OCCURRENCE OF *OSTEODYSTROPHIA FIBROSA* IN HORSES, KNOWLEDGE, ATTITUDE AND PRACTICES OF HORSE GROOMS IN THREE SELECTED LOCAL GOVERNMENT AREAS, KADUNA STATE, NIGERIA

BY

RUKAYA ALTINE NASIRU

DEPARTMENT OF VETERINARY MEDICINE,

FACULTY OF VETERINARY MEDICINE,

AHMADU BELLO UNIVERSITY,

ZARIA

APRIL, 2021

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RUKAYA ALTINE NASIRU

A DISSERTATION SUBMITTED TO THE SCHOOL OF POSTGRADUATE STUDIES, AHMADU BELLO UNIVERSITY, ZARIA, NIGERIA IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF THE DEGREE OF MASTER OF SCIENCE IN EQUINE MEDICINE

DEPARTMENT OF VETERINARY MEDICINE,

FACULTY OF VETERINARY MEDICINE,

AHMADU BELLO UNIVERSITY,

ZARIA.

APRIL 2021

DECLARATION

I declare that the work reported in this dissertation, titled '**Occurrence of *Osteodystrophia Fibrosa* in horses, knowledge, attitude and practices of horse grooms in threeselected Local Government Areas, Kaduna State, Nigeria"** was carried out by me in the Department of Veterinary Medicine, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria, Nigeria. The information derived from literature has been duly acknowledged in the text and in the list of references provided. No part of this dissertation has been presented for another degree or diploma at any university.

Rukaya Altine NASIRU ……. ……

(Name of Student) Signature Date

CERTIFICATION

This dissertation, entitled" **Occurrence of *Osteodystrophia Fibrosa* in horses, knowledge, attitude and practices of horse grooms in threeselected local government areas, Kaduna state, Nigeria"** carried out by Rukaya Altine NASIRU meets the regulations governing the award of the degree of Master of Science of Ahmadu Bello University, Zaria, and is approved for its contribution to knowledge and literary presentation.

Dr. S.N.A. Sai'du ……. ……..

Chairman, Supervisory Committee Signature Date

Prof K.B. Kadima …… ..……

Member, Supervisory Committee Signature Date

Prof. P.A. Abdu

Head Department of Veterinary Medicine ....

Signature Date

Prof. Sani .A. Abdullahi …..... .....

The Dean, Signature Date

School of Postgraduate Studies

DEDICATION

This dissertation is dedicated to God Almighty and Captain Abdulrahman Jumah Hassan.

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ABSTRACT

Osteodystrophia Fibrosa also known as bran disease, nutritional Secondary Hyperparathyroidism (NHS) is a is a dietary calcium: phosphorus imbalance. The aim of this study was to determine the occurrence of *Osteodystrophia fibrosa* along with knowledge, attitude and practices of horse grooms in horses. The occurrence of *Osteodystrophia fibrosa* (bran disease) was studied in three selected LGAs of Kaduna State, Nigeria. The three LGAs were Igabi, Zaria and Sabon Gari LGAs. A total of 59 horses of both sexes consisting of 3 breeds (Argentine, West African Barb and Sudanese) were used. Purposive and convenience sampling methods were used during the study. The clinical signsand clinical parameters (Respiratory, pulse rate and rectal temperature)were evaluated. The serum biochemical values, haematological values, faecal calcium and phosphorous levels were also evaluated. The feed fed to the horses were also analyzed for oxalate and phytate levels. The rate of occurrence of *Osteodystrophia fibrosa*was 74.6%.The knowledge, attitude and practice of the horse grooms were also evaluated. Theclinical signs of *Osteodystrophia fibrosa* observed were enlarged facial bone, poor body condition score and lameness. The vital parameters (RPT) were within normal values. The haematological values of horses with *Osteodystrophia fibrosa*were all within normal values. There was no statistically significant difference ($p>0.05$) between respiratory rate, pulse rate, rectal temperature and haematological valuesof equine *Osteodystrophia fibrosa* and non *Osteodystrophia fibrosa*horses. The serum calcium and phosphorus levels equine *Osteodystrophia fibrosa* horses were 2.71±0.03 mmol/L and 1.26±0.08mmol/L respectively.The parathyroid hormone of horses with and without *Osteodystrophia fibrosa*was found to be 93.01±20.44 pg/ml and 38.9±3.50 pg/ml respectively. The values were statistically significant($p<0.05$)between the clinically and non-clinically diseased

horses.Similarly, the oxalate and phytate levels in the horses' feeds 0.02 and 0.51m/100kg respectively. The faecal calcium level of the horses from Igabi LGA had the highest values of 75.89± 6.55mg/kg for the non-clinically diseased horses and 82.85±4.17mg/kg for the clinically diseased horses and that the faecal phosphorus values for the clinically diseased horses were also highest in Igabi LGA with values of 2801.76±212.51mg/kg. The high level of calcium and phosphorus in the faeces of these horses could be as a result of high oxalate and phytate in the feeds of the horses. The knowledge, attitude and practice of the horse grooms showed they had a good understanding of the condition as85.7% of the grooms knew the condition, 90.5% had good attitude while 71.4% had good practice towards the condition. The study has shown that *Osteodystrophia fibrosa* exists in the study area and that the nature of the horses' feeds could have something to do with the occurrence of the condition. The study has further shown that there is no age predisposition for the occurrence of the condition while the clinical and haematological valuesof the affected horses do not seem to be adversely affected by the condition.it was suggested more horse feeds should be examined to determine their oxalate and phytate levels in Nigeria.

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LIST OF ABBREVIATIONS, SYMBOLS AND ACRONYMS

<	Less than
>	Greater than
≤	Less than or equal to
°C	Degree Celsius
µg/µl	Microgram per microliter
µg/ml	Microgram per milliliter
µL	Microlitre
µm	Micrometre
C	Clinically diseased
Ca	Calcium,
EDTA	Ethylene diamine tetraacetic acid
g	Gram
g/ml	Gram per milliliter
GRAN	Granulocytes
Hg	Haemoglobin
LGAs	Local Government Areas
LYM	Lymphocytes
MCH	Mean corpuscular haemoglobin
MCHC	Mean corpuscular haemoglobin concentration
MCV	Mean corpuscular volume
mg/kg	Milligram per kilogram
mL	Millilitre

Nc	Non-clinically diseased,
NEU	Neutrophils
NHS	Nutritional Secondary Hyperparathyroidism
PCV	Packed cell volume
Ph	Phosphorous
PTH	Parathyroid Hormone
RBC	Red blood cell
SEM	Standard error of mean
WBC	White blood cell
W/A Barb	West African Barb

CHAPTER ONE

1.0 INTRODUCTION

1.1 Background

Osteodystrophia fibrosa also known as bran disease was first described in 400 A.D (Stewart *et al.,* 2010). It is known as Nutritional Secondary Hyperparathyroidism (NHS) which is considered a rare disease (Stewart *et al.,* 2010). It has been reported in horses in Australia (Groenendyk and Seawright,1974; Caple *et al.*,1982; McKenzie, 1982), Canada (Menard *et al.*,1979), Ethiopia (Clarke *et al.*,1996), Hawaii (Gusman, 2004), Honk Kong (Mason *et al.,* 1988), Japan (Chiba *et al.*,2000; Sasaki *et al.,* 2005), Netherlands (Benders *et al.*,2001), the Philippines (Gonzalez and Villegas, 1928), South Africa (Ronen *et al.*,1992), Spain (Estepa *et al.*,2006), and the United States of America (Joyce *et al.*,1971; David*et al.*,1997). *Osteodystrophia fibrosa* have been treated by field veterinarian but cases are not documented in nigeria.

The condition has been reported to be induced by persistent imbalance of calcium and phosphorus in the diet of horses (Krook and Lowe, 1964; Krook, 1968; Joyce *et al.*,1971; Schryver *et al.*,1971; Argenzio *et al.*,1974; Walthall and McKenzie, 1976; Capen *et al.*, 1982; Mason *et al.*,1988) and grazing horses on very low-quality pastures (Groenendyk and Seawright, 1974; Hoskin and Gee, 2004). Several tropical grasses such as blue grass (*Setaria anceps*), camolo grass (*Cenchrus*sp), guinea grass (*Panicum maximum* var. *trichoglume*), kikuyu grass (*Pennisetum clandestinum*), pangola grass (*Digitaria eriantha*, formerly *D. decumbens*), and some *Brachiaria* species contain oxalic acid at levels that significantly interfere with calcium and other cations utilization by horses (Franceschi and

Horner, 1980; Blayney *et al.*,1981a; Gartner *et al.*,1981; McKenzie and Schultz, 1983; Hintz *et al.*,1984; McKezie *et al.*,1994; Cheeke, 1995; Marais, 2001; Allan *et al.*,2007).

Secondary hyperparathyroidism is exhibited by an excessive production of parathyroid hormone (PTH) due to decreased serum ionized calcium or magnesium (Stewart *et al.*, 2010). Regardless of its genesis, persistent hypocalcemia stimulates hyperactivity of the parathyroid glands and a secondary hyperparathyroidism is induced (Krook, 1968). The most common cause of secondary hyperparathyroidism is dietary calcium:phosphorus imbalance and is referred to as Nutritional Secondary Hyperparathyroidism. A diet high in phosphorus, low in calcium or with a calcium:phosphorus ratio of less than 1:1 will cause Nutritional Secondary Hyperparathyroidism notwithstanding, if dietary calcium is supplied at optimal or even excessive levels (Krook, 1968). In addition, high phosphorus diets depress calcium and magnesium digestibility and availability (Schryver *et al.*,1971; Van Doorn *et al.*,2004) especially diet high in phytates such as dusa or wheat bran.

Horses of all ages can come down with the condition, although those between 2 and 8 months of age are more vulnerable and the lesions are more severe compared to those seen in adult horses (Krook and Lowe, 1964). However, increasing age is not considered a factor of resistance. Conditions becoming predisposition factors through host factors such as breed or sex are not reported, although high levels of calcium required during pregnancy may influence the development and course of the disease in mares (Krook and Lowe, 1964; Menard *et al.*,1979; Stewart *et al.*,2010). The prevalence and incidence of the condition may vary from 1 to 100% and it is also closely related to diet composition (Krook and Lowe, 1964).

