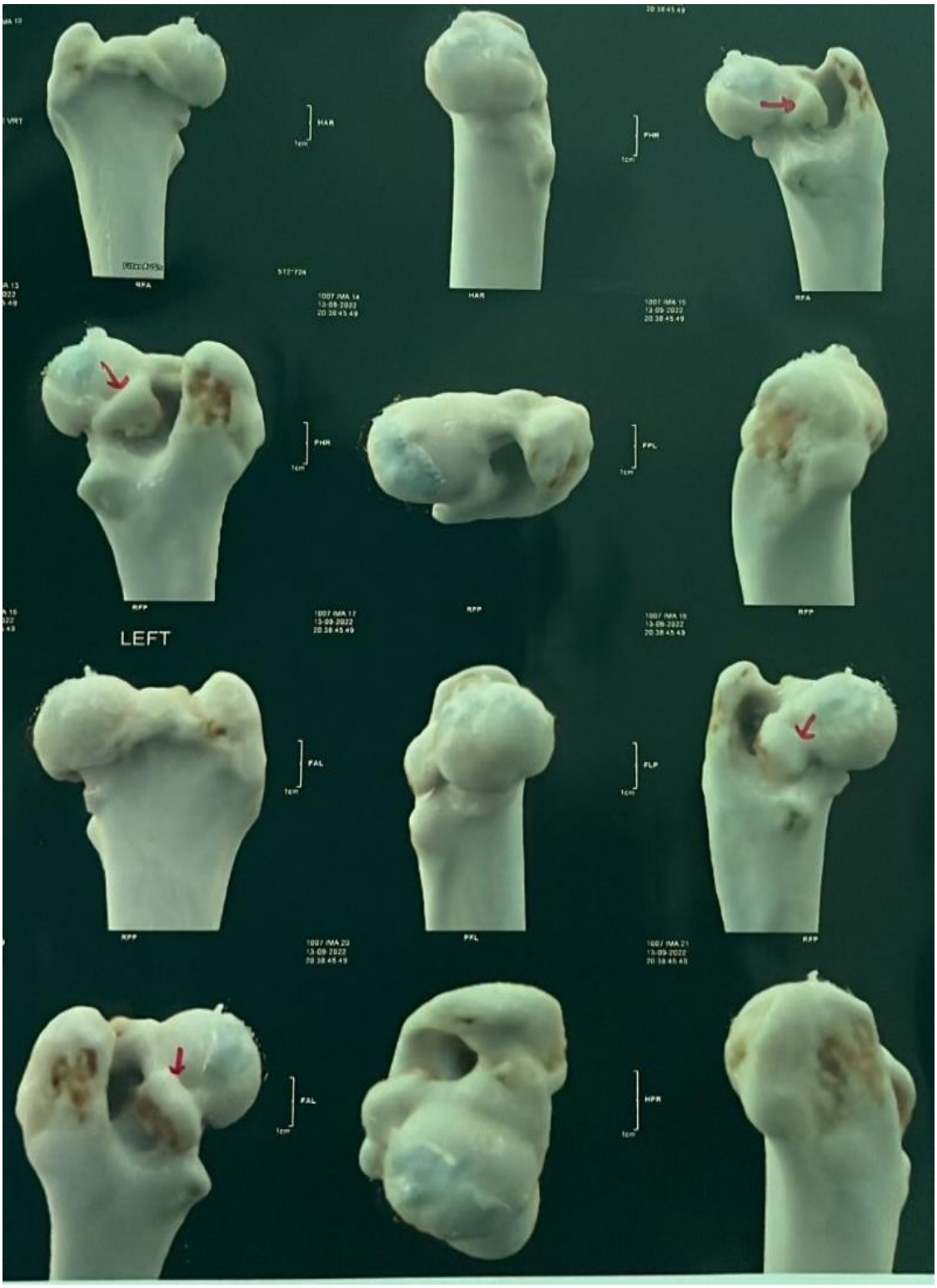

Fig. 4.48. CT of OA dog showing hypertrophied femoral head and osteophytes at femoral neck (bilateral)

4.12 Therapeutic efficacy

In the present study, 20 positive cases of osteoarthritis were randomly divided into 2 groups with 10 in each. Dogs belonging to group 1 were treated with carprofen @2.2-4.4 mg/kg body weight for 5 days, application of diathermy @ twice a day (Fig. 4.49-4.50) maintenance with lubrihans@ 1tab/ 10kg for 30 days whereas group II dogs received Carprofen @2.2-4.4 mg/kg body weight for 5 days, application of TENS @ twice a week (Fig. 4.51 and 4.52) for 4 weeks and maintained with Ashwagandha @500-1000 mg/kg for 30 days. Out of 10 dogs in group I, 5 dogs (50%) showed improvement in all the parameters by day 30, where as in group II, out of 10 dogs, 8 (80%) dogs showed improvement in all the parameters by day 30. The results were furnished in Fig. 4.59.

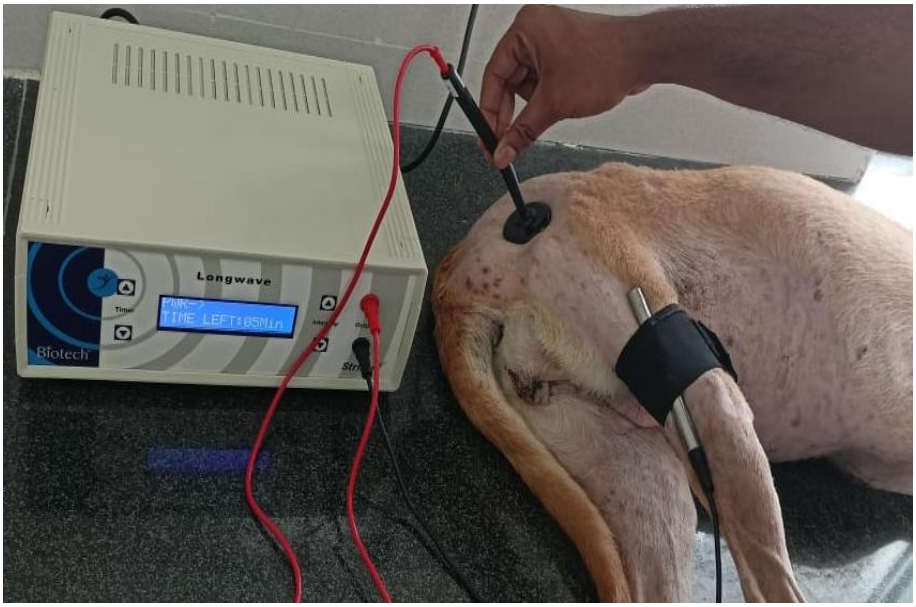

Fig. 4.49: Application of Diathermy in OA affected Geriatric Dog

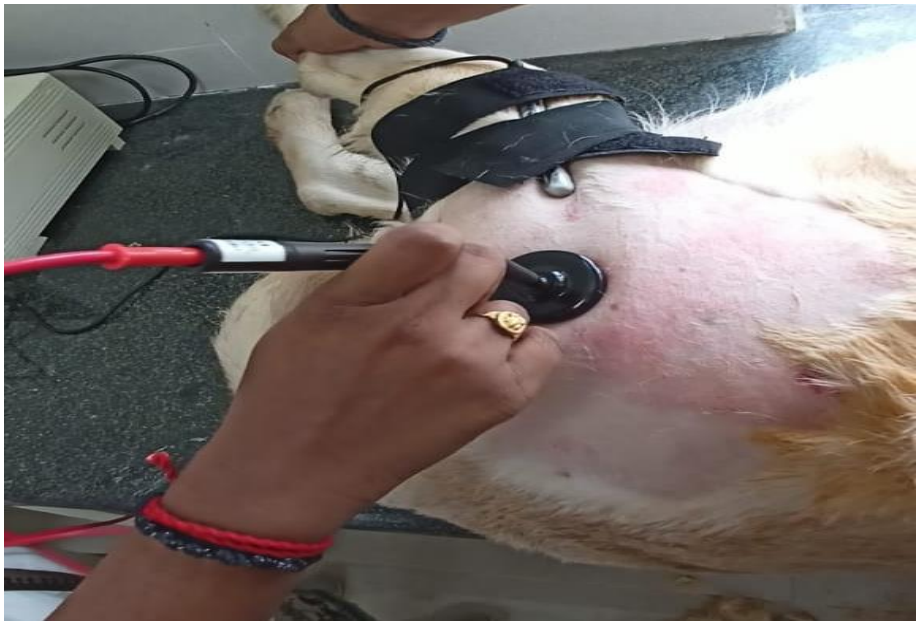

Fig. 4.50: Application of deep heat at the hip joint in OA affected Geriatric dog

Fig. 4.51: Application of TENS in mild OA affected Geriatric dog

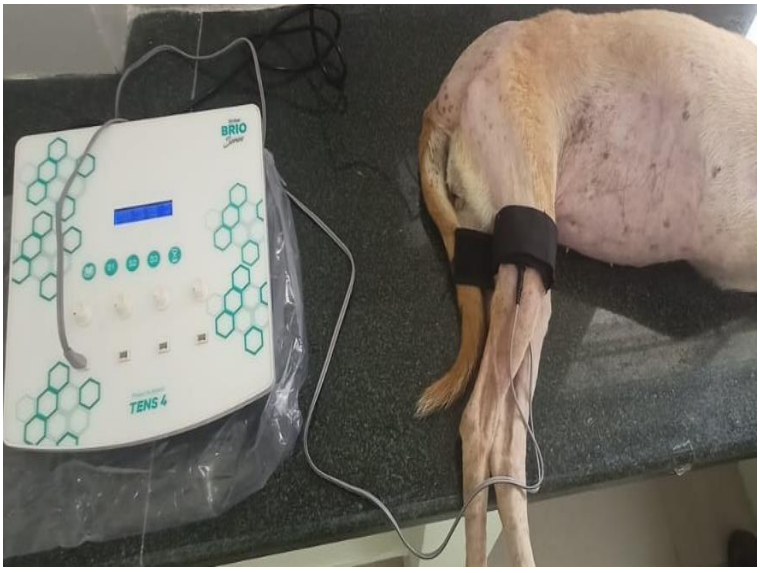

Fig. 4.52: Application of TENS in a severely affected OA dog at the level of lower pelvis and affected hip joint muscle

Fig 4.53: Dog showing improvement by day 30 in terms of weight bearing and lameness.

Fig. 4.54: Dog showing improvement by day 30 after therapy in interms of able to bear weight on hindlimbs.

Fig. 4.55: Dog showing improvement by day 30 after therapy in interms of gait improvement

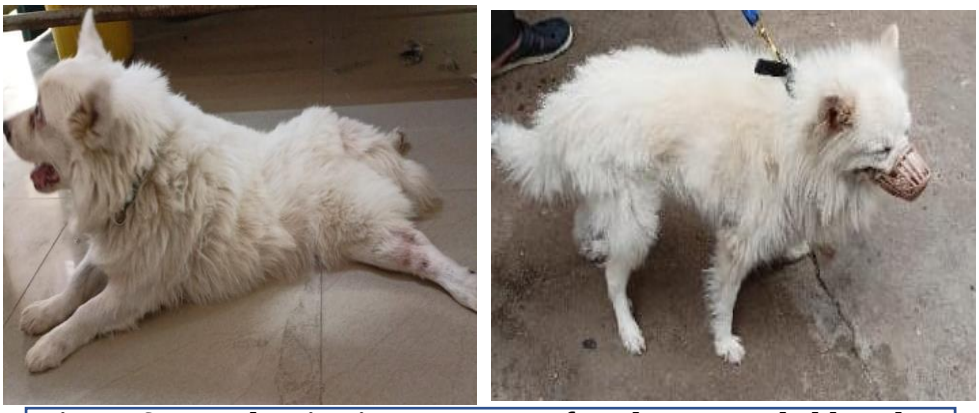

Fig. 4.56: Dog showing improvement after therapy and able to bear weight on its hind limbs

Fig. 4.57: Dog showing drastic reducion in body weight in after therapy and able to rise and walk on his hindlimbs.