Clinical findings of *Osteodystrophia fibrosa* include lameness, fracture, growth retardation, poor body condition, enlarged and deformed bones of the face, narrowed nasal passages and loss of lamina dura surrounding the molars (Ospina *et al.,* 2014). Radiographic analysis may show decreased bone and teeth density (Ronen *et al.*,1992; Rosol and Capen, 1997; Toribio, 2010; Toribio, 2011).

Though the condition is poorly documented in international and national literatures, some reports indicate that diagnosis of the condition could be achieved through history of grazing on kikuyu grass for long periods of time without any supplement rich in calcium and balance of phosphorus (Ospina *et al.,* 2014). Susceptibility of equine species due to reduced capacity to metabolize calcium oxalates, an increased risk to develop the condition in pregnant females due to its reproductive condition have also been considered to aid in the diagnosis of the condition. The definitive diagnosis of *Osteodystrophia fibrosa* was established by a correlation of the nutritional and management data, clinical evaluation, gross and histopathological analyses (Ospina *et al.,* 2014).

Preventive measures for *Osteodystrophia fibrosa* may be to avoid the use of pastures high in oxalate levels, though this may not always be practicable, especially in certain areas where the soil is rich in such compound. In such situations, supplementation with calcium may be necessary. Animals grazing pastures high in oxalates may require about 20mg/kg calcium and 10mg/kg phosphorus on a daily basis in their diet (Toribio, 2010).

1.2 Statement of the Research Problems

Horses in Nigeria are used for polo, racing, leisure, traditional ceremonies and local sugar production (Ehizibolo *et al.*,2011; Baba, 2016). In many situations, horses are also confronted with diseases that adversely affect their uses (Mshelia *et al.*,2010; 2012; Baba, 2016; 2019). Some of these diseases include *Osteodystrophia fibrosa*, Equine influenza, African horse sickness, Equine piroplasmosis, brucellosis, Ulcerative lymphangitis, and colic (Mshelia *et al.*,2010; Edeh, 2018; Baba, 2019).

The population of horses in Nigeria is apparently on the increase with increase in their uses (Ehizibolo *et al.,* 2011). This indicates the possibility for more cases of Secondary hyperparathyroidism, should the condition exist in Nigeria. The apparent paucity of the report of the condition by horse owners and even their grooms could be as a result of the seeming low number of practicing Veterinarians working in the equine industry, as Baba (2016; 2019) and Edeh, (2018) reported horse owners and grooms putting their hands-on traditional medicine practicesto treat their horses on ailment like ulcerative lymphangitis. The poor disease reporting along with poor surveillance network in the country could also add to the seeming poor records on the disease in Nigeria. Not only that, certain horse diseases require specific skills in their diagnosis. For example, there is the need to evaluate feeds and get very good history on the soil-plant mineral contents, horses' management practices and the history of concentrates feeding of horses by the owners. The poor laboratory back-up for the diagnosis of animal diseases could also support the claim of none existence of the disease in Nigeria.

The poor knowledge, attitude and practices of grooms on horse diseases could also contribute in their not being able to appreciate the condition being in their horses, let alone take appropriate steps to look for treatment against the condition.

1.3 Justification of the Research

There is increasing interest in polo, racing, traditional festivals and even leisure riding of horses in Nigeria. The increasing use of horses for equitation as a result of increasing security challenges has also made it necessary to use very healthy horses. On a more routine basis, especially in traversing areas where vehicles could ordinarily not be used horses are conveniently used. Horses are being imported to meet the purpose and requirements of these activities. To meet up with these needs, breeding horses is advocated. However, in situations where poor nutrition through lack of proper knowledge on what to feed these horses on, especially foals could predispose such horses to *Osteodystrophia fibrosa*. Horse breeding is a lucrative business as it is providing opportunities for actors to realize income and at the same time distribute foals that could be used for polo, racing, traditional durbar among others.

There is also the need to document the actual position of the condition in horses in Nigeria as doing so will allow for proper advice to horse owners on how to manage their horses so as achieve the desired goals of keeping them. There is also the need to document the clinical features of the condition along with its associated haematological parameters so as to enable equine clinicians to effectively diagnose and provide definite management for the condition in the Nigerian situation.

Horse keeping in Nigeria is becoming a big business, especially through racing, week-end riding by youths and for marriage processions. If this condition is present in these horses, they may not be available for these activities. There is also the need to examine horse feeds with the aim of assessing their nutritive value as well as their oxalate and phytate levels.

1.4 Aim and Objectives of the Study

1.4.1 Aim of the Study

To determine the occurrence of *Osteodystrophia fibrosa* along with knowledge, attitude and practices of horse grooms in horses in 3 selected Local Government Areas in Kaduna State, Nigeria.

1.4.2 Objectives of the Study

1. To determine occurrence of*Osteodystrophia fibrosa* in horses in 3 selected Local Government Areas of Kaduna State, Nigeria.
2. To determine the level of some serum biochemical and haematological parameters in horses with *Osteodystrophia fibrosa*inthe 3 selected Local Government Areas of Kaduna State.
3. To determine the levels of calcium and phosphorus in the faeces as well as the levels of phytate and oxalate in feeds commonly fed to horses in the 3 selected Local Government Areas of Kaduna State.
4. To determine the knowledge, attitude and practices of horse grooms with regard to *Osteodystrophia fibrosa*in the 3 selected Local Government Areas of Kaduna State.

1.4.3Research Questions

1. Were there horses*Osteodystrophia fibrosa* in the 3 selected Local Government Areas in Kaduna State, Nigeria and if so what were the clinical features of the condition in these horses?

2 What were some of the serum biochemical and heamatological parameters of horses with *Osteodystrophia fibrosa* in the 3 selected Local Government Areas in Kaduna state, Nigeria?

3 What were the levels of calcium and phosphorus in faeces, as well as phytate and oxalate in horse feeds in the 3 selected Local Government Areas in Kaduna state, Nigeria?

4 What were the knowledge, attitude and practices of horse grooms with regards to *Osteodystrophia fibrosa*in the selected Local Government Areas in Kaduna State, Nigeria?

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Definition

Osteodystrophia fibrosa is a disorder of the calcium metabolism in animals and humans characterized by bone resorption, connective tissue proliferation, poor mineralization and immature bone appearance (Thompson 2007; Toribio,2010).It is considered a rare condition. It is also referred to as "bran disease", "bighead", "Miller's disease", osteitis fibrosa, swollen face disease, and equine osteoporosis.Nutritional secondary hyperparathyroidism (NSH). (Anguiano, 2016).

2.2 Geographical Distribution

Nutritional secondary hyperparathyroidism (NHS) has been reported in horses in Australia (Groenendyk and Seawright 1974; McKenzie, 1982; Caple *et al.,* 1982), Canada (Menard *et al.,* 1979), Ethiopia (Clarke *et al.,* 1996), Hawaii (Gusman 2004), Hong Kong (Mason *et al.,* 1988), Japan (Chiba *et al.,* 2000; Sasaki *et al.,* 2005), Netherlands (Benders *et al.,* 2001), New Zealand (Hoskin and Gee 2004), Panama (Williams *et al.,* 1991); the Philippines (Gonzalez and Villegas 1928), South Africa (Ronen *et al.,* 1992), Spain (Estepa *et al.,* 2006) and the United States of America (USA) (Joyce *et al.,* 1971; David *et al.,* 1997). There does not seem to be any report of the condition in Nigerian horses as search in national and international literature on the condition in Nigeria did not yield any result.

2.3 Aetiology

Secondary hyperparathyroidism known as nutritional secondary hyperparathyroidism (NSH)is a dietary calcium: Phosphorus imbalance. A diet high in phosphorus, low in calcium or that with calcium: phosphorus ratio of less than 1:1 will cause NSH regardless of whether dietary calcium is supplied at optimal or even in excessive levels (Krook, 1968). In addition, high phosphorus diets depress calcium and magnesium digestibility (Schryver *et al.,* 1971; van Doorn *et al.,* 2004). This includes diets high in grain or bran. Grains and bran are high in phosphorus and low in calcium (Lacitignola *et al.,* 2018) They also contain chemicals known as phytates, which bind to calcium in the gut, forming calcium-phytatecompounds which cannot be absorbed by the horse ((Lacitignola *et al.,* 2018).

Certain grasses have high oxalates content that bind, calcium to form calcium-oxalate (Ca[COO]2) which is insoluble at the alkaline pH in the horse's intestinal tract (Stewart *et al.,*2010). Several tropical grasses such as blue grass (*Setaria anceps*), camolo grass (*Cenchrus*sp.), guinea grass (*Panicum maximum* var. *trichoglume*), kikuyu grass (*Pennisetum clandestinum*), pangola grass (*Digitaria eriantha*, formerly *D. decumbens*), and some *Brachiaria* species contain oxalic acid at levels that significantly interfere with calcium and other cations utilization by horses (Franceschi and Horner, 1980; Blayney *et al.,*1981a; Gartner *et al.,*1981; McKenzie and Schultz, 1983; Hintz *et al.,*1984; McKezie *et al.,*1994; Cheeke, 1995; Marais, 2001; Allan *et al.,*2007; NSW DPI, 2008).

In the horse, most ingested oxalate is utilized as a carbon source by bacteria in the caecum. The bulk of dietary calcium is absorbed in the upper small intestine of the horse (Schryver *et al.,* 1970). Therefore, calcium freed by bacterial action in the lower alimentary tract

would be unavailable for absorption (McKenzie *et al.,* 1981). Grasses with more than 0.5% oxalate or calcium: oxalate ratios of less than 0.5 result in a negative calcium balance and are capable of inducing hypocalcaemia in horses (Walthall and McKenzie 1976; Blayney *et al.,* 1981a,b; McKenzie 1985, 1988; McKenzie *et al.,* 1981; McKenzie and Schultz 1983).

An equine diet with 1% oxalate content reduces calcium absorption by 66% and increases faecal calcium excretion (Schwartzman *et al.,* 1978). Oxalate content of grasses under conditions of rapid growth may exceed 6% which is enough to bind the entire calcium content of the grass (Marais 1990).

Horses have species differences in their ability to utilize the calcium bound in calcium oxalate (Stewart *et al.,*2010). They have unique features with regard to calcium metabolism including: hightotal serum and ionized calcium concentrations compared with other animal species as they have poorly regulated intestinal absorption of calcium; high urinary fractional clearance of calcium, low serum concentrations of vitamin D, a high calcium set-point (Toribio *et al.,* 2003) and complete inability to utilize calcium bound to oxalic acid in grasses (Blayney *et al.,* 1981; McKenzie and Schultz 1983).

Parathyroid Hormone (PTH) is responsible for the minute-to-minute regulation of extracellular calcium (Ca^{2+}) concentrations. The relationship between serum Ca^{2+} concentrations and PTH is inverse and sigmoidal in the horse (Aguilera- Tejero *et al.,* 1998; Toribio *et al.,* 2001). This enables the parathyroid gland to respond rapidly to negligible changes in Ca^{2+} concentrations. High phosphorus and low calcium diets induce parathyroid cell hyperplasia (Krook and Lowe 1964) and increased serum PTH concentrations in the horse (Benders *et al.,* 2001). Parathyroid Hormone stimulates calcitriol synthesis and

Ca^{2+}reabsorption in the kidney and its uptake in the intestine; osteoclastic resorption in the bones and inhibition of phosphate (Pi) absorption in the renal tubules.

Interaction of PTH with PTH receptors in the osteoblasts increases the synthesis and release of receptor activators and macrophage colony-stimulating factor (M-CSF) both of which are essential regulators of osteoclast function (Toribio, 2004). The increased synthesis and release of receptor activators and M-CSF stimulates osteoclasts, resulting in PTH-induced bone resorption (Toribio 2004). In the kidneys, PTH acts in the proximal renal tubules to induces natriuresis and diuresis and inhibits HCO_3 reabsorption. There are no PTH receptors in the intestine, however PTH indirectly increases Ca^{2}+ and decreases Pi absorption in the intestinal tract by increasing synthesis of calcitriol. Parathyroid hormone stimulates bone resorption and elevated serum calcitriol causes increased plasma phosphate (Pi) concentrations which may interact with $Ca^{2+}$to form insoluble precipitates and thereby drops plasma Ca^{2}+ concentrations. To avoid dangerous insoluble Ca - P precipitates, PTH decreases Pi reabsorption in the kidney and increases Pi elimination. The end result is decreased Pi reabsorption and increased fractional urinary clearance of Pi (Toribio 2004).

2.4 Clinical Signs

The onset of clinical signs after first grazing high oxalate pasture is 2 to 8 months, with young, pregnant and lactating horses being more susceptible (Walthall and McKenzie 1976). Horses of all ages can develop the condition but those between 2 and 8 months of age are more prone to the condition. The lesions are more severe in young horses compared to the adult (Krook and Lowe, 1964; Menard *et al.*,1979; Stewart *et al.*,2010). However, increasing age is not considered a factor of resistance (Krook and Lowe,1964).

Predisposition to the condition by host factors such as breed and sex do not seem to have been reported. The prevalence and incidence of the condition may vary from 1 to 100 % (Krook and Lowe, 1964).

There are three classical clinical manifestations of this condition and they include ill-thrift, lameness and swelling of the maxilla and mandible (Stewart *et al.,*2010). The clinical signs may present solely as a serous nasal discharge, mild swelling of the distal pasterns, facial distortion, sinusitis or upper respiratory tract noise (Freestone and Seahorn, 1993). Difficulty in passing a stomach tube may also be noticed, which could be due to dental eruption cysts in 2-, 3- and 4-years old horses. There may also be maxillary sinus disease or tooth root infection (Stewart *et al.,*2010).

The first clinical sign that may be seen is usually intermittent shifting lameness due to focal periosteal avulsion, torn or detached ligaments and tendons, or subepiphyseal microfractures (Stewart *et al.,*2010). Joint pain may result from articular cartilage erosions due to loss of underlying trabeculae (Joyce *et al.,* 1971; Brewer, 1987). Lameness is usually insidious with general tenderness of the joints or a stiff stilted gait. it varies from mild shortening of stride, especially at faster paces, to a reluctance to move, recumbency and difficulty in rising. These changes are thought to result from separation of tendon and ligament attachments, and from pain due to defective mineralization and osteoporosis which may result in weakening and bending of the bones (McKenzie, 1984). Some horses prefer to canter rather than trot while others are reluctant to move. The appetite is often normal though affected horses are often seen eating dirt.

The most severe changes occur in the skull bones, especially the maxilla, mandible and nasal bones giving the condition the expressive name of 'bighead'. The mineral content of the facial bones and mandible is replaced with increased amounts of osteoid and fibrous tissue, a process termed *Osteodystrophia fibrosa*. Changes to the maxillary and mandibular bones are bilateral but not necessarily symmetrical (Ospina *et al.*,2014).

These changes, in combination with resorption of the *laminae dura* may result in problems with mastication late in the course of the condition. However, difficulty in chewing and dental pain may be the first sign. Reduced feed intake can result in weight loss and poor body condition. In advanced cases, marked distortion of the mandible can lead to gradual separation of the upper and lower incisors so that they become completely unopposed, and the horse may be less able to masticate short grass (Ospina *et al.*,2014).

The condition typically develops slowly, therefore, horses adapt and learn to cope remarkably well (Stewart *et al.*,2010). This contrasts with the situation where incisors are rapidly taken out of occlusion, as occurs with over aggressive filing of the incisors. Mouths of NSH affected horses often have very "clean" periodontal structures (less gingivitis than the average horse) in the mandibular and maxillary dental arcades, possibly due to a reduced risk of feed trapping and gingivitis because the cheek mucosa is no longer apposed with the teeth. However, bone loss in NSH peridontal disease is a forerunner to more severe manifestations such as vertebral and long bone fractures (Whalen and Krook, 1996). In young growing horses, the skeletal calcium cannot be mobilized in the skeleton rapidly enough to maintain a normal serum calcium level and the result is rapid development of clinically detectable NSH. In the adult animal, however, the dietary deficiency can be compensated for by the parathyroids, and *osteitis fibrosa* occurs only in severe deficiency.

Therefore, NSH in the adult horse is frequently characterized by osteoporosis due to increased bone apposition and even more accelerated bone resorption. Low-grade secondary hyperparathyroidism is associated with osteoporosis and elevated serum PTH levels in adult animals (Jowsey *et al.,* 1974). In adult horses there is slight parathyroid hyperplasia, but the degree of parathyroid over activity is too low to result in the classical clinical findings (Krook, 1968; Mason *et al.,* 1988). A high incidence of vertebral compression fractures may be the only manifestation of NSH in the adult horse and Caple *et al.* (1982) speculated that much of the lameness, tendonitis and spontaneous fractures in racehorses may be due to undetected NSH.

2.5 Pathology

2.5.1 Gross pathology

The most important pathological change is osteolytic resorption of the outer circumferential lamellae of long bones (Gries, 1966). There is no definite site of predilection for the resorption of the outer circumferential lamellae. Any bone may be affected and temporary healing may explain the shifting lameness (Krook, 1968).

2.5.2 Histopathology

The mandible bones would have severely reduced bone mass, multiple irregular bone spicules surrounded by abundant connective tissue, microfracture of spicules, osteoid formation and hemorrhages. Masson´s trichrome staining confirmed that the bone tissue would be replaced by conjunctive fibrous tissue distributed among bone spicules and trabeculae, various degrees of bone mineralization (various shades of bone matrix), and multiples foci of bone resorption by osteoclasts(Ospina *et al.*,2014). Parathyroid gland

would have mild hyperplasia and hypertrophy of parathyroid chief cells with severe intracytoplasmic vacuolar changes and increased cytoplasmic to nucleus ratio. Some chief cells also would have accumulation of brown material of unknown origin. The kidney would have slight microcirculatory changes. The liver severe thickening of Glisson´s capsule with multiples nodules of plasma cells macrophages, lymphocytes and giant cells under the capsule, mild congestion and disorganization of hepatic cords, atrophy of hepatocytes, and mild fibrosis in the portal area, moderate hyperplasia of biliary ducts and mild lymphoplasmacytic infiltrate foci. The small intestine will contain mild luminal mucus, mild intestinal villus atrophy, desquamated epithelial cells, distended lymphatic vessels(Ospina *et al.*,2014).

2.5.3 Clinical pathology

Dietary oxalate produces a net loss of calcium and phosphorus primarily through the faecal route in horses (Swartzmann *et al.,* 1978). There are no consistent abnormalities in serum calcium, phosphorus, magnesium or alkaline phosphatase levels, but faecal calcium and faecal calcium: phosphorus ratios may be increased (Krook and Lowe, 1964). The plasma calcium concentration is often normal even though the calcium: phosphorus ratio is disturbed because the parathyroid gland functions to maintain ionized calcium concentration. Serum alkaline phosphatase may be increased in some horses (Stewart *et al.*,2010). Common laboratory findings are normocalcaemia, normophosphataemia to hyperphosphataemia and increased fractional excretion of phosphorus (Stewart *et al.*,2010). Fractional excretion of calcium does not reflect calcium balance because $CaCO_3$ crystals are formed in the bladder. The urinary fractional clearance of calcium and phosphorus have been proposed as methods to estimate calcium and phosphorus intake, but interpretation

can be difficult because horses eliminate large amounts of calcium in their urine (Brewer, 1987).

2.6 Diagnosis

History, physical examination, clinical signs, clinical pathologic data and diet analysis can be used as a means of diagnosis for NSH (Mason and Watkins, 1988; Freestone and Seahorn, 1993).

The definitive diagnosis is obtained by measuring the serum parathyroid hormone level using the human radioimmunoassay that has been validated for the horse (Benders *et al.*, 2001).