Fig. 4.58: Dog showing improvement in weight reduction and able to bear weight and walk normally after 30 days of therapy.

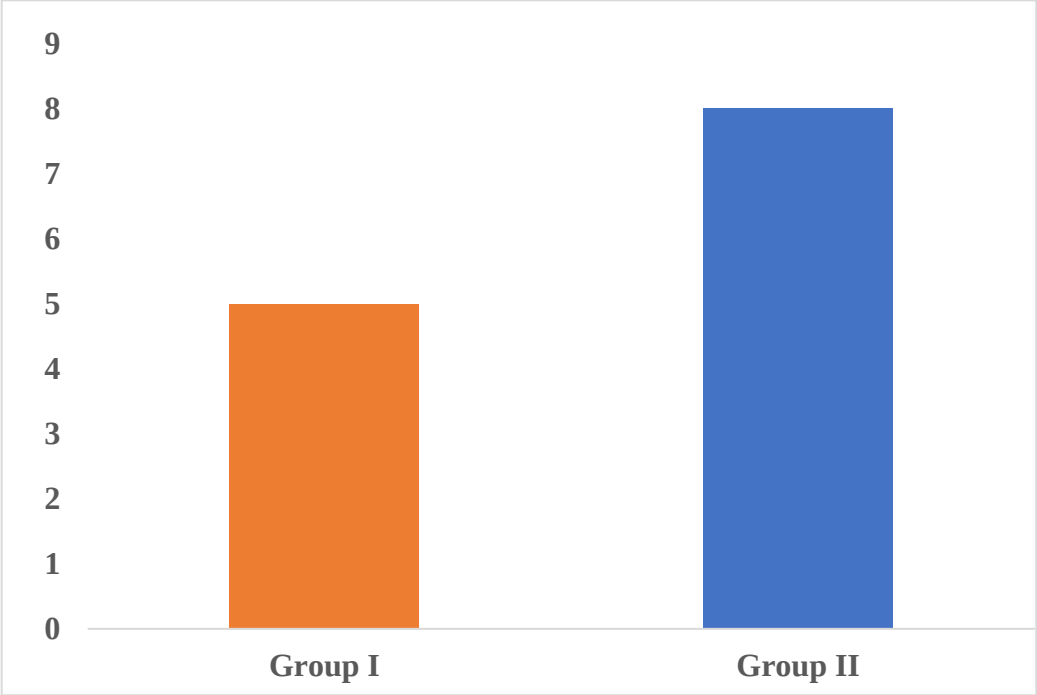

Fig. 4.59: Therapeutic efficacy in group I and group II dogs.

CHAPTER V

DISCUSSION

In the present investigation, a total of 3040 geriatric dogs were bought to Veterinary Clinical Complex (VCC), Campus Veterinary Hospital, College of Veterinary Science, Rajendranagar; Veterinary Hospital Bhoiguda and also dogs from the peripheral hospitals presented with the signs of lameness, inability to bear weight and exercise intolerance were selected and subjected to various diagnostic techniques like clinical examination, radiography, ultrasonography, CT scanning and were further estimated for complete blood analysis, serum biochemistry and synovial fluid analysis for confirmatory diagnosis of osteoarthritis (OA). Among the positive cases, 20 dogs were randomly selected and divided into two groups as group I and II and given two different therapeutic regimens. However, 10 apparently healthy geriatric dogs were taken as control.

In the current study, among 3040 geriatric dogs, 350 were diagnosed positive for OA with the overall incidence of 11.51%. These findings are nearer to the reports by Prasad (2012) and Triakoso (2013) with the incidence rate of 9% and 7.2%, respectively. However low incidence of 1.28% was reported by Jain *et al.* (2015) and higher incidence was recorded in studies conducted by Millis *et al.* (2014), Lamani *et al.* (2019) and Anderson *et al.* (2020) with the incidence of 68%, 32% and 20%, respectively. These variations in the incidence rates compared to present findings could be due change in the total number of animals screened and selection criteria.

In the present study, with respect to age wise incidence of OA, dogs aged between 10-15 years (62.86%) were more prone and is in consistent with the findings of DeGroot *et al.* (2004), Smith *et al.* (2006), Elliot *et al.* (2007), Mele (2007), Bland (2015), Lamani *et al.* (2019), Wright *et al.* (2019) and Anderson *et al.* (2020). However, studies conducted by Prasad *et al.* (2012) reported the high incidence in >4 years (82%) aged dogs. Higher incidence of OA during old age could be due to accumulation of Advanced Glycation end (AGE's) products according to age progression which has been shown to increase tissue stiffness, decrease synthesis and increase the degradation of the extracellular matrix, as well as affect the cellular process within the joint and may predispose to the development of OA in aged dogs (DeGroot

et al., 2004). At geriatric age, large active muscle mass may inhibit the transformation of passive laxity into the functional laxity as the dog make attempts to move and which decreases the stresses on the articular cartilage that leads to degenerative changes in the joints (Smith *et al*., 2006).

In the present study, breed wise incidence of OA was reported to be higher in Labrador Retriever (45.71%) and is in agreement with the findings of Elliott *et al.* (2007), Mele (2007), Anderson *et al.* (2018), Lamani *et al.* (2019), Anderson *et al.* (2020), Johnson *et al.* (2020) and O'Neil *et al.* (2020). The higher incidence in Labrador Retrievers might be due to the high body weights they possess and the inherent defects of this breed (Anderson *et al.,* 2018) and high body condition scores they possess (Johnson *et al.*, 2020). In contrary to this, some of the studies reported the higher incidence in German Shepherd breed (Bland, 2015 and Prasad *et al.,* 2012). The variations in these findings could be due to difference in the topographical distribution of breeds.

In the present study, with respect to gender wise incidence of OA, 64% of male dogs were affected and is in accordance with the findings of Fernandes *et al.* (2002); Smith *et al.* (2006); Mele (2007); Prasad *et al.* (2012); Jain *et al.* (2015) and Lamani *et al.* (2019). The higher incidence of OA in male dogs might be due to the sex hormones interacting with loci that contribute to the pathogenesis of osteoarthritis or that hormones impact transcriptional regulation and leads to local tissue expression, leading to OA (Hays *et al.,* 2007). In contrast to the present findings, some of the earlier studies reported the higher incidence of OA in females than males (Meeson *et al.,* 2019 and Whitehair *et al.,* 1993). However, Anderson *et al.* (2020) and Henrotin *et al.* (2005) reported that sex would not be a significant risk factor for occurrence of osteoarthritis in dogs.

On perusal of Table 4.4, joint wise incidence of OA in geriatric dogs, hip joint (82.57%) is the most commonly involved and is in accordance with the reports of Peach *et al.* (2005), Ginja *et al.* (2009), Sanghi *et al.* (2009) and Stabile *et al.* (2019). The higher incidence of hip osteoarthritis might be due to the fact that hips carry most of the body weight throughout the life, which is very hard on the articular cartilage and therefore as the age advances, the degeneration process may take place quickly, resulting in osteoarthritis (Lamani *et al.,* 2019).

In the present investigation, among the risk factor involved in occurrence of OA in geriatric dogs overweight (57.71%) found to be major factor, followed by neutering (12.86%), slippery floor (9.43%), over use of calcium (8.57%), heavy exercise (5.71%), underlying joint diseases (4.29%) and Hypothyroidism (1.42%).

Similar observation of overweight as major risk factor for OA was made by Johnson *et al.* (2020) with 55.8%. The present results are also in near accordance with findings of McLaughlin and Roush (2002), Lund *et al.* (2006), Marshall *et al.* (2009), Runge *et al.* (2010), Sanderson *et al.* (2012) and Anderson *et al.* (2020). Metabolic and systemic consequences of obesity may play a significant role in the aetiology of OA wherein fat produces systemic inflammatory factors like cytokines and adipokines, which were distinct adipose tissue-produced components with considerable inflammatory qualities (Mosley *et al.*, 2022). Further in obese cases increased body weight leads to increased pressure on the weight bearing joints and predisposes for the development of OA (Anderson *et al.*, 2020) and obesity primarily causes an increase in mechanical loading, which in turn causes mechanical stress, more wear and tear and ultimately cartilage degeneration and results in OA (Loef *et al.,* 2019).

In the present study, neutering is reported as the second most risk factor associated with OA in dogs and is in accordance with findings of German (2006), Meeson *et al.* (2019), Preet *et al.* (2021), O'Neill *et al.* (2020) and Anderson *et al.* (2020). Neutering predisposes dogs for the development of osteoarthritis due to the imbalanced gonadal hormones affecting the growth rate and development of the bones (Anderson *et al.*, 2020) and in the absence of gonadal hormones, an abnormal growth plate closure may increase the likelihood of a clinically obvious joint problem as the age advances in dogs (Riva *et al*., 2013). The changes that occur in sex hormones after neutering, thought to cause behavioural changes, most notably increased food seeking and decreased physical activity leading to OA (Bermingham *et al.*, 2014 and Raffan *et al.*, 2015).

Heavy exercise is one of the risk factors for the development of OA in dogs and the present findings are in accordance with the studies conducted by Mele (2007), Bland *et al.* (2015) and Anderson *et al.* (2020). Exercise particularly at young age may leads to OA which might be due to over use and damage to the developing joints (Anderson *et al.*, 2020).

In the present study, slippery floor is reported as one of the risk factors associated with OA in dogs and is in accordance with the studies of Alsaleem et al. (2013), Witte *et al.* (2019), Capon (2021) and Goldberg (2022). Dogs raised on slippery floor may develop osteoarthritis as the age advances, which might be due to the fact that as dogs moves on slippery floor, some of the back muscles and joints needs to stabilize themselves on such floors and movement will be restricted in some parts of the dogs body, while other parts will have to work harder than normal to compensate. Over time, this inefficient movement can become habitual and the back muscles and joints may develop osteoarthritis as age advances.

Further, over use of calcium during young age is one of the risk factors for the development of OA in geriatric dogs and similar observations are reported by Richard and Toll (1997), Raditic and Athens (2019) and Lauten (2006). Excess use of calcium during the young age causes suppression of the parathyroid gland and this excess calcium may leaches out from body if feed continuously and makes bone thin and weak leading to hip dysplasia and inturn leads to OA as the age advances.

In the present study, underlying joint diseases like hip dysplasia accounted for 4.29% in occurrence of OA in dogs, which is in accordance with the findings of Sandell (2012), Alsaleem (2013), Ramirez-Flores *et al.* (2017) and Meeson *et al*. (2019). This might be due to fact that hip dysplasia leads to loss of normal joint conformation in younger stages of life and as age advances the trauma to hip joint increases and leads to OA in geriatric age.