2.6.1 Differential diagnoses

Some differentials for *Osteodystrophia fibrosa* are Osteomalacia, Osteoporosis, Lameness and Actinomycosis.

2.7 Treatment

Horses affected can be treated by correcting their dietary imbalance. The strategy to prevent or treat 'Big Head' depends on the cause and some briefsare given below.

I. Imbalanced diet: The diet should be balanced to meet with the horse's calcium and phosphorus requirements as to have a calcium to phosphorus ratio of at least 1:1.Ideally ratio 2:1. Phosphorus should not be supplied in excess. Di-calcium phosphate and limestone can be added to the feed to increase the calcium content of thefeeds and supplements (www.hygain.com)

II. Oxalated Pastures: Horses should graze grasses that are poor in oxalates where possible. If they graze on high-oxalate grasses, the growth of a legume component in the pasture, such as lucerne, is highly recommended due to their high calcium content. Horses grazing oxalate-rich grass should be supplemented with additional calcium and phosphorus. Those with high calcium requirements such as gestating or lactating mares and growing horses require even higher levels of calcium to be added to their diet (www.hygain.com).

III. Tiludronate, a bisphosphonate medication licensed for "the control of clinical signs associated with navicular syndrome," appears to have a new use for treatment of big head disease in horses. This medication helps horses with various bone fragility disorders by binding to and inhibiting the action of osteoclasts, which are cells that break down bone. When administered at the dose licensed for horses with navicular disease, horses and ponies with NSH benefit from this medication but only if given in conjunction with a balanced diet (Lacitignola *et al.,* 2018).

2.8 Prevention and Control

Daily requirements of Calcium and Phosphorus for different types of average (500kg) horses are important. For calcium, maintenance level requires 20-25g, while for lactating mare right after birth the highest calcium requirement is 70-85g and 36-40g for young growing horses. The phosphorous requirement for maintenance, lactating mare and young growing horses are 14-18g,39-41g and 20-22g respectively (Lacitignola *et al.,* 2018).

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 Study Area

The study was carried out in three (3) selected Local Government Areas of Kaduna State (fig 3.1).The state is located within the geographical coordinates of latitude 10°20'N and longitude 7°45'E. The State has 23 Local Government Areas (LGAs) (Sambo, 2015). It has three senatorial districts (SD) namely North, Central and South SDs (Fig. 3.1). The occupation of most of the people in the State includes farming of crops, sugar cane and brown sugar production. There are no less than 2500 horses in the state (Aliyu, 2014).

3.2 Ethical Clearance

Approval for animal use was sought and obtained from the Ahmadu Bello University, Zaria Committee on Animal Use and Care with approval number as ABUCAUC/2020/73.

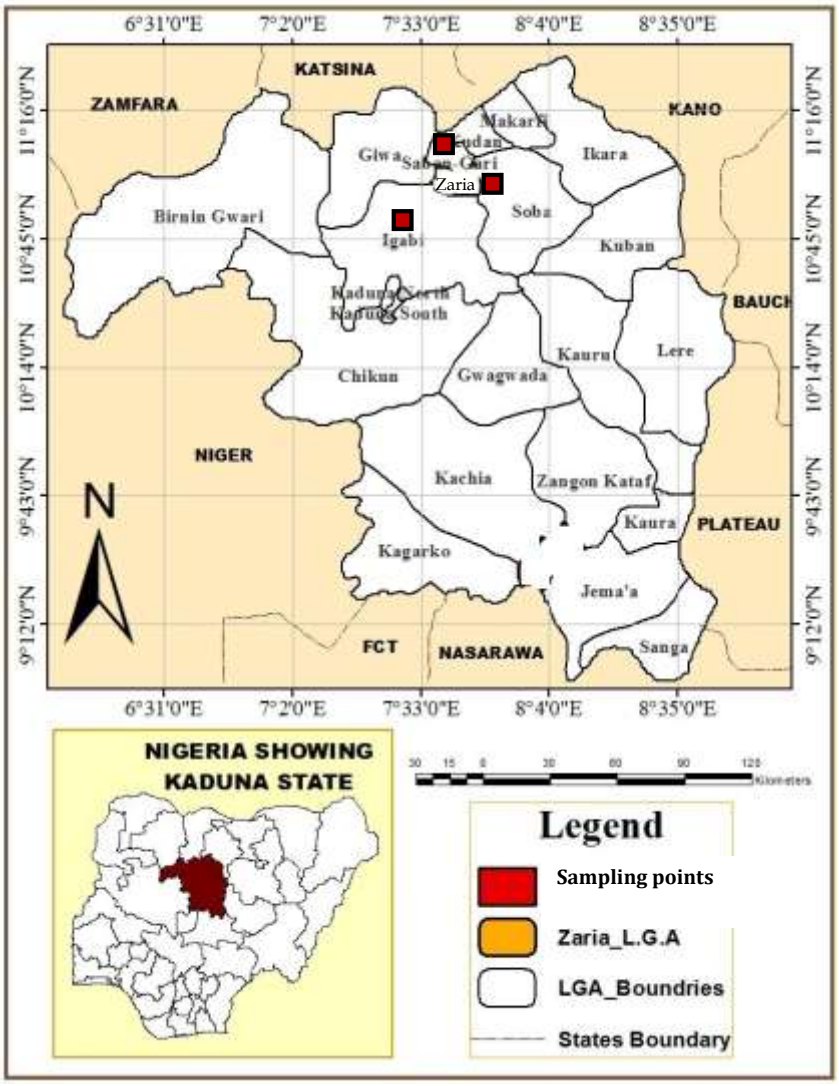


Figure 3.1:Map of Kaduna state showing the 3 selected LGAs, for the study.

Source: Administrative map of Kaduna State

3.3 Study Design

Purposeful and convenience sampling techniques were used for this study. Purposeful sampling technique was used to select the LGAs with the highest population of polo, racing and traditional ceremonial horses, whilst, convenience sampling technique was used to select horse stables with the agreement of horse owners and grooms to participate in the study. All horses within stables were examined for the presence of *Osteodystrophia fibrosa*.

3.4 Animals Studied

The guideline by Payne (1978) was used to determine the breed of the horses while sex of sampled horses was determined using the presence of the various sex organs for males and females. The ages of the horses were determined by using dentition as described by Musa (2013).

3.5 Samples Collection

3.5.1 Physical examination

The vital parameters (respiratory rate, pulse rates and rectal temperature) of all animals under study were taken as a routine for clinical examination of the horses. Rectal temperature was taken in degree Celsius (^{0}C) using a digital thermometer while pulse rate was taken in beats per minute at the external maxillary artery while the respiratory rate was taken in cycles per minute by examining the inspiration and expiration at the external nares of the sampled horses. Complete physical examination of the entire body of the horse, starting from the head was carried out. For those showing swollen head, palpation of the facial bones and percussion of the sinuses were done for evidence of malformations, along

with examination of the long bones for possible signs of the condition. Furthermore, the horses were walked to check for lameness which could be associated with *Osteodystrophia fibrosa*.Halter head restraint was used to restrain the horses.

3.5.2 Blood samples collection

About 10ml of whole blood was collected through a venipuncture of the jugular vein using a disposable sterile 10ml syringe and 18G needle after disinfecting the site for blood collection using chlorhexidine. Before blood collection each horse was restrained using head restraint by an assistant. Of the 10ml blood samples collected, 5ml was dispensed into a sterile Ethylene Diamine tetra-acetic acid (EDTA) (1mg/ml of blood) containing sample tubes for haematological analyses, while the remaining 5ml was placed in a plain tube without EDTA.

The collected blood samples without EDTA were transported to the DNA Laboratory, Kinkino Government Reservation Area (G.R.A) Kaduna in cold packs for serum biochemical analyses. In the Laboratory, each of the blood samples was centrifuged at 800×g for 15minutes. Serum was harvested and used for analysis of calcium, phosphorous and parathyroid hormone levels (PTH). Similarly, the blood samples with EDTA were transported in cold packs to the Clinical Pathology Laboratory, Medical Health Care Services ABU, Zaria for haematological analyses.

3.5.3 Faecal samplescollection

About 5g of faecal sample were collected from the rectum of each horse under study into polythene bags and transported to the Institute of Agricultural Research (IAR), Ahmadu

Bello University, Zaria laboratory where they wererefrigerated at 4^0C and used for analyses.

3.5.4 Roughage/concentrate feed samples collection

About 5g each of roughage and concentrates samples being fed to the horses in the stables under the study were also collected in well labelled polythene bags according to the stables and sent for identification at the Department of Biological Science, ABU Zaria while analysis of oxalate and phytase levels in the samples was carried out at the Department of Animal Sciences,Faculty of Agriculture Ahmadu Bello University Zaria.

3.6 Laboratory Investigations

3.6.1 Haematological parameters determination

The blood collected was analyzed using the method of Margi (1995). The Packed cell volume (PCV), the total red blood cells (RBC) count, the total white blood cells count (tWBC) and haemoglobin (Hb) concentration were determined using Mindray Bc 3600 haematology autoanalyzer Germany.

3.6.2 Serum biochemical parameters determination

The serum calcium and phosphorous levels were determined with the modified Clark-collip (1925) procedure, using automated Mindray Bs 230 machine. Parathyroid hormone levels were determined with the automated Chemiluminescent Technique (Weeks,1992), using the Mindray cli 1000i machine for the analysis.

3.6.3 Faecal calcium and phosphorous levels determination

The calcium and phosphorous levels were determined in faeces using an automatedmethod described by Henry (1915).

3.6.4 Feed samplesanalysis

Oxalate level was determined by titration method (Onwuka, 2005). Exactly 2g of the sample was dispensed in 190 ml of distilled water in 250ml volumetric flask. Ten milliliters of 6M HCL was added after which it was digested at 100^0C for 1 hour then cooled and made to volume before filtration. The filtrate was precipitated with ammonium hydroxide and the precipitate was dissolved in 10.0 ml of 20% sulphuric acid. The solution was titrated with 0.05M potassium permanganate.

Phytate level was determined using Reddy and Love (1999) method. Four grams of the ground sample was soaked in 100 ml of 2% HCl for 5 hours and then filtered. To 25 ml of the filterate, 5 ml 0.3% ammonium thiocyanate solution was added. The mixture was then titrated with Iron III chloride solution until a brownish-yellow colour that persisted for 5minutes was obtained.

3.7 Determination of Knowledge, Attitude and Practices of Horse Grooms on *Osteodystrophia Fibrosa.*

A structured questionnaire was prepared for the determination of knowledge, attitude and practices of horse grooms on *Osteodystrophia fibrosa* in the study area. The questionnaire was close-ended and was aimed at determining the biodata of respondents along with their educational standard, their source of knowledge of signs and lesions of*Osteodystrophia*

fibrosa as well as on the mode of treatment of the condition. Their attitude along with practices with regards to the condition was also determined.

3.8. Data Analysis

Data obtained were expressed as means and standard errors of means and presented in tables and charts. One -way analysis of variance (ANOVA) was used to compare data obtained from the study. The questionnaire was analyzed using the Chi square test for association. Values of $P \leq 0.05$ were considered statistically significant. SPSS version 26 was used for the analyses.

CHAPTER FOUR

4.0 RESULTS

4.1 Distribution of Horses Under the Study

A total of 59 horses were used for the study. Nineteen of the horses were from Igabi LGA while 20 horses each were fromZaria and Sabon Gari LGA had (Fig 4.1). They were also housed in various types of houses (Appendix XIV).

The sex distribution of the horses shows 28 male and 31 female horses were sampled during the study (Fig 4.1, Appendix III).

The breed distribution of the horses sampled showed 25 Argentine horses,21 Sudanese and 13 West African Barb breeds were sampled (Fig 4.1,Appendix III).

The age distribution of the horses indicated that 11 were of the ages 1-5years, while 21, 15and 12 horses were of the ages of 6-10years old, 11-15years old and 16-20 years of age respectively (Fig 4.1, Appendix III).

The occurrence rate of Osteodystrophy fibrosa was 74.6%.

4.2. Clinical Features of *Osteodystrophy fibrosa*

4.2.1 Clinical findings

The vital parameters of the 59 horses examined during the course of the study were within the normal ranges (Table 4.1). The overall mean respiratory rate for clinically diseased horses was 37.5 ±0. 22 beats per minute while in the non-clinically diseased horses, the respiratory rate was 37.4± 0.40. The mean pulse rates were 10.55±0.74 cycles per minute

and 10.53± 0.74 cycles per minute for the clinically and non-clinically diseased horses respectively and the overall mean rectal temperatures were 37.4°C±0.05°C and 37.6 ±0.67°C for clinically and non-clinically diseased horses respectively(Table 4.1).

Other clinical signs observed were enlarged facial bones (Plate I and II; Appendix XVI; Appendix XVII), poor body condition score with range 2/5 (Plate III; Appendix XVIII).

4.3 Serum Biochemical and Haematological Parameters of Sampled Horses

4.3.1 Biochemical parameters of sampled horses

The biochemical parameters of the horses under the study are presented in Figure 4.2. The horses in Zaria LGA had the highest calcium value for the non-clinically diseased horses with mean value of 2.82 ± 0.02 mmol/L, while the horses that were clinically diseased were found to be in Igabi LGA and had the highest value of 2.71 ± 0.03 mmol/L. Also, for the non-clinically diseased horses, Phosphorous levels were highest in horses in Sabon Gari LGA with a value of 1.20 ± 0.11 mmol/L while horses in Zaria LGA had the highest mean Phosphorous value of 1.26 ± 0.08 mmol/L in the clinically diseased horses (Fig. 4.2; Appendix IV).

The serum Parathyroid Hormone levels for horses under the study was highest in Igabi LGA for the non-clinically diseased horses with a mean value of 38.9 ± 3.50 pg/ml while horses in Zaria LGA had the highest value for the clinically diseased horses with mean SEM of 93.01 ± 20.44 pg/ml (Fig. 4.3; Appendix V).

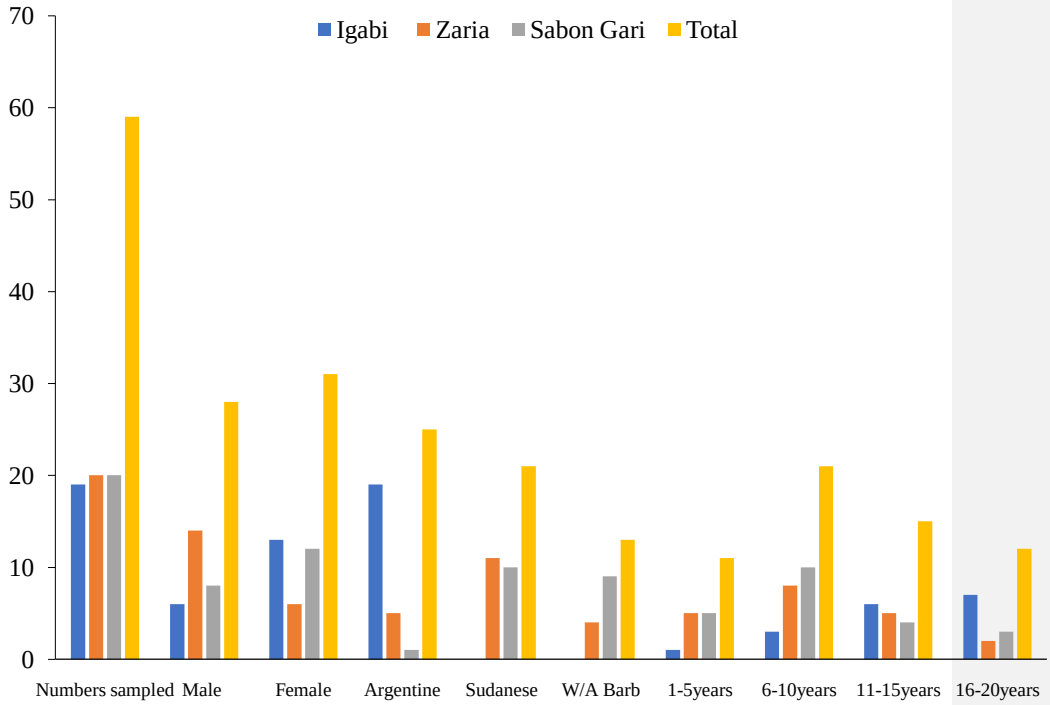


Figure 4.1: Distribution of horses sampled from the study areas.

Table 4.1: Mean (±SEM) values of vital parameters of sampled horses with clinical and non-clinical *Osteodystrophia fibrosa* in 3 selected Local Government Areas of Kaduna State.

Vital parameters	Clinically diseased	Non-clinically diseased	Normal range (ref value*
Pulse rate (cpm)	37.5 ±0. 22	37.4± 0.40	36-40
Respiratory rate (bpm)	10.55±0.74	10.53± 0.74	8-15
Temperature (°C)	37.4 ±0.05	37.6 ±0.67	37.5-38.5

*Sambo W.K (2015) P>0.05



Plate I:Swollen facial tissue (Arrow) in a horse with *Osteodystrophia Fibrosa*



Plate II: Bilateral enlargementof facial bones (mandibula and maxilla) in a horse with *Osteodystrophia Fibrosa* (Arrow)



Plate III: Body condition score of 2/5 in a horse with *Osteodystrophia Fibrosa* (arrow).

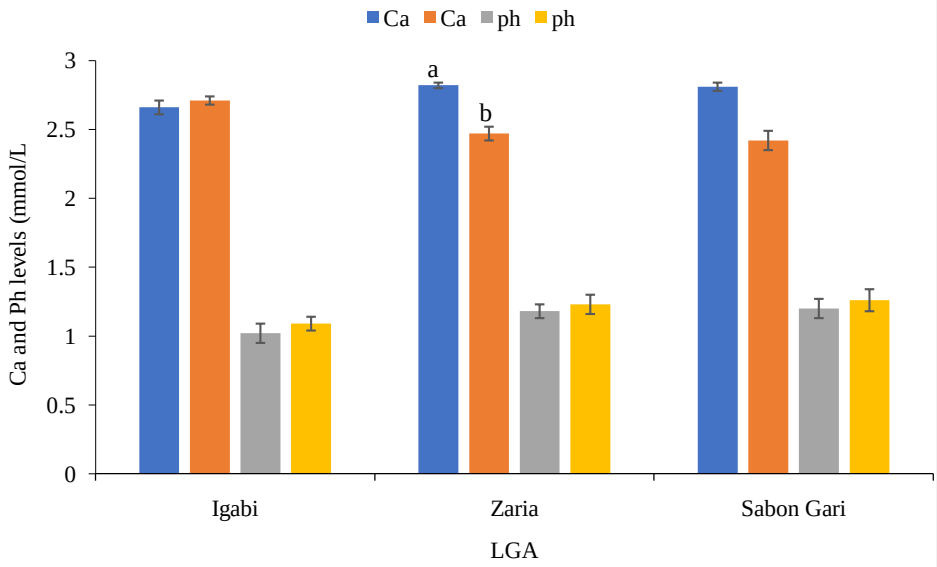


Figure 4.2: Mean (±SEM) levels of calcium and phosphorous in clinically and non - clinically bran diseased horses in the three selected local government areas in Kaduna state.
Key: Ca Nc=calcium Non-clinically diseased, Ca C=calcium clinicallydiseased,PhNc=phosphorous Non-clinically diseased, Ph C=phosphorous clinically diseased.[a,b]Means with different letters are significant at $p < 0.05$.