In the present study, Hypothyroidism accounted for 1.43% in occurrence of OA in dogs. This finding is in consistent with the reports of German (2006) and Kutzler (2020) and slightly lower (2.8%) percent reported by Juge *et al.* (2017). This observation could be substantiated that hypothyroidism leads to accumulation of certain proteins in the body especially at the joints resulting in joint and surrounding muscle damage and as the age advances, leads to degenerative changes. However, according to Kutzler (2020), OA in hypothyroid dog might be because of effect of gonadectomy on the joints.

In the present study, among the clinical signs recorded, pain is most common condition exhibited by all the osteoarthritic affected joints, which is in agreement with the findings of

Henrotin *et al.* (2005), Beale (2004); Goldring and Goldring (2006), Lindey and Taylor (2010), Ranganath (2012), Meeson *et al.* (2019) and Lawrence *et al.* (2022).

In the present study, with respect to physical parameters, there is no significant rise in the body temperature, heart and respiratory rates in both the groups I and II before and after therapy when compared to apparently healthy dogs throughout the study period. These findings were matching with the findings of Suheb *et al.* (2022) and Alam *et al.* (2006), with the observation of no significant changes in physiological parameters like temperature, heart and respiratory rates in a dog suffering with osteoarthritis and the values reported by them were within the reference range. Osteoarthritis affects only the localised portions of the damaged joint and has no systemic reactions, hence there are no significant changes in physiological parameters.

In the current study, among the clinical parameters, there is significant improvement in weight bearing in group I and group II dogs after therapy. Slight improvement is seen by day 15 in both group I and group II dogs and by day 30 much more significance is seen in both the groups compared to day 15. When compared between the groups, Group II dogs showed more response in clinical parameters, which is in accordance with the study conducted by Johnson *et al.* (2020), who reported more response in weight bearing with TENS compared to other physical rehabilitation techniques. Similarly, Riley *et al.* (2021) and Alves *et al.* (2022) reported more response in weight bearing with Ashwagandha compared to the dogs received oral nutraceuticals.

In the present study, the therapy resulted in considerable improvements in joint mobility in both groups I and II. Group I animals getting lubrihans, prednisolone and diathermy showed modest response by day 30, which is in consistent with the findings of McCarthy *et al.* (2007), who reported improvement in dogs receiving oral nutraceuticals by day 30. Whereas, group II dogs treated which received carprofen, Ashwagandha and TENS showed much improvement in joint motion by day 30 than group I dogs and is in accordance with the findings of Berte *et al.* (2012), who reported significant improvement in dogs underwent TENS than diathermy. Similarly, Krutika *et al.*, (2016) reported more improvement in Ashwagandha received group by day 30 compared to steroid treated group.

In the present study, significant difference in pain scores is seen in both group I and group II. Severe pain is noticed in more than half dogs in both the groups compared to apparently healthy dogs. Both groups showed reduction in pain by day 30, which is in accordance with the study conducted by McCarthy *et al.* (2007); Krutika *et al.* (2016) and Lamani *et al.* (2019). However, group II showed more improvement than group I by day 30, which is in accordance with Krutika *et al.* (2016), where they discussed that the most prominent Withanolides extracted from the leaves and dried roots of *Withania somnifera* is Withaferin A, a steroidal lactone, a physiologically active steroid, has been linked to anti-inflammatory action and this activity of Withaferin A is comparable to that of sodium succinate hydrocortisone helping in relieving the pain associated with OA. King (2018) reported that transcutaneous electrical stimulation (TENS) could be used to relieve pain through the stimulation of sensory nerve fibres and this procedure is conducted through the stimulation of either of two pain channels, the pain gate mechanism or the endogenous opioid mechanism by electrode placement attached to the skin. Pain relieving by NSAID, carprofen is due to suppression of prostaglandins which play a key role in the inflammatory process and also NSAIDs supress the effect of NF-B, a transcription factor for proinflammatory proteins like chemokines, adhesion molecules and cytokines as well as supress protein-I action and inhibit ROS, which aids in the relief of arthritis related pain (Osafo *et al.*, 2017).

According to table 4.9 & 4.10, there was a significant reduction in lameness score in both the groups after the therapy. Among the two groups, Group II showed higher significance than Group I, which is in consistence with the findings of Sharma *et al.* (2020) who documented that Ashwagandha had good relief effect compared to NSAIDs with least side effects and similarly Kirkby Shaw *et al.* (2019) stated that TENS provide greater relief in pain and disability among all the physical rehabilitation techniques.

In the present study, with respect to haematological findings in OA dogs, there is no significant variations reported in Hb (g%), TEC (10^6 cells/mm^3**)**, TLC (10^3 cells/mm^3) and differential leukocyte count (%) before and after therapy in both the groups and also there was no significant difference in between the groups, which is in accordance with the findings of Alam *et al.* (2006), McCarthy *et al. (*2007), Edamura *et al.* (2012), Molina *et al.* (2014) and Musco *et al.* (2019). Reddy *et al.* (2013) in their study on the OA induced rats and identified

that rats receiving ashwagandha does not show any significant changes in haematological parameters. Even in long term use of Ashwagandha, there will be no impact on haematological parameters, indicating no adverse effect. However, Prasad *et al.* (2012) reported significant increase in Hb (g%), TEC (10^6 cells/mm^3**)**, TLC (10^3 cells/mm^3) and differential leukocyte count i.e., Neutrophils (%), Lymphocytes (%), Eosinophils (%), Monocytes (%) by day 15 in dogs treated with NSADIs. The difference findings of haematology in OA may be due to the number of treatment days, as in present study NSAIDs were given only for 5 days.

In the current study, serum biochemical parameters like ALT (IU/L), AST (IU/L) and Creatinine (g/dL) are within the normal range before and after therapy in both the groups, as documented by Coles (1986), Alam *et al.* (2006), McCarthy *et al.* (2007) Musco *et al.* (2019) and Tandon and Yadav (2020). CRP was found to be within the normal range in group I and group II dogs before and after treatment which is in accordance with Hurter *et al.* (2005); Fujiki *et al.* (2007) and Hillstrom *et al.* (2016). However, Maiko and Bagirova (2009) reported that dogs treated with NSAIDs had significant increase in serum biochemical parameters like ALT, AST and creatinine, which is in contrary to the present study where NSAIDs were recommended only for 5 days. No change in serum biochemical properties even in long term use of Ashwagandha indicates no adverse effect on liver and kidney (Tandon and Yadav, 2020).

In the present study, synovial fluid volume did not differ significantly in both group I and group II dogs before and after therapy during the study period which is in accordance with the studies conducted by Coles (1986), Innes (1995), Fernandes (2002), MacWilliams and Friedrichs (2003), Anirudh and Ranganath (2015) and Wood and Gibson (2020). The reason for no change in synovial fluid volume in OA geriatric dogs is attributed to the noninflammatory response within in the joint.

In the present study, the colour of the synovial fluid obtained from group I and group II dogs ranged from colourless, pale yellow to straw coloured and red tinged. These findings are in agreement with MacWilliams and Friedrichs (2003). Normal synovial fluid colour ranges from colourless to light yellow (Jacques *et al.* 2002; Fernandes *et al.* 2002; Clements, 2006 and Sherry, 2014). The red tinge colour of synovial fluid in the present study might be due to haemorrhage during collection as reported by Coles (1986) and Jacques *et al.* (2002).

Viscosity of the synovial fluid is reported to be normal in most of the dogs but some dogs showed decrease in the viscosity, which correlates with the findings of Fernandes *et al.* (2002), Jacques *et al.* (2002) and MacWilliams and Friedrich (2003). Decrease in viscosity in OA cases is attributed to dilution of synovial fluid by hyaluronidase and lysosomal enzymes (Fernandes *et al.*, 2002) and also might be because of deficiency of polymerized hyaluronic acid or dilution from excess serum (Hopper, 1993). Decreased in viscosity may be due to decrease in concentration of hyaluronic acid as well as decrease in its chain length and molecular weight of (Herrero-Beaumont *et al.*, 2007).

In the present study, the synovial fluid staining revealed the presence of synovial macrophages, neutrophils and monocytes, which is in accordance with the findings of Mahaffey (2002) and MacWilliams and Friedrichs (2003). However, Steel (2008) revealed metachromatic granules in macrophages in synovial fluid staining of OA horses.

In the present investigation, the protein concentration in synovial fluid is significantly higher in both groups before therapy and after therapy significant reduction is noticed in both the groups. These findings are in agreement with the reports of Jacques *et al.* (2002), MacWilliams and Friedrichs (2003), Xu *et al.* (2009), Anirudh and Ranganath (2015) and Olsen *et al.* (2019). According to Shahid *et al.* (2018), synovial fluid sample must be free of blood contamination in order to estimate total protein content and failing to do so could result in falsely positive results.

In the present study, there was no significant difference in total nucleated cell counts in synovial fluid before and after therapy in both the groups, which is in accordance with the findings of Fernandes *et al.* (2002), MacWilliams and Friedrichs (2003) and Wood and Gibson (2020). Normal synovial fluid WBC count ranges from 0-2900 (Alleman *et al.*, 2007). These normal levels of WBC in synovial fluid is indicative of noninflammatory condition.

In the present study, there was no significant difference in mononuclear cell count in synovial fluid before and after therapy in both group I and II dogs, which is in consistency with the findings of Jacques *et al.* (2002), McLaughlin and Roush (2002) and MacWilliams and Friedrichs (2003). Also, there is no significant difference in neutrophilic count in synovial fluid before and after therapy in both the groups, which is in consistency with the findings of MacWilliams and Friedrichs (2003) and Wood and Gibson (2020).

In the current study, with respect to synovial fluid analysis, except significant elevation in total protein, there was no any significant change in the other parameters, indicating that osteoarthritis is a noninflammatory and is a degenerative condition that affects joints and is primarily caused by ageing, with little effect on the synovial fluid parameters.

In the present study, ventrodorsal view of hip joints are taken for radiographic view to know the bony changes in osteoarthritis. Similarly, Adams (2000), Lascelles and Robertson (2010) and Runge *et al.* (2010) also preferred the same sight for radiographic view of osteoarthritis changes. In the present study, joint space narrowing, osteophyte formation at the joint margin and irregular bone surfaces are seen on the 0^{th} day of examination, which is in accordance with the findings of Reed (2002), Peach *et al.* (2005), Lorenz and Richter (2006), Meeson *et al.* (2019) and Alves *et al.* (2021). Asesthesiophytes, which are osteophytes that originate at the location of a joint capsule's bone attachment or a nearby ligament or tendon insertion (Resnick and Niwayama, 1983). Radiographic changes in osteoarthritis can be seen only in chronic cases and this may not be effective diagnostic aid in early diagnosis of osteoarthritis and visible radiographic changes are observed in advanced cases (McLaughlin and Roush 2002 and Corfield *et al*., 2007).

In the present study, the common ultrasound findings seen in OA dogs are joint effusions, formation of osteophytes and bone erosions. Similar observations are also made by Boulocher *et al.* (2008), Goranav (2012); Wenham *et al.* (2014), Roemer et al. (2020) and Singh *et al.* (2021) in the USG examination of OA dogs. Ultrasound helps in identifying the early signs of osteoarthritis, which may not be visible under radiography (Gundi and Bertoni, 2001).