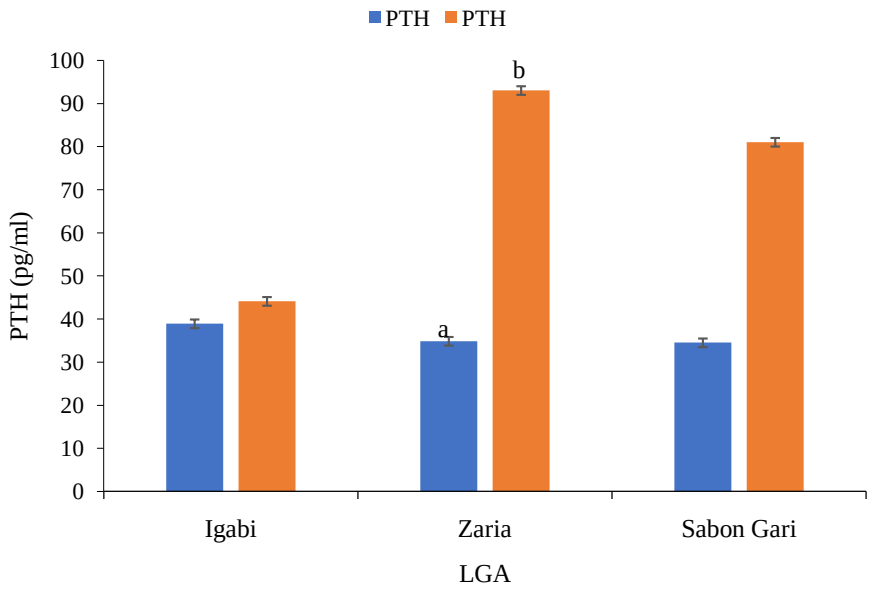


Figure 4.3: Mean (±SEM) Levels of parathyroid hormones in clinically and non -clinically bran diseased horses in the three selected local government areas in Kaduna state.

Key: PTH Nc = Parathyroid hormone Non-clinically diseased, PTH C= Parathyroid hormone clinically diseased. [a,b] Means with different letters are significant at $p < 0.05$.

4.3.1.1 Serum calcium and phosphorous levels of sampled horses by sex

The mean Calcium and Phosphorus levels by sex of horses used in the study showed that the Calcium values were highest in male horses for both clinically and non-clinically diseased horses with mean and SEM of 2.54± 0.05 mmol/L and 2.76± 0.03 mmol/L respectively(Fig 4.4; Appendix VI).

The phosphorous values for clinically diseased horses were same in male and Female with mean and SEM of 1.20± 0.06 mmol/L and highest in Female horses in the non-clinically diseased horses with mean SEM of 1.16± 0.07 mmol/L (Fig 4.4; Appendix VI).

The Parathyroid hormone levels by sex showed that the male horses were having the highest value for the clinically diseased horses with a mean SEM of 84.18± 13.80 pg/ml while the female had the highest value of 62.10± 6.19 pg/ml for the non-clinically diseased horses (Fig 4.5; Appendix VII).

4.3.1.2 Serum calcium and phosphorous levels of sampled horses by breed

The serum calcium and phosphorus levels by breed are presented in Figure 4.6. The serum calcium value was highest in Argentine horses in the clinically diseased horses (2.67± 0.04 mmol/L) while in the non-clinically diseased horses, the calcium value was highest in the W/A Barb (2.84± 0.03 mmol/L). The phosphorous value in the clinically diseased horses was highest in the Sudanese horses with a mean SEM of 1.34± 0.07 mmol/L. As for the

non-clinically diseased horses it was highest in W/A Barb breed with a mean SEM of 1.33± 0.15 mmol/L (Fig 4.6; Appendix VIII).

The Parathyroid hormone values across the sampled breeds showed that the W/A Barb horses had the highest values for both clinically diseased and non-clinically diseased horses with values of 91.72± 12.77 pg/ml and 36.59± 1.77 pg/ml respectively (Fig 4.7; Appendix VIX).

4.3.1.3 Serum calcium and phosphorous levels of sampled horses by age

The serum calcium and Phosphorus values for the sampled horses by age shows that the calcium values for the clinically diseased horses was highest in horses with the age range of 16-20 with a value of 2.72± 0.06 mmol/L while the non-clinically diseased horses had highest value with mean SEM of 2.88± 0.02 mmol/L in the 1-5years age bracket (Fig. 4.8; Appendix X).

The phosphorous value of the clinically diseased horses was highest in the 6-10 years age bracket with the mean SEM 1.27± 0.06 mmol/L while for the non-clinically diseased horses, the highest value of 1.33± 0.15 mmol/L was seen in the 6-10years age bracket (Fig. 4.8; Appendix X).

The values for Parathyroid hormone for the age groups of horses are shown Figure 4.8. Clinically diseased horses within the 6-10years old bracket had the highest value of 96.91± 16.66 pg/ml while for the non-clinically diseased horses the highest value (34.86± 3.15 pg/ml) was seen in horses within the 16-20years old age bracket (Fig 4.9; Appendix XI).

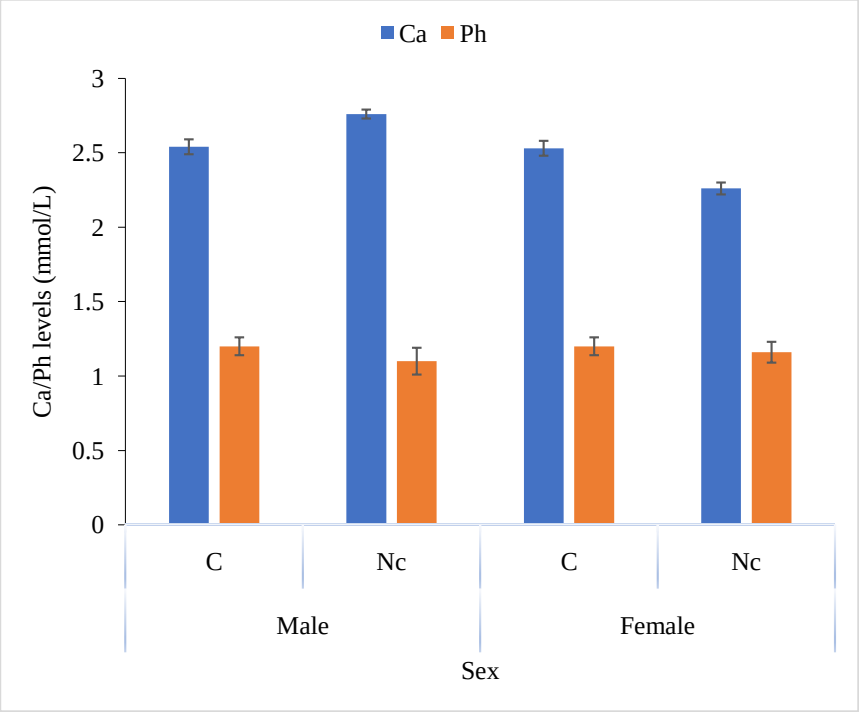


Figure 4.4: Mean (±SEM) levels of calcium and phosphorous in clinically and non - clinically bran diseased horses based on sex.Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased

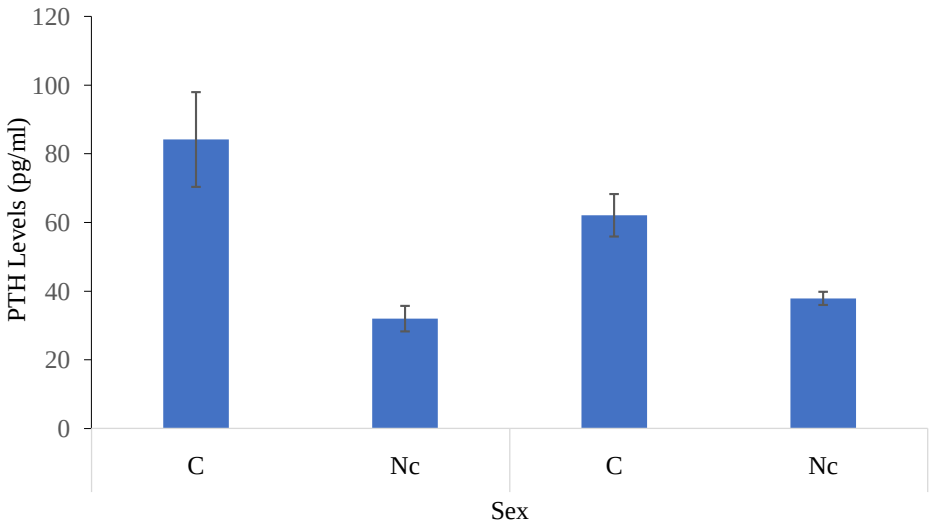


Figure 4.5: Mean (±SEM) levels of parathyroid hormones in clinically and non -clinically bran diseased based on sex.
Key: PTH= Parathyroid hormone, Nc=Non-clinically diseased, C=clinically diseased

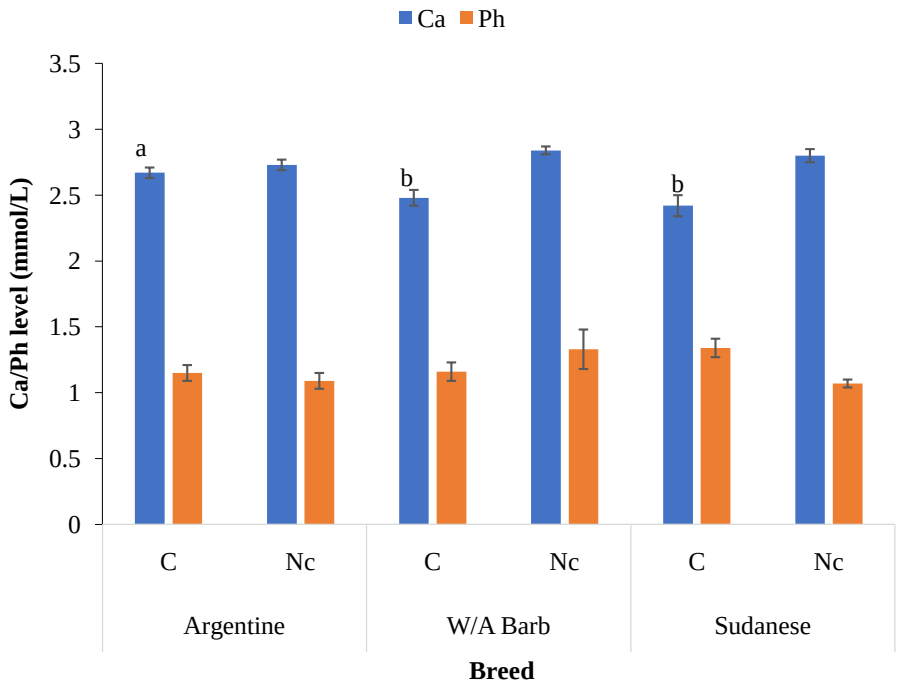


Figure 4.6: Mean (±SEM) levels of calcium and phosphorous in clinically and non-clinically bran diseased horses based on breed.Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased.[a,b] Means with different letters are
significant at $p < 0.05$.

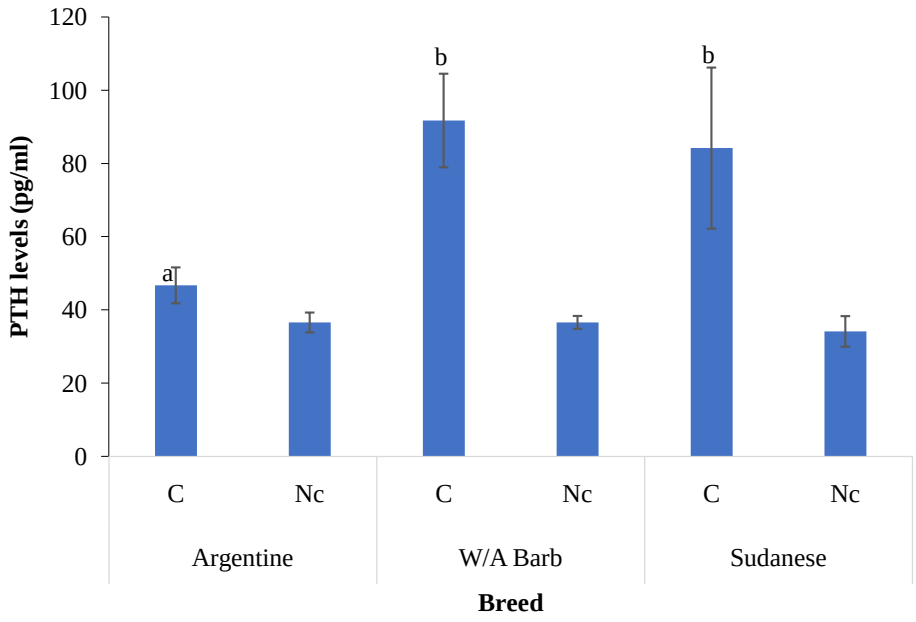


Figure 4.7: Mean (±SEM) levels of parathyroid hormones in clinically and non -clinically bran diseasedhorses based on breed.

Key: PTH= Parathyroid hormone,Nc=Non-clinically diseased, C=clinicallydiseased.[a,b] Means with different letters aresignificant at $p < 0.05$.

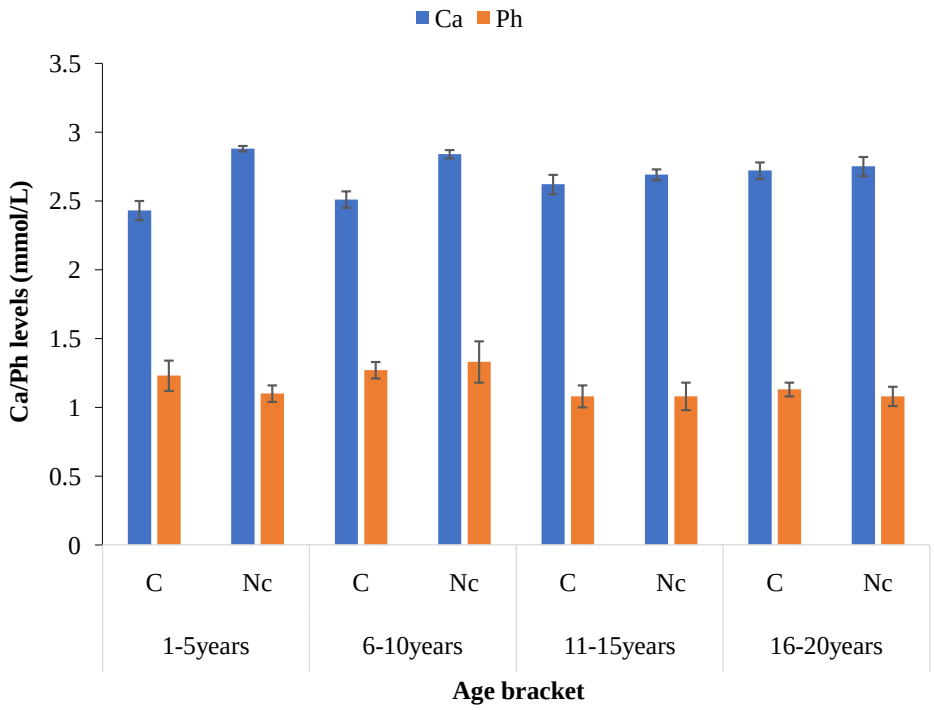


Figure 4.8: Mean (±SEM) levels of calcium and phosphorous in clinically and non - clinically bran diseased horses based on age.Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased

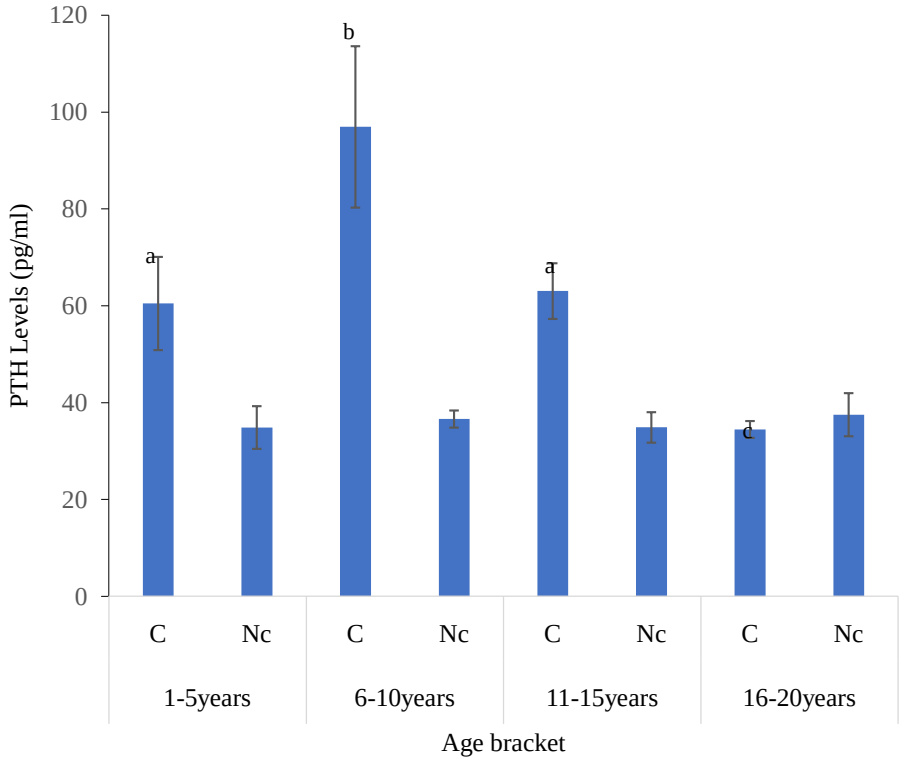


Figure 4.9: Mean (±SEM) levels of parathyroid hormones in clinically and non -clinically bran diseased horses based on age.Key: PTH= Parathyroid hormone, Nc=Non-clinicallydiseased, C=clinically diseased. Means with different letters aresignificant at $^{a,b}p < 0.05$, $^{c}p < 0.01$.

4.3.2 Haematological parameters of sampled horses

The haematological values of horses with clinical and non-clinical disease are shown in Table 4.2. Horses in Zaria LGA had the highest WBC value of 9.20 ±1.17(×10^9/L) and 8.79 ±0.65(×10^9/L) for both clinically diseased and non-clinically diseased horses respectively. Among the non-clinically diseased horses, those in Igabi LGA had the highest LYM value (2.88 ±0.86(×10^9/L) while the LYM for the clinically diseased horses were found in Zaria LGA with the highest value of 2.74 ±0.37(×10^9/L). The NEU value was highest (3.20 ±0.37(×10^9/L) for non-clinically diseased horses in Sabon Gari LGA while the highest value (3.50±0.33(×10^9/L) for clinically diseased horses was seen in horses in Igabi LGA. The GRAN value for the non-clinically diseased horses was highest (53.50 ±10.50(%) in horses in Sabon Gari LGA and those with highest value in clinically diseased horses were seen in Zaria LGA with a mean SEM of 56.07 ±2.28(%) (Table 4.2).

The RBC in clinically diseased horses was highest (6.71±0.29×10^6/ µl) in horses in Sabon Gari LGA while horses with non-clinical disease in Zaria LGA had the highest (6.07±0.30×10^6/ µl) RBC value. With regard to haemoglobin, horses in Zaria LGA with non-clinical disease had the highest value of 10.98±0.71 g/dl while those with clinical disease in Sabon Gari LGA had the highest Hg of 10.96 ±0.58 g/dl. Similarly, horses with both clinical and non-clinical disease were found in Sabon Gari LGA to have the highest PCV values of 32.14 ±1.64% and 29.78 ±3.10% respectively. Considering MCV, non-clinically diseased horses in Sabon Gari LGA had the highest MCV with 48.02±0.56 fl while clinically diseased horses in Igabi LGA had the highest MCV value of 46.59±69fl. Also, with regard to MCH, horses with non-clinical disease in Sabon Gari LGA had the

highest value of 19.90± 0.42 pg while the clinically diseased horses in Zaria LGA had highest value of 16.83±0.40 pg.

With regard to MCHC, horses without clinical disease in Sabon Gari LGA had highest (35.66± 0.50 g ⁄dl) MCHCwhile, the clinically diseased horses in Zaria LGA had the highest values of 35.73±0.35 g⁄dl respectively (Table 4.2).