In the present study, CT scanning of osteoarthritis dog revealed presence of calcified growth with visible osteophyte on femoral neck on both the sides and also the extent of damage to the bone are clearly visible and these findings may not be appreciated by either radiographic or ultrasonographic examination. Similar findings are noticed by Kalichman and Hunter (2007), Wenham *et al.* (2014), Demehri *et al.* (2016), Roemer *et al.* (2020) and Jone *et al.* (2022) in their study on OA in dogs. CT is more useful in diagnosing the osteoarthritis as it gives a complete 3-dimensional view of affected bone and also helps to know the progression of illness (Demehri *et al.*, 2016; Roemer *et al.*,2020) and gives additional information of

calcified bones, hypertrophied bone (Wenham *et al.,* 2014) and even a single osteophyte formation (Jone *et al.*, 2022) which is not visible under plain radiography and MRI.

In the present study, the 20 positive cases were randomly divided into two groups. Group I dogs were treated with Inj prednisolone for 5 days followed by diathermy and lubrihans for 4 weeks, whereas Group II dogs were treated with Tab Carprofen for 5 days followed by TENS and Ashwagandha for 4 weeks. In the present study, group I dogs receiving nutraceuticals showed improvement in clinical signs and weight bearing by day 30, as reported by Lamani *et al.* (2019) and McCarthy *et al.* (2007), were they reported that dogs receiving nutraceuticals showed slight improvement by day 30 and dogs receiving prednisolone showed reduction in pain by day 3 as reported by Scott (2007), who reported that dogs receiving corticosteroids helped in reducing the inflammation associated with OA in dogs. Linchitz and Sorell (2003), reported that diathermy can be used in dogs suffering with osteoarthritis, where the hot waves penetrate deep upto 3-5 cms and helps in relieving pain associated with OA, which is in consistent with present day findings, where dogs receiving diathermy showed significant improvement by end of 2nd week of therapy, but slight adverse reactions like skin burns were noticed during course of treatment. Group II dogs receiving NSAIDs showed improvement in severity of pain by day 3 as reported by McCarthy *et al.* (2007) and Alves *et al.* (2017). Dogs receiving TENS showed drastic reduction in pain and severity of illness by end of 1st week, where TENS was applied for 15-20 minutes for 1-2 times a day as reported by Levine and Bockstahler (2014). Dogs receiving Ashwagandha showed good response to therapy by end of 2nd week of therapy, which is in consistent of findings of Sachin *et al*. (2017). To conclude, in the present study, dogs maintained with Ashwagandha showed more response than maintained with Lubrihans, which might be due to the effect of Ashwagandha on controlling obesity, which is a major risk factor for the progression of OA in dogs (Sachin *et al.,* 2017 and Sharma *et al*., 2020). Ashwagandha controls the level of cortisol in body caused because of old age. High cortisol in the body allows more deposition of fat, which predisposes to many of the age related issues like diabetes, hypertension and OA and Ashwagandha, when given, it controls the level of cortisol and thereby inhibits the deposition of excess fat in the body and controls obesity (McEwen, 2008). Similarly, group that received Ashwagandha showed drastic reduction in the body weight and showed good response in alleviating the signs of pain and discomfort.

Based on the findings in the present study, it could be concluded that osteoarthritis is a age related condition affecting geriatric dogs, 10-15 years age group being more vulnerable. With regard to diagnostic protocols, OA does not show any effect on haematological, serum and synovial fluid parameters except increase in protein concentration in synovial fluid with typical radiographical changes like osteophyte formations, subcondral bone sclerosis, joint space reduction and loss of femoral head contour. Among the imaging techniques CT is found to be more effective than radiography and ultrasonography. In the present study, it could be concluded that dogs maintained with Ashwagandha showed good response in terms of weight bearing and obesity, which is a major risk factor for the development of OA, when compared to Lubrihans supplemented group. Among the physical rehabilitation techniques, TENS showed good response in terms of relieving pain, weight bearing and also procedure is with less stress, compare to diathermy which is having certain adverse effects like skin burns and causes stress on dog during procedure. TENS can be recommended in severely affected OA as a part of routine physical rehabilitation technique in aged dogs and should be done atleast twice a day for minimum of 4 weeks to achieve good response. As osteoarthritis in geriatric dogs requires lifelong therapy, Ashwagandha along with TENS could be recommended to relieve pain with least or no adverse effects.

CHAPTER VI

SUMMARY

The current study "**CLINICO-DIAGNOSTIC AND THERAPEUTIC STUDIES ON OSTEOARTHRITIS IN GERIATRIC DOGS**" was investigated with the objectives of evaluating various factors involved in occurrence of osteoarthritis in geriatric dogs, various diagnostic techniques for osteoarthritis in geriatric dogs, management with appropriate therapy and to assess the incidence of osteoarthritis in geriatric dogs.

The geriatric dog cases that were brought to the Veterinary Clinical Complex, Campus Hospital, College of Veterinary Science, Rajendranagar and Veterinary Hospital, Bhoiguda during the period of January 2022 to June 2022 were screened for osteoarthritis. In addition, referral cases from practising veterinarians in and around Hyderabad and surrounding areas were also included.

In the current study, 3040 geriatric dogs presented during above period, out of which 350 dogs were presented with the signs of lameness, inability to bear weight and exercise intolerance were subjected to various diagnostic techniques like clinical examination, radiography, ultrasonography, CT scanning and were further estimated for complete blood analysis, serum biochemistry and synovial fluid analysis for confirmatory diagnosis of Osteoarthritis (OA). Age wise incidence of osteoarthritis was highest in dogs more than 10-15 years 220 (62.86%) followed by 6-10 years 105 (30%) and least in more than 15 years dogs 25 (7.14%). Breed wise incidence was found to be higher in Labrador Retriever 160 (45.71%) followed by German Shepherd 72 (20.57%), Rottweiler 38 (10.86%), non-descript 33 (9.42%), Pug 25 (7.14%), Spitz 13 (3.71%), Golden Retriever 8 (2.29%) and least was recorded in Dalmatian 1 (0.29%). With regards to gender wise incidence, males found to be more prone for osteoarthritis 210 (60%) compared to females140 (40%). Joint wise incidence was found to be high in hip joint 75 (82.57%), followed by elbow joint 54(15.43%) and least in stifle 07 (2%). The major risk factor associated with osteoarthritis was obesity, seen in 202 (57.71%) dogs, followed by neutering (12.86%), slippery floor (9.43%), overuse of calcium (8.57%), heavy exercise (5.71%), underlying joint issues (4.29%) and hypothyroidism (1.42%).

Pain (100%) was the most common clinical sign which was present in all the dogs presented, followed by reluctant to move in 310 (88.57%), lameness in 250 dogs (71.43%), sluggish rising in 180 (51.42%), difficulty in walking upstairs or jumping in 150 (42.87%), stiffness in 50 (14.28%) and reduced appetite in 20 (5.71%).

Dogs with osteoarthritis were divided randomly into two groups containing 10 in each and investigated further. However, 10 apparently healthy dogs were considered for comparison. Physiological parameters were recorded on day 0, 15 and 30 days and were within the normal range in both the groups before and after therapy and showed non significant difference ($P>0.05$) when compared to apparently healthy dogs. Clinical parameters like Weight bearing, Joint motion, Lameness grading and Pain scores were recorded on day 0, 15 and 30 in both group I and group II dogs and these parameters showed significant higher ($P<0.01$) when compared to apparently healthy dogs before therapy, whereas after therapy, showed significant reduction ($P<0.05$) compared to day 0. However, Group II showed more significant reduction ($P<0.05$) in all these parameters than group I by day 30.

Blood and serum samples from two groups were collected at 0, 15 and 30 day intervals and wide range of haematological and selected biochemical parameters were evaluated and compared with the healthy control. There was no significant difference ($P>0.05$) in Hb (g%), Total leucocyte count (10^3 cells/mm^3) and differential leukocyte count (%) before and after therapy when compared to apparently healthy dogs. There was no significant difference ($P>0.05$) in ALT (IU/L), AST (IU/L), creatinine (mg/dL) and CRP (g/L) when compared to apparently healthy dogs, before and after therapy. These findings demonstrated that the haematological and biochemical markers in osteoarthritis remain normal. Synovial analysis in the present study revealed normal volume, normal to decreased in severely affected OA dogs, colour ranging from colourless to red tinged and showed significantly high ($P < 0.05$) total protein on day 0 when compared to apparently healthy dogs and after therapy significantly ($P < 0.05$) reduced by day 30 in both the groups, whereas there was no significant difference before and after therapy findings of TNCC (cells/min) and DLC (%) when compared to healthy control. Present findings indicate that in synovial fluid analysis except increased total protein, there was no changes in other the parameters of synovial fluid. Though the synovial analysis

does not show any significant variations in osteoarthritis, it is an important diagnostic technique to differentiate inflammatory and non-inflammatory arthropathies.

Radiography examination demonstrated advanced osteoarthritis symptoms such as joint space narrowing, subchondral bone sclerosis and the presence of osteophytes at the joint edges on day 0 of the study and by day 30 of therapy, no much difference was noted, however when the cases were followed up for 90 days, there was a significant drop in key signs with osteophyte density reduction. However, the soft tissue damage associated with OA cannot be observed on radiography, hence, ultrasonography was used. Ultrasonography of joints, which was examined by using linear transducer of 10-12 Hz revealed low grade myositis and tendinitis and by day 30 of therapy, myositis and tendinitis disappeared in both the groups. Further, CT was carried out in some of the dogs, to know the progress of the disease which revealed presence of hypertrophied femoral neck, which was not evident in plain radiography. Based on these findings, it could be concluded that CT is found to be more accurate in the diagnosis of osteoarthritis, followed by radiography and ultrasonography helps in identifying the soft tissue damage.

In the current study, a complete diagnosis of osteoarthritic cases was made using clinical signs, radiography, ultrasonography, CT, haematology and serum biochemistry parameters and synovial fluid analysis and positive cases were randomly assigned to two groups, Group I and Group II, each with ten dogs and were given two different therapeutic regimens. Group I dogs were treated with Inj Prednisolone for 5 days, Tab. Lubrihans for 30 days and application of Diathermy with an interval of twice a day in more severe cases and twice a week in less severe cases for 30 days and Group II received Tab. Carprofen for 5 days, oral Ashwagandha powder for 30 days with application of TENS twice a day in more severe cases and twice a week in less severe cases for 30 days. Group II dogs significant reduction in clinical signs compared to Group I, indicating that Ashwagandha, in conjunction with TENS and carprofen is more effective in treating osteoarthritis with no side effects than corticosteroids, which have an effect on the kidney after long-term use.

The current study findings and observations lead to the conclusion that, among the various diagnostic techniques available, radiography alone may not be sufficient for complete diagnosis of osteoarthritis because early stages are not visible under radiography and it should

be combined with other techniques such as ultrasound and CT for complete diagnosis. Dogs in Group II performed better than dogs in Group I, indicating that among the current treatment agents available in the market, Tab Carprofen @ 4.4 mg/kg once a day for 5 days, application of TENS for 4 weeks with an interval of twice a week in severely affected dogs and in less severe cases, it should be applied twice a day and maintenance with Ashwagandha could be recommended for acute or chronic cases of osteoarthritis geriatric dogs, as they does not produce any adverse effects on aged dogs and further Ashwagandha showed drastic improvement in weight reduction, an important risk factor associated with osteoarthritis and is highly recommended in all osteoarthritic cases irrespective of their age in both acute and chronic cases of osteoarthritis.

LITERATURE CITED

Adams W M, Dueland R T, Daniels R, Fialkowski, J P and Nordheim E V. 2000. Comparison of two palpation, four radiographic and three ultrasound methods for early detection of mild to moderate Canine Hip Dysplasia. *Veterinary Radiology and Ultrasound* **41** (6): 484-490.

Alam M R, Lee H B, Park S Y, Lee Y H, Kim L S, Kang H S and Kim N S. 2006. Changes in the Hematobiochemical Parameters in Experimental Stifle Osteoarthritis in Dogs. *Pakistan Journal of Biological Science* **9**: 2869-2874.

Alleman A R, Denicola D B and Wamsley H L. 2007. Synovial Fluid Analysis: 79th Western Veterinary Conference: 10.

Al-Saleem S, Alshahrani A and Al-Khaldi Y. 2013. Obesity among patients attending Primary Care Centers, Aseer Region, Saudi Arabia. *Saudi Journal of Obesity* **1** (2): 67-77.

Alves J C, Santos A M and Jorge P I. 2017. Effect of an oral joint supplement when compared to carprofen in the management of hip osteoarthritis in working dogs. *Topics in companion animal medicine* **32** (4): 126-129.

Alves J C, Santos A, Jorge P, Lavrador C and Carreira L M. 2020. Clinical and Diagnostic Imaging Findings in Police Working Dogs Referred for Hip Osteoarthritis. *BMC Veterinary Research* **16** (1): 1-11.

Alves J C, Santos A, Jorge P, Lavrador C and Carreira L M. 2021. Intraarticular Triamcinolone Hexacetonide, Stanozolol, Hylan GF 20 and platelet Concentrate in a naturally occurring Canine Osteoarthritis model. *Scientific Reports* **11** (1): 1-9.

Alves J C, Santos A, Jorge P, Lavrador C and Carreira L M. 2022. Characterization of Weight-bearing Compensation in Dogs with Bilateral Hip Osteoarthritis. *Topics in Companion Animal Medicine* **49**: 1-5.

Anderson K L, O'Neill D G, Brodbelt D C, Church D B, Meeson R L, Sargan D, Summers J F, Zulch H and Collins L M. 2018. Prevalence, Duration and Risk Factors for Appendicular Osteoarthritis in A UK Dog Population Under Primary Veterinary Care. *Scientific Reports* **8** (1): 1-12.

Anderson K L, Zulch H, O Neill D G, Meeson R L And Collins L M. 2020. Risk Factors for Canine Osteoarthritis and its Predisposing Arthropathies: A Systematic Review. *Frontiers In Veterinary Science* **7** (20): 1-38.

Anirudh A and Ranganath L. Synovial Fluid Analysis in Dogs with Elbow, Hip and Stifle Joint Disorders. 2015. *International Journal of Applied and Pure Science and Agriculture* **1** (7): 2394-5532.

Aragon C L, Hofmeister E H and Budsberg S C. 2007. Systematic Review of Clinical Trials of Treatments for Osteoarthritis in Dogs. *Journal of the American Veterinary Medical Association* **230** (4): 514-521.

Aspinall V. 2011. The Complete Textbook of Veterinary Nursing 2: *The Complete Textbook of Veterinary Nursing*, 4rth edition. Elsevier Health Sciences. 1-10

Aulakh K S, Lopez M J, Hudson C, Gaschen L, Fabiani M, Beale B, Andrews F M, Liu C C and Lattimer J. 2021. Prospective Clinical Evaluation of Intra-Articular Injection of Tin-117m (117msn) Radiosynoviorthesis agent for management of naturally occurring elbow Osteoarthritis in Dogs: A Pilot Study. Veterinary Medicine: *Research and Reports* **12** (21): 117-128.

Barker T, Rogers, V E, Henriksen V T, Trawick R H, Momberger N G and Lynn Rasmussen G. 2021. Circulating IL-10 is compromised in patients predisposed to developing and in patients with severe knee osteoarthritis. *Scientific reports* **11** (1) :1-10.

Beale B S. 2004. Use of Nutraceuticals and Chondroprotectants in Osteoarthritic Dogs and Cats. Veterinary Clinics: *Small Animal Practice* **34** (1): 271-289.

Bennett D and May C. 1995. Joint Diseases of Dogs and Cats. *Textbook Of Veterinary Internal Medicine, 3rd Edition.* Philadelphia: WB Saunders. 2032-77.

Bergamino C, Etienne A L and Busoni V. 2015. Developing a technique for Ultrasound-Guided injection of the adult Canine Hip. *Veterinary Radiology and Ultrasound* **56** (4): 456-461.

Bermingham E N, Thomas D G, Cave N J, Morris P J, Butterwick R F and German A J. 2014. Energy requirements of adult dogs: a meta-analysis. *Plos one* **9** (10): 1-23

Berte L, Mazzanti A, Salbego F Z, Beckmann D V, Santos R P, Polidoro D and Baumhardt R. 2012. Immediate physical therapy in dogs with rupture of the cranial cruciate ligament submitted to extracapsular surgical stabilization. *Arquivo Brasileiro de Medicina Veterinaria e Zootecnia* **64** (1): 01-08.

Bland S D. 2015. Canine Osteoarthritis and Treatments: A Review. *Veterinary Science Development* **5** (2): 84-89.

Boulocher C, Duclos M E, Arnault F, Roualdes O, Fau D, Hartmann D J and Viguier E. 2008. Knee Joint Ultrasonography of The ACLT Rabbit experimental model of Osteoarthritis: Relevance and Effectiveness in Detecting Meniscal Lesions. *Osteoarthritis and Cartilage* **16** (4): 470-479.

Bui L M and Bierer T L. 2003. Influence Of Green Lipped Mussels (*Perna Canaliculus*) in alleviating signs of Arthritis in Dogs. *Veterinary Therapeutics* **4** (4): 397-407.

Bullock J, Rizvi S A, Saleh A M, Ahmed S S, Do D P, Ansari R A and Ahmed J. 2018. Rheumatoid Arthritis: A brief overview of the treatment. *Medical Principles and Practice* **27** (6): 501-507.

Capon H. 2021. Incorporating a rehabilitation into the management of Canine Osteoarthritis. *Companion Animal* **26** (7): 139-146.

Chalmers H J, Dykes N L, Lust G, Farese J P, Burton-Wurster N I, Williams A J and Todhunter R J. 2006. Assessment of bone mineral density of the femoral head in dogs with early osteoarthritis. *American journal of veterinary research* **67** (5): 796-800.

Clark R D. 2015. *Medical, Genetic and Behavioural risk factors of Kuvaszok and Komondor,* 2nd edition. pp 1-5.

Clegg D O, Reda D J, Harris C L, Klein, M A, O'Dell J R, Hooper M M, Bradley J D, Bingham III C O, Weisman M H, Jackson C G and L and N E. 2006. Glucosamine, Chondroitin Sulfate and the two in combination for Painful Knee Osteoarthritis. *New England Journal of Medicine* **354** (8): 795-808.

Clements D. 2006. Arthrocentesis and Synovial Fluid Analysis in Dogs and Cats. *In Practice* **28** (5): 256–262.

Clouet J, Vinatier C, Merceron C, Pot-Vaucel M, Maugars Y, Weiss P, Grimandi G. and Guicheux J. 2009. From Osteoarthritis Treatments to Future Regenerative Therapies for Cartilage. *Drug Discovery Today* **14** (19-20): 913-925.

Coles E H. 1986. *Veterinary Clinical Pathology,* 4rth edition. W.B. Saunders company, Philadelphia. pp 241-266.

Corfield G S, Read R A, Eastley K A, Richardson J L, Robertson I D and Day R. 2007. Assessment of the hip reduction angle for predicting osteoarthritis of the hip in the Labrador Retriever. *Australian veterinary journal* **85** (6): 212-216.

Corral M J, Moyaert H, Fernandes T, Escalada M, Tena J K S, Walters R R and Stegemann M R. 2021. A prospective, randomized, blinded, placebo-controlled multisite clinical study of bedinvetmab, a Canine Monoclonal Antibody targeting nerve growth factor in dogs with Osteoarthritis. *Veterinary Anaesthesia and Analgesia* **48** (6): 943-955.

Daems R, Van Hecke L, Schwarzkopf I, Depuydt E, Broeckx S Y, David M and Spaas J H. 2019. A feasibility study on the use of Equine chondrogenic induced mesenchymal stem cells as a treatment for natural occurring osteoarthritis in Dogs. *Stem Cells International* **2019**: 1–11.

Debbie G. 2006. *Pain Management in Small Animals, 1st Edition*, Elsevier, China. pp 1-15.

DeGroot J, Verzijl N, Wenting-Van Wijk M J, Jacobs K M, Van El B, Van Roermund P M and Lafeber F P. 2004. Accumulation of advanced glycation end products as a molecular mechanism for aging as a risk factor in Osteoarthritis. *Arthritis and Rheumatism: Official Journal of the American College of Rheumatology* **50** (4): 1207-1215.

Demehri S, Guermazi A and Kwoh C K. 2016. Diagnosis and longitudinal assessment of osteoarthritis: review of available imaging techniques. *Rheumatic Disease Clinics* **42** (4): 607-620.

Devkar M S T. 2015. Studies in validation of Withania Somnifera (L). Dunal (Ashwagandha) by Physical, Chemical and Biological approaches with respect to major bioactive Withanolides (Doctoral Dissertation, Bharati Vidyapeeth). A Thesis Submitted to Bharati Vidyapeeth Deemed University, Pune.

Dragos D, Gilca M, Gaman L, Vlad A, Iosif L, Stoian I and Lupescu O. 2017. Phytomedicine In Joint Disorders. *Nutrients* **9** (1): 1-18.

Dyce K M, Sack W O and Wensing C J G. 1996. *Textbook Of Veterinary Anatomy,* 2nd edition, W.B. Saunders Company, Philadelphia. pp 14-15.

Edamura K, King J N, Seewald W, Sakakibara N and Okumura M. 2012. Comparison of oral robenacoxib and carprofen for the treatment of osteoarthritis in dogs: a randomized clinical trial. *Journal of Veterinary Medical Science*. 1-29.

Elliott D, Servet E and Biourge V. 2007. Nutritional management of Canine Osteoarthritis. *Veterinary Focus* **17** (3): 43-48.

Evans H E and De Lahunta A. 2013. *Miller's anatomy of the dog-E-Book.* Elsevier health sciences. 127-151.

Fernandes F R, Grindam C B, Lipowitz A J and Perman V. 2002. Synovial Fluid Analysis: Preparation of smears for Cytological examination of Canine Synovial Fluid. *Journal of American Animal Hospital Association* **19**: 727-734.

Flaherty M J. 2019. Rehabilitation therapy in perioperative pain management. Veterinary Clinics: *Small Animal Practice* **49** (6): 1143-1156.

Fujiki M, Shineha J, Yamanokuchi K, Misumi K and Sakamoto H. 2007. Effects of treatment with polysulfated Glycosaminoglycan on serum cartilage oligomeric matrix protein and C-Reactive protein concentrations, serum matrix metalloproteinase-2 and-9 activities and lameness in dogs with osteoarthritis. *American Journal of Veterinary Research* **68** (8): 827-833.

German A J. 2006. The growing problem of obesity in Dogs and Cats. *The Journal of Nutrition* **136** (7): 1940-1946.

Ginja M M D, Silvestre A M, Colaco J, Gonzalo-Orden J M, Melo-Pinto P, Orden M A, Llorens-Pena M P and Ferreira A J. 2009. Hip dysplasia in Estrela Mountain dogs: Prevalence and genetic trends 1991–2005. *The Veterinary Journal* **182** (2): 275-282.

Goggs R, Vaughan-Thomas A, Clegg P D, Carter S D, Innes J F, Mobasheri A, Shakibaei M, Schwab W and Bondy C A. 2005. Nutraceutical therapies for Degenerative Joint Diseases: A Critical Review. *Critical Reviews in Food Science and Nutrition* **45** (3): 145-164.

Goldberg M E. 2022. Osteoarthritis in Canines Part 2: Physical Rehabilitation. *The Veterinary Nurse* **13** (1): 10-15.

Goldring S R and Goldring M B. 2006. Clinical aspects, Pathology and pathophysiology of osteoarthritis. *Journal of Musculoskeletal and Neuronal Interactions* **6** (4): 376-378.

Goldston R T and Hoskins J D. 1995. *Geriatrics and Gerontology of the Dog and Cat, Edn 2*, WB Saunders Co. pp 1-20.

Goranov N V. 2012. Clinical Changes in sodium monoiodoacetate-Induced Stifle Osteoarthritis model in Dogs. *Veterinary World* **5** (3): 138-144.

Gossec L and Dougados M. 2004. Intra-articular treatments in osteoarthritis: from the symptomatic to the structure modifying. *Annals of the rheumatic diseases* **63** (5): 478-482.

Gundi G and Bertoni G. 2001. Echographic Examination of the stifle joint affected by Cranial Cruciate Ligament Rupture in the Dog. *Veterinary Radiology and Ultrasound* **42** (3): 266-270.

Hanks J, Levine D and Bockstahler B. 2015. Physical agent modalities in Physical therapy and Rehabilitation of Small Animals. *Veterinary Clinics: Small Animal Practice* **45** (1): 29-44.

Hawks D. 2002. Alternative Medicine: Musculoskeletal System. Clinical Techniques in *Small Animal Practice* **17** (1): 41-49.

Hays L, Zhang Z, Mateescu R G, Lust G, Burton-Wurster N I and Todhunter R J. 2007. Quantitative Genetics of secondary hip joint osteoarthritis in a Labrador Retriever–Greyhound Pedigree. *American Journal of Veterinary Research* **68** (1): 35-41.

Hazewinkel H A W. 2006. *Disease-Modifying Drugs in Canine Osteoarthritis*. in Proceedings of the 13th annual ESVOT Congress, Munich, Germany.

Henrotin Y, Sanchez C and Balligand M. 2005. Pharmaceutical and Nutraceutical Management of Canine Osteoarthritis: Present and future Perspectives. *The Veterinary Journal* **170** (1): 113-123.

Herrero-Beaumont, G Ivorra J A R, del Carmen Trabado, M, Blanco F J, Benito P, Martín-Mola E, Paulino J, Marenco J L, Porto A, Laffon A and Araujo D. 2007. Glucosamine sulfate in the treatment of knee osteoarthritis symptoms: a randomized, double-blind, placebo-controlled study using acetaminophen as a side comparator. *Arthritis & Rheumatism: Official Journal of the American College of Rheumatology* **56** (2): 555-567.

Hielm-Bjorkman A K, Kuusela E, Liman A, Markkola A, Saarto E, Huttunen P, Leppaluoto J, Tulamo R M and Raekallio M. 2003. Evaluation of methods for assessment of pain associated with Chronic Osteoarthritis in Dogs. *Journal of the American Veterinary Medical Association* **222** (11): 1552-1558.

Hillstrom A, Bylin J, Hagman R, Bjorhall K, Tvedten H, Konigsson K and Kjelgaard-Hansen M. 2016. Measurement of Serum C-Reactive Protein concentration for

discriminating between suppurative arthritis and osteoarthritis in Dogs. *BMC Veterinary Research* **12** (1): 1-10.

Hopper P E. 1993. Immune-mediated joint diseases: *Textbook of Small Animal Surgery*,2nd edition. Philadelphia, A, Saunders. 1928-1937.

Hurter K, Spreng D, Rytz U, Schawalder P, Ott-Knusel F and Schmokel H. 2005. Measurements of C-Reactive Protein in serum and lactate dehydrogenase in serum and synovial fluid of patients with osteoarthritis. *The Veterinary Journal* **169** (2): 281-285.

Hyytiainen H K, Molsa S H, Junnila J T, Laitinen-Vapaavuori O M and Hielm-Björkman A K. 2012. Use of bathroom scales in measuring asymmetry of hindlimb static weight bearing in dogs with osteoarthritis. *Veterinary and Comparative Orthopaedics and Traumatology* **25** (05): 390-396.

Impellizeri J A, Tetrick M A and Muir P. 2000. Effect of weight reduction on clinical signs of lameness in dogs with hip osteoarthritis. *Journal of American Veterinary Medical Association* **216**: 1089-1091.

Innes J. 1995. Diagnosis and treatment of Osteoarthritis in Dogs. *In Practice* **17** (3): 102-109.

Innes J. 2006. Diet and Disease: Exploring the link through nutrigenomics. *The Canadian Veterinary Journal* **47** (1): 68-70.

Jacques D, Cauzinille L, Bouvy B and Dupre G. 2002. A retrospective study of 40 dogs with polyarthritis. *Veterinary Surgery* **31** (5): 428-434.

Jain S, Patel P R and Raval S K. 2015. A Study of Diseases in Geriatric Dogs. *Indian Journal of Animal Research* **49** (6): 866-868.

Johnson K A, Lee A H and Swanson K S. 2020. Nutrition and Nutraceuticals in the changing management of osteoarthritis for Dogs and Cats. *Journal of the American Veterinary Medical Association* **256** (12): 1335-1341.

Johnston S A, Mclaughlin R M and Budsberg S C. 2008. Nonsurgical Management of Osteoarthritis in Dogs. *Veterinary Clinics of North America: Small Animal Practice* **38** (6): 1449-1470.

Jone G M, Pitsillides A A and Meeson R L. 2022. Moving beyond the limits of detection: the past, the present, and the future of diagnostic imaging in canine osteoarthritis. *Frontiers in Veterinary Science* **9**: 1-32.

Juge P A, Berard L, Kotti S, Doursounian L, Sautet A, Simon T, Berenbaum F, Nourissat G and Sellam J. 2017. Cardiometabolic risk factors in primary centred and Rotator Cuff-Related shoulder Osteoarthritis: A Comparative Study. *RMD Open* **3 (**1): 1-5.

Kalichman L and Hunter D J. 2007. October. Lumbar facet joint osteoarthritis: a review. *In Seminars in arthritis and rheumatism* 37 (2): 69-80.

Kim S E, Pozzi A, Yeh J C, Lopez-Velazquez M, Au Yong J A, Townsend S, Dunlap A E, Christopher S A, Lewis D D, Johnson M D and Petrucci K. 2019. Intra-articular umbilical cord derived mesenchymal stem cell therapy for Chronic Elbow Osteoarthritis in Dogs: A Double-Blinded, Placebo-Controlled Clinical Trial. *Frontiers In Veterinary Science*. 1-23.

King M. 2018. Use of Electrophysical agents (Epas) in Small Animal Veterinary Practice. *Companion Animal* **23** (10): 585-591.

Kirkby Shaw K, Alvarez L, Foster S A, Tomlinson J E, Shaw A J and Pozzi A. 2019. Fundamental principles of rehabilitation and musculoskeletal tissue healing. *Veterinary Surgery* **49** (1)- 22-32.

Kogan L, Hellyer P and Downing R. 2020. The Use of Cannabidiol-Rich Hemp Oil Extract to treat Canine Osteoarthritis-Related Pain: A Pilot Study. *American Holistic Veterinary Medical Association Journal* **58** (2020): 1-10.

Krutika J, Tavhare S, Panara K, Kumar P and Karra N. 2016. Studies of ashwagandha (*Withania somnifera dunal). International Journal of Pharmaceutical and Biological science Archive* **7** (1): 1-11.

Kutzler M A. 2020. Possible relationship between long-term adverse health effects of gonad-removing surgical sterilization and Luteinizing hormone in Dogs. *Animals* **10** (4): 599-610.

Lamani T S D, Ranganath L, Nagaraja B N, Satyanarayana M L, Jamuna K V and Kamran C A. 2019. Study on the influence of Nutraceuticals on Osteoarthritis in Dogs. *International Journal of Current Microbiology and applied Sciences* **8** (7): 978-984.

Lascelles B D X and Robertson S A. 2010. DJD-associated pain in Cats: What can we do to promote patient comfort? *Journal of Feline Medicine and Surgery* **12** (3): 200-212.

Lauten S D. 2006. Nutritional risks to Large-Breed Dogs: From weaning to the geriatric years. Veterinary Clinics: *Small Animal Practice* **36** (6): 1345-1359.

Lawrence Z B, Rogers R and Schmitt Lavin E. 2022. Case Study: Degenerative Joint Disease in Canines. *Mako: NSU Undergraduate Student Journal* **2022** (1): 1-18.

Levine D and Bockstahler B. 2014. Electrical Stimulation. *Canine Rehabilitation and Physical Therapy* **2**: 342-58.

Linchitz R and Sorell P. 2003. Physical Therapy Techniques. In Pain Medicine a Comprehensive Review. Mosby, Philadelphia.

Lindley S and Taylor P. 2010. Chronic Pain. In *BSAVA Manual of Canine and Feline Rehabilitation, Supportive and Palliative Care*. BSAVA Library. 18-30.

Loef M, Schoones J W, Kloppenburg M and Ioan-Facsinay A. 2019. Fatty acids and osteoarthritis: different types, different effects. *Joint Bone Spine* **86** (4): 451-458.

Lorenz H and Richter W. 2006. Osteoarthritis: Cellular and Molecular Changes in Degenerating Cartilage. *Progress in Histochemistry and Cytochemistry* **40** (3): 135-163.

Lund E M, Armstrong P J, Kirk C A and Klausner J S. 2006. Prevalence and Risk factors for obesity in adult dogs from private US Veterinary Practices. *International Journal of Applied Research in Veterinary Medicine* **4** (2): 177-186.

MacWilliams P S and Friedrichs K R. 2003. Laboratory Evaluation and interpretation of Synovial Fluid. *Veterinary Clinics of North America: Small Animal Practice* **33** (1): 153–178.

Mahaffey E A. 2002. Synovial Fluid. Diagnostic Cytology and Hematology of the Horse, 163–170.

Malek S, Sample S J, Schwartz Z, Nemke B, Jacobson P B, Cozzi E M, Schaefer S L, Bleedorn J A, Holzman G and Muir P. 2012. Effect of Analgesic therapy on clinical outcome measures in a randomized controlled trial using client-owned dogs with hip Osteoarthritis. *BMC Veterinary Research* **8** (1): 1-17.

Marshall W G, Bockstahler B A, Hulse D A and Carmichael S. 2009. A Review of Osteoarthritis and Obesity: Current Understanding of the relationship and benefit of obesity treatment and prevention in the Dog. *Veterinary and Comparative Orthopaedics and Traumatology* **22** (05): 339-345.

Masiero S, Pignataro A, Piran G, Duso M, Mimche P, Ermani M and Del Felice A. 2020. Short-Wave Diathermy in the clinical management of Musculoskeletal Disorders: A Pilot Observational Study. *International Journal of Biometeorology* **64** (6): 981-988.

May S A. 1995. Degenerative joint disease (osteoarthritis, osteoarthrosis, secondary joint disease). Houlton Jef; Collinson, RW Manual of small animal arthrology. 62-74.

McCarthy G, O'Donovan J, Jones B, Mcallister H, Seed M and Mooney C. 2007. randomized Double-Blind, Positive-Controlled Trial to assess the efficacy of Glucosamine/Chondroitin Sulfate for the treatment of Dogs with Osteoarthritis. *The Veterinary Journal* **174** (1): 54-61.

McEwen B S. 2008. Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. *European Journal of Pharmacology* **583** (2-3): 174–185.

Mclaughlin R M and Roush J K. 2002. Symposium on Osteoarthritis. *Veterinary Medicine* **95**: 107-110.

Mclaughlin R. 2000. Management of Chronic Osteoarthritic Pain. *Veterinary Clinics: Small Animal Practice* **30** (4): 933-949.

Meeson R L, Todhunter R J, Blunn G, Nuki G and Pitsillides A A. 2019. Spontaneous Dog Osteoarthritis-A One Medicine Vision. *Nature Reviews Rheumatology* **15** (5): 273-287.

Mele E. 2007. Epidemiology Of Osteoarthritis. *Veterinary Focus* **17** (3): 4-10.

Millis D L, Francis D and Adamson C. 2005. Emerging Modalities in Veterinary Rehabilitation. *Veterinary Clinics: Small Animal Practice* **35** (6): 1335-1355.

Millis D L, Tichenor M, Hecht S. Prevalence of osteoarthritis in dogs undergoing routine dental prophylaxis. 2014. In: Proceedings of the 39th World Small Animal Veterinary Association Meeting; Cape Town, South Africa.

Millis D L. 2015. Physical Therapy and Rehabilitation in Dogs. *Handbook Of Veterinary Pain Management*,2nd edition. pp 2383-421.

Molina D V, Alzate V D, Ruiz B J, Urrea A M and Tobon J J. 2014. Analgesic Effect and Side effects of Celecoxib and Meloxicam in Canine Hip Osteoarthritis. *Revista MVZ Cordoba* **19** (3): 4289-4300.

Monteiro B P, Lambert C, Bianchi E, Genevois J P, Soldani G and Troncy E. 2019. Safety and Efficacy of reduced dosage Ketoprofen with or without tramadol for Long-Term treatment of Osteoarthritis in Dogs: A Randomized Clinical Trial. *BMC Veterinary Research* **15** (1): 1-11.

Moreau M, Dupuis J, Bonneau N H and Desnoyers M. 2003. Clinical Evaluation of a Nutraceutical, Carprofen and Meloxicam for the treatment of Dogs with Osteoarthritis. *Veterinary Record* **152** (11): 323-329.

Mortellaro C M. 2003. Pathophysiology Of Osteoarthritis. *Veterinary Research Communications* **27** (1): 75-78.

Mosley C, Edwards T, Romano L, Truchetti G, Dunbar L, Schiller T, Gibson T, Bruce C and Troncy E, 2022. Proposed Canadian Consensus Guidelines on Osteoarthritis Treatment Based on OA-COAST Stages 1–4. *Frontiers in Veterinary Science* **9** (22): 1-23.

Musco N, Vassalotti G, Mastellone V, Cortese L, Della Rocca G, Molinari M L and Lombardi P. 2019. Effects of a Nutritional Supplement in Dogs affected by Osteoarthritis. *Veterinary Medicine and Science* **5** (3): 1-11.

Nganvongpanit K, Boonsri B, Sripratak T and Markmee P. 2013. Effects of One-Time and Two-Time Intra-Articular Injection of Hyaluronic Acid Sodium Salt after Joint Surgery in Dogs. *Journal Of Veterinary Science* **14** (2): 215-222.

O'Neill D G, Brodbelt D C, Hodge R, Church D B and Meeson R L. 2020. Epidemiology and Clinical Management of Elbow Joint Disease in Dogs Under Primary Veterinary Care in the UK. *Canine Medicine and Genetics* **7** (1): 1-15.

Olsen A, Johnson V, Webb T, Santangelo K S, Dow S and Duerr F M. 2019. Evaluation of Intravenously delivered Allogeneic Mesenchymal Stem Cells for treatment of Elbow Osteoarthritis in Dogs: A Pilot Study. *Veterinary and Comparative Orthopaedics and Traumatology* **32** (03): 173-181.

Osafo N, Agyare C, Obiri D D and Antwi A O. 2017. Mechanism of Action of Nonsteroidal Anti-Inflammatory Drugs. *Nonsteroidal Anti-Inflammatory Drugs,* chapter 2. 1-5.

Paradis M, Sauve F, Charest J, Refsal K R, Moreau M and Dupuis J. 2003. Effects Of Moderate to Severe Osteoarthritis on Canine Thyroid Function. *The Canadian Veterinary Journal* **44** (5): 407-412.

Paul S, Chakraborty S, Anand U, Dey S, Nandy S, Ghorai M and Dey A. 2021. *Withania Somnifera* (L.) Dunal (Ashwagandha): A Comprehensive Review on Ethnopharmacology, Pharmacotherapeutics, Biomedicinal and Toxicological Aspects. *Biomedicine and Pharmacotherapy* **143**: 5-40.

Peach C A, Carr A J and Loughlin J. 2005. Recent Advances in the Genetic Investigation of Osteoarthritis. *Trends in Molecular Medicine* **11** (4): 186-191.

Pers Y M and Jorgensen C. 2013. Mesenchymal Stromal Cells: Updates and Therapeutic Outlook in Rheumatic Diseases. *Journal of Clinical Medicine* **2** (4): 201-213.

Pettitt R A and German A J. 2015. Investigation and Management of Canine Osteoarthritis. *In Practice* **37 (**15): 1-8.

Piel M J, Kroin J S, Van Wijnen A J, Ranjan K C and Hee-jong I M. 2014. Pain assessment in animal models of osteoarthritis. *Gene* **537** (2): 184-188.

Prasad A, Shafiuzama Mohd, Ayyappan S, Suresh Kumar R and Jayaprakash R. 2012. Incidence Of Coxofemoral Joint Affections in dogs. A Clinical Study of 575 Patients. *Intas Polivet* **13** (2): 281-283.

Preet G S, Turkar S, Gupta S and Kumar S. 2021. Dog obesity: Epidemiology, risk factors, diagnosis and management: A review paper. *Journal of Pharma Innovation* **10** (5): 698-705.

Rademacher N, Ohlerth S, Doherr M G, Gaschen L, Stoffel M H and Lang J. 2005. Doppler Sonography of the Medial Arterial Blood Supply to the Coxofemoral Joints of 36 Medium to Large Breed Dogs and its Relationship with Radiographic Signs of Joint Disease. *Veterinary Record* **156** (10): 305-309.

Raditic D and Athens G. 2019. Nutrition and Osteoarthritis: What Do We Know? https://todaysveterinarypractice.com.

Raffan E, Smith S P, O'Rahilly S and Wardle J. 2015. Development, factor structure and application of the Dog Obesity Risk and Appetite (DORA) questionnaire. *Peer Journal* **3**: 2-27.

Ramirez-Flores G I, Angel-Caraza D, Quijano-Hernández I A, Hulse D A, Beale B S. and Victoria-Mora J M. 2017. Correlation between Osteoarthritic changes in the Stifle Joint in Dogs and the results of Orthopaedic, Radiographic, Ultrasonographic and Arthroscopic Examinations. *Veterinary Research Communications* **41 (**2): 129-137.

Ranganath L. 2012. Arthritis in Dogs. 4th National Symposium on Companion Animal Practice. 129-139.

Reddy K R, Babu S V, Raghavendra N, Sridhar M, Nipanikar S U and Kuber V V. 2013. Safety assessment of tlpl/ay/03/2008, a polyherbal formulation in sprague dawley rats. *Toxicology International* **20** (1): 77-86.

Reed W. 2002. Radiographer reporting-aspects of the UK Experience. Radiographer: *The Official Journal of the Australian Institute of Radiography* **49** (3): 147-149.

Resnick D and Niwayama G. 1983. Entheses and enthesopathy. Anatomical, pathological, and radiological correlation. *Radiology* **146** (1): 1-9.

Richardson D C and Toll P W. 1997. Relationship of Nutrition to Developmental Skeletal Disease in Young Dogs. *Veterinary Clinical Nutrition* **4** (1): 6-13.

Riegger-Krugh C, Millis D L and Weigel J P. Canine Anatomy. Millis D L, Levine D, Taylor RA (eds): *Textbook of Canine Rehabilitation and Physical Therapy,* 1st edition, Philadelphia, Elsevier. 38-99.

Riley L M, Satchell L, Stilwell L M and Lenton N S. 2021. Effect of massage therapy on pain and quality of life in dogs: A cross sectional study. *Veterinary Record* **189** (11): 1-10.

Riva G T, Hart B L, Farver T B, Oberbauer A M, Messam L L M, Willits N and Hart L A. 2013. Neutering dogs: effects on joint disorders and cancers in golden retrievers. *PloS one* **8** (2): 1-7.

Rocha B D and Torres R C S. 2007. Ultrasonic and Radiographic Study of Laxity in Hip Joints of Young Dogs. *Arquivo Brasileiro De Medicina Veterinária E Zootecnia* **59**: 90-96.

Roemer F W, Demehri S, Omoumi P, Link T M, Kijowski R, Saarakkala S, Crema M D and Guermazi A. 2020. State of the art: Imaging of osteoarthritis—Revisited 2020. *Radiology* **296** (1): 5-21.

Roush K, Mclaughlin R M and Radlinsky M A O. 2002. Understanding the Pathophysiology of Osteoarthritis. *Veterinary Medicine* **95**: 108-112.

Runge J J, Kelly S P, Gregor T P, Kotwal S and Smith G K. 2010. Distraction Index as a Risk Factor for Osteoarthritis Associated with Hip Dysplasia in Four Large Dog Breeds. *Journal Of Small Animal Practice* **51** (5): 264-269.

Rychel J K. 2010. Diagnosis and Treatment of Osteoarthritis. *Topics In Companion Animal Medicine* **25** (1): 20-25.

Sachin P D, Thanoch M and Singh N P. 2017. Ashwagandha (*Withania Somnifera*) a Rasayna in Ayurveda and Benefits of its use than other performance enhancing substances in Sports Medicine–A Review Article. *International Journal of Physical Education and Sports* **2** (12): 16-24.

Sandell L J. 2012. Etiology Of Osteoarthritis: Genetics and Synovial Joint Development. *Nature Reviews Rheumatology* **8** (2): 77-89.

Sanderson S L. 2012. The Epidemic of Canine Obesity and its Role in Osteoarthritis. *Israel Journal of Veterinary Medicine* **67** (4): 195-202.

Sanghi D, Avasthi S, Srivastava R N. and Singh A. 2009. Nutritional Factors and Osteoarthritis: A review article. *Internet Journal of Medical Update* **4** (1): 42-53.

Scott H and Witte P. 2011. Investigation of Lameness in Dogs: 1. Forelimb. *In Practice* **33** (1): 20-27.

Scott H. 2007. Current Medical Therapies for Canine and Feline Osteoarthritis. *Veterinary Focus* **17** (3): 18-23.

Shahid M, Manchi G, Brunnberg L and Raila J. 2018. Use of proteomic analysis to determine the protein constituents of synovial fluid samples from the stifle joints of dogs with and without osteoarthritis secondary to cranial cruciate ligament rupture. *American journal of veterinary research* **79** (4): 397-403.

Sharma R, Chauhan A and Chaudhary P. 2020. Withania Somnifera: Therapeutic Uses and Phytochemical Constituents. *International Medico-Legal Reporter Journal* **3** (1): 72-77.

Sherry K. 2014. Cytology of Synovial Fluid. *Manual of Diagnostic Cytology of the Dog and Cat*. 111-125.

Siegal Mordecai (Ed.; 1995). UC Davis School of Veterinary Medicine Book of the Dogs; Chapter 5, "Geriatrics", By Aldrich, Janet. Harper Collins.

Singh A P, Chandak S, Agarwal A, Malhotra, A, Jain, A and Khan A A. 2021. Utility of High-Resolution Sonography for Evaluation of Knee Joint Pathologies as a Screening Tool. *Journal of Diagnostic Medical Sonography* **37** (6): 556-567.

Smith G K, Mayhew P D, Kapatkin A S, Mckelvie P J, Shofer F S and Gregor T P. 2006. Evaluation Of Risk Factors for Degenerative Joint Disease associated with Hip Dysplasia in German Shepherd Dogs, Golden Retrievers, Labrador Retrievers, and Rottweilers. *Journal of the American Veterinary Medical Association* **219** (12): 1719-1724.

Stabile M, Samarelli R, Trerotoli P, Fracassi L, Lacitignola L, Crovace A and Staffieri F. 2019. Evaluation of the effects of undenatured type II collagen (UC-II) as compared to robenacoxib on the mobility impairment induced by osteoarthritis in dogs. *Veterinary sciences* **6** (3): 1-11.

Steel C M. 2008. Equine Synovial Fluid Analysis. *Veterinary Clinics of North America: Equine Practice* **24** (2): 437–454.

Sugiuchi H, Ando Y, Manabe M, Nakamura E, Mizuta H, Nagata S and Okabe H. 2015. Measurement of total and differential white blood cell counts in synovial fluid by means of an automated haematology analyser. *Journal of Laboratory and Clinical Medicine* **146** (1): 36–42.

Suheb S, Mahesh V and Nagaraja B N. 2022. Successful Management of Stifle Joint Osteoarthritis in a Police Dog with Pentosan Polysulfated Sodium: A Case Report. *International Journal of Research in Engineering and Science* **10** (3): 10-13.

Syrcle J. 2017. Hip Dysplasia: Clinical Signs and Physical Examination Findings. *Veterinary Clinics: Small Animal Practice* **47** (4): 769-775.

Taguchi T, Koh R, Takawira C, Rademacher N, Gilad GM, Aronson RD and Lopez MJ. 2018. Agmatine for Pain Management in Dogs with Coxofemoral Joint Osteoarthritis: A Pilot Study. *Frontiers In Veterinary Science* **5** (18): 311-331.

Tandon N and Yadav SS. 2020. Safety and Clinical Effectiveness of Withania Somnifera (Linn.) Dunal Root in Human Ailments. *Journal Of Ethnopharmacology* **255**: 1-55.

Triakoso N. Incidence rate and Small Animal Geriatric Diseases in Veterinary Teaching Hospital Airlangga University Surabaya on 2010-2011. 2013. Conference: International Seminar: The Role of Veterinary Science to Support Millenium Development Goals; and the 12th Asian Association of Veterinary School Congress. 1-5

Verrico C D, Wesson S, Konduri V, Hofferek C J, Vazquez-Perez J, Blair E and Halpert M M. 2020. A Randomized, Double-Blind, Placebo-Controlled Study of Daily Cannabidiol for The Treatment of Canine Osteoarthritis Pain. *Pain* **161** (9): 2191-2202.

Wenham C Y J, Grainger A J and Conaghan P G. 2014. The role of imaging modalities in the diagnosis, differential diagnosis and clinical assessment of peripheral joint osteoarthritis. *Osteoarthritis and Cartilage* **22** (10): 1692-1702.

Whitehair J G, Vasseur P B and Willits N H. 1993. Epidemiology of cranial cruciate ligament rupture in dogs. *Journal of the American Veterinary Medical Association* **203**(7): 1016-1019.

Whittem T, Richards L, Alexander J, Beck C, Knight C, Milne M, Rockman M, Saunders R. and Tyrrell D. 2021. A randomised controlled masked clinical trial of two treatments for osteoarthritis in dogs. *Australian Veterinary Journal* **99** (7): 267-272.

Witte P G. 2019. Hip Dysplasia-Understanding the Disease. *Companion Animal* **24** (2): 77-81.

Wood R D and Gibson T. 2020. Synovial Fluid Analysis of The Dog and Cat. *Veterinary Cytology* .727-735.

Wright A, Amodie D, Cernicchiaro N, Lascelles B and Pavlock A. 2019. Pvm1 Diagnosis and Treatment Rates of Osteoarthritis in Dogs Using a Health Risk Assessment (Hra) Or Health Questionairre for Osteoarthritis in General Veterinary Practice. *Value In Health* **22**: 387-389.

Xu Q R, Dong Y H, Le Chen S, De Bao C and Du H. 2009. Degeneration of Normal Articular Cartilage Induced by Late Phase Osteoarthritic Synovial Fluid in Beagle Dogs. *Tissue and Cell* **41**(1): 13–22.

APPENDIX-A

Proforma for the examination of Osteoarthritis in Geriatric Dogs

A. General Information: Group: _____

1) Name of Owner: _____

2) Address: _____

3) Contact No.: _____

B. Details of Dog:

1) Age: _____

2) Sex: _____

3) Breed: _____

C. Anamnesis:

1) Lameness, able to bare weight on joints and pain.

2) General condition: Normal/thin/emaciated/obese.

3) Checking the joint mobility and functioning of nerves at the joints.

D. Clinical Examination:

1) Body weight (kg): _____

2) Rectal Temperature (°F): _____

3) Mucosal membranes: Pinkish/Pale/congested_____

4) Respiratory Rate:_____/ minute

5) Heart Rate:_____/ minute

6) Weight bearing, pain scores, joint mobility and lameness grading_____

E. Radiography: Plain

F. Ultrasonography (B-mode)

G. CT scanning

LABORATORY EXAMINATION

1.Hematological parameters:

SL.NO	Parameter	0th Day	15th Day	30th Day
1	Hb (g/dl)			
2	TEC ($\times 10^6$ /µl)			
3	TLC ($\times 10^3$ /µl)			
4	Neutrophils (%)			
5	Lymphocytes (%)			
6	Monocytes (%)			
7	Eosinophils (%)			

2. Serum Biochemical parameters:

Sl. No.	Parameter	0th Day	15th Day	30th Day
1	Creatinine (mg/dL)			
2	ALT (IU/L)			
3	AST (IU/L)			
4	C- Reactive protein (g/dL)			

3. Synovial fluid analysis

Sl. No	**Parameter**	**0th Day**	**15th Day**	**30th Day**
1.	Volume (mL)			
2	Colour			
3	Viscosity			
4	Total protein (g/dL)			
5	TNCC (cells/mL)			
6	Mononuclear cells (%)			
7	Neutrophils (%)			

APPENDIX- B

ESTIMATION OF SYNOVIAL FLUID PARAMETERS

Method: Automated serum Analyser

Procedure:

- Osteoarthritis dogs were kept in lateral recumbency with the affected joints on top after shaving the arthrocentesis sites.
- Scrub the area with chlorhexidine or povidine-iodine.
- By using 22G or 23G in large dogs and 25G in smaller dogs, apply a little amount of negative pressure and aspirate the fluid from the joint.
- Place the collected fluid in EDTA vails for further evaluation
- Add 1-2 drops of hyaluronidase to the synovial fluid collected to remove the viscosity of synovial fluid and keep undisturbed for 1-2 minutes
- Place the cleared fluid in fluid slot and place the kit for estimation of total protein, TNCC, monocyte and neutrophil in the kit slot and run the machine.
- The results will be displayed on the computer attached to the machine
- The total protein will be estimated in g/dL, TNCC in cells/mL, monocytes and neutrophils in %.

APPENDIX- C

SYNOVIAL FLUID STAINING

Method: Giemsa staining

Procedure:

- After collection of synovial fluid, immediately place a drop of fluid on the clean glass slide.
- Prepare thick smears with the help of another glass slide.
- Allow the smear to dry.
- Fix the smear with methanol.
- Place Giemsa stain on the glass slide and leave for 30 minutes.
- Wash off the stain and air dry the smear.
- Put a drop of oil and examine under 100x

APPENDIX- D

CT SCANNING

Procedure:

- Before proceeding to CT, animal should be fasted for 6 and anesthesia was given to dog, to calm the animal during procedure. Dogs were anesthetized with Propofol@4 mg/kg.
- Look the response of the animal and when animal enters into unconscious state, place the animal on CT machine bed.
- The animals should be place in dorso-lateral position to know the osteoarthritis changes of hip joint.
- CT was performed on twin beam dual energy spectral CT scanner which exposes 70KvP of energy from the X-ray tubes, which helps in accelerating the electrons from cathode to anode and 600 mA with a focal spot of 0.4x0.5 and with the speed of 720 mm/s^8 with turbo flash and the exposure time was 20 sec.

The computer attached to it produces the 3D images of osteoarthritic hip joint

APPENDIX- E

APPLICATION OF DIATHERMY

Method: Application of Diathermy

Procedure:

- Properly shave the affected area with savlon or trim the area where diathermy should be applied (note: do not apply spirit on the area).
- After shaving the area, allow it for air dry.
- After complete drying of the shaved area, put the diathermy conducting gel to the treatment probe and place the treatment probe on the area where pain is prominent and apply the earth probe towards the lower side of the joint.
- Run the diathermy unit in continuous mode, where it adjusts the frequencies automatically.
- Based on intensity of pain, apply the diathermy for a minimum of 30 minutes and can extend to 45 min, but not more than, if so check for any skin burns.
- After completion of procedure, remove the probes, wipe off the area with any clean towel.
- Repeat the procedures for every alternate day, if pain is less severe and repeat 2-3 times a day for 4 weeks if pain is very severe.

APPENDIX- F

APPLICATION OF TENS (Transcutaneous Electric Nerve Stimulator)

Method: Application of TENS

Procedure:

- Before application of TENS, shave the area with savlon or trim the hair completely from that area
- After shaving, apply the TENS probes to the affected joint, one above and one below.
- If the dog experience severe pain and paraplegia, place one probe at the level of lower pelvis region and one at the level of hindlimb.
- Run the machine and select the osteoarthritis mode which is present in the TENS machine and run for 15-20 minutes
- Remove the probes
- Repeat the procedure on alternate days if pain is severe and repeat 2-3 times a day if pain is severe for 4 weeks.