4.3.2.1 Haematological values of horses by sex

The haematological values of horses under the study by sex has shown that the WBC in the clinically diseased horses was higher in male horses ($7.76\pm0.53\times10^9$ ⁄L) than in female horses with a value of ($7.56\pm0.54\times10^9$ ⁄L) (Table 4.3). However, in non-clinically diseased horses it was higher in females ($8.87\pm0.75\times10^9$ ⁄L) than in males ($5.94\pm0.58\times10^9$ ⁄L). The LYM in the clinically diseased horses was higher in males ($2.70\pm0.23\times10^9$ ⁄L) than in females with a value of ($2.46\pm0.21\times10^9$ ⁄L). Furthermore, the LYM count in the non-clinically diseased horses was higher ($2.24\pm0.18\times10^9$ ⁄L) in females than in males ($2.18\pm0.12\times10^9$ ⁄L). With regard to NEU count, the value for the clinically diseased horses was higher ($3.10\pm0.25\times10^9$ ⁄L) in the males than in females ($2.90\pm0.28\times10^9$/L). As for the corresponding values for the non-clinically diseased horses, females had higher NEU value of $3.11\pm0.26\times10^9$ ⁄L) than males ($2.80\pm0.20\times10^9$ ⁄L). The GRAN for the clinically and non-clinically diseased horses were both higher as 59.42±1.88 and 54.50±9.50 % in females than in males as 53.17±2.26 and 43.00±0.00% respectively (Table 4.3)

The RBC count for the clinically diseased horses was higher in females ($6.55\pm0.22\times10^6$ ⁄µl)than in males ($6.30\pm0.25\times10^6$⁄ µl) while the corresponding value for the non-clinically

diseased horses was higher in males (6.12±0.32×10^6/ µl) than in females (5.73±0.16×10^6/ µl)(Table 4.3).

The Hg value for the clinically diseased horses was higher (10.58±0.46g/dl) in females than in males (10.38±0.46 g/dl) while the corresponding value for non-clinically diseased horses was higher (11.28±0.58g/dl) in males than in females (10.68±0.56g/dl). The PCV for the clinically diseased horses was higher (30.46±1.14 %) in females than in males (29.48±1.29 %) while for non-clinically diseased horses it was higher in males (30.98±3.16 %) than in females (25.59±1.33 %)(Table 4.3).

The MCV for the clinically and non-clinically diseased horses were both slightly higher (46.33±0.48 fland 46.64±0.88 fl respectively) in females than in males (46.19±0.38 fl and 46.50±0.90 fl respectively (Table 4.3). As for MCH, the value for clinically diseased horses was higher in males (16.71±0.15 pg) than in females (16.52±0.12 pg) while for non-clinically diseased horses, the value was higher in females (16.98±0.14 pg) than in males (16.50±0.51 pg)(Table 4.3).

The MCHC for both the clinically and non-clinically diseased horses were higher in males (35.90±0.28 g/dl and 35.92±0.52 g/dl respectively) than in females (35.75±0.33 g/dl and 35.01±0.27 g/dl) respectively (Table 4.3).

4.3.2.2 Haematological values of horses by age

The haematological parameters of horses under the study by age are presented in Table 4.4. The WBC in the clinically diseased horses was highest in 6-10year old (8.24±0.63×10^9/L), followed by 11-15year old (8.08±0.77×10^9/L), 1-5year old (6.73±0.73×10^9/L) and least by those in the 16-20year (6.48±0.10×10^9/L) old age bracket. Similarly, for the non-clinically

diseased horses, the WBC was highest in the 16-20year old bracket ($9.14\pm1.10\times10^9$/L) followed by those in the 6-10year old age bracket ($8.90\pm1.45\times10^9$ /L), 1-5year old ($7.40\pm1.99\times10^9$ /L) and least in the 11-15year old group of horses ($6.35\pm0.13\times10^9$ /L) (Table 4.4).

The LYM for the clinically diseased horses was highest ($2.78\pm0.28\times10^9$/L) in 6-10year old and least in the 11-15year old horses ($2.37\pm0.32\times10^9$ /L) while for the non-clinically diseased horses the LYM values were highest ($2.34\pm0.05\times10^9$ /L) in the 16-20year old group of horses and least ($1.80\pm0.80\times10^9$/L) in the 6-10year old group horses(Table 4.4).

The NEU counts for the clinically diseased horses was highest ($3.55\pm0.41\times10^9$/L) in the 1-5year old horses and least ($2.73\pm0.27\times10^9$/L) in the 11-15year old horses. Similarly, the NEU for the non-clinically diseased horses was highest ($3.33\pm0.33\times10^9$/L) in horses within the age bracket of 1-5year and 6-10year old and least ($2.50\pm0.29\times10^9$/L) in the 11-15year old age bracket (Table 4.4).

As for the GRAN for the clinically diseased horses, the horses in the 11-15year old had the highest value of 57.71±3.50 % and horses in the 16-20year old bracket had the least (0.00±0.00) (Table 4.4).

The RBC count for the clinically diseased horses was highest in the 6-10year old group of horses and the least value was seen in the 11-15year old group of horses ($6.76\pm0.27\times10^6$/ µl) and $5.97\pm0.35\times10^6$/ µl respectively (Table 4.4). The corresponding values for the non-clinically diseased horses were $6.67\pm0.36\times10^6$/ µl for the 1-5year old horses and $5.56\pm0.12\times10^6$/µl for 16-20year old horses respectively (Table 4.4).

The Hg values for clinically diseased horses was highest (11.11±0.51 g/dl) in horses 6-10years old and least (9.33±0.54 g/dl) in the 11-15year old horses while for the non-clinically diseased horses it was highest (11.80±0.78 g/dl) in the 11-15year old and least (9.50±0.39 g/dl) in the 16-20year old horses (Table 4.4).

As for PCV for clinically diseased horses, it was highest (31.48±1.34 %) in the 6-10year old horses and least (28.44±1.81%) in the 1-5year old horses while for the non-clinically diseased horses, the value was highest (32.77±4.60% in the 1-5year old horses and least (23.22±1.56%) in the 16-20year old horses (Table 4.4).

The values for MCV for the clinically diseased horses was highest (46.58±0.56 fl) in the 11-15year old horses and least (45.90±0.62 fl) in the 1-5year old horses while in the non-clinically diseased horses, the highest value was 48.30±0.32 fl for horses in the 6-10years old age bracket and least (45.68±1.54 fl) for the 16-20years old horses (Table 4.4).

The MCH for the clinically diseased horses was highest (16.83±0.21 pg) in horses within the age range of 16-20years old and least (16.49±0.13 pg) in horses within 11-15years old. The corresponding MCH highest and lowest values for the non-clinically diseased horses were 17.10±0.36pg (6-10years) and 16.48±0.65pg (11-15years) respectively (Table 4.4).

The highest value MCHC for the clinically diseased horses was 36.28±0.44 g/dl in horses with the age bracket of 1-5years, while the lowest value 35.28±0.55 g/dl in horses between the age bracket of 11-15years.The highest and lowest corresponding values for the non-clinically diseased horses were 35.48±0.78 g/dl and 35.18±0.38 g/dl respectively (Table 4.4).

4.3.2.3 Haematological values of horses by breed

The haematological parameters of the horses by breed are presented in Table 4.5. The highest WBC value ($8.20\pm0.92\times10^9$/L) for the clinically diseased horses was seen in the Sudanese breed of horses while the lowest value ($6.76\pm0.49\times10^9$/L was seen in Argentine breed of horses. Similarly, for the non-clinically diseased horses, the highest value ($8.90\pm1.45\times10^9$ /L) was observed in the WA Barb breed while the lowest value ($6.93\pm1.97\times10^9$/L) was obtained in the Sudanese breed of horses (Table 4.5).

As for LYM, the highest value ($2.84\pm0.56\times10^9$/L) in the clinically diseased horses was in the Sudanese breed of horses while the lowest value ($2.50\pm0.11\times10^9$/L) was seen in the Argentine breed of horses. The corresponding LYM values for the non-clinically diseased horses were 2.36±0.06 and $1.80\pm0.80\times10^9$/L respectively (Table 4.4).

The highest NEU value ($3.56\pm0.28\times10^9$/L) for the clinically diseased horses was recorded in the Argentine breed of horses while the lowest value ($2.50\pm0.24\times10^9$/L) was seen in the WA Barb breed of horses.The values for the non-clinically diseased horses were $3.33\pm0.33\times10^9$/L in the WA Barb breed of horses and $2.67\pm0.33\times10^9$/L in the Sudanese breed of horses(Table 4.4).

The highest value (58.40±2.28%) of GRAN for clinically diseased horses was seen in Sudanese breed of horses while the lowest value (54.06±2.32%) was recorded in WA Barb breed of horses. Similarly, the corresponding values for the highest (54.50±9.50%) and lowest (0.00±0.00) values for the non-clinically diseased horses were seen in WA Barb and Argentine breed of horses respectively (Table 4.5).

The highest value (6.79±0.24×10^{6}/ µl) for RBC count for clinically diseased horses were seen in WA Barb horses while the lowest value (5.95±0.27×10^{6}/ µl) was recorded in the Argentine breed of horses. Similarly, for the non-clinically diseased horses, the highest value was seen in the Sudanese breed of horses and the lowest (5.67±0.12×10^{6}/ µl) was in the Argentine breed of horses (Table 4.5).

The highest value (11.26±0.77g/dl) for Hg in the clinically diseased horses was observed in the Sudanese breed of horses while the lowest (9.74±0.52 g /dl) was seen in Argentine breed of horses (Table 4.4).

The highest value (30.57±1.81%) for PCV in the clinically diseased horses was seen in the Sudanese breed of horses while the lowest value (26.90±1.18%) was observed in the Argentine breed of horses. With regard to non-clinically diseased horses, the highest value for PCV (36.13±2.58%) was observed in Sudanese breed of horses and the lowest value (24.61±1.05%) was observed in the Argentine breed of horses (Table 4.5)

The MCV for the clinically diseased horses showed that WA Barb breed of horses had the highest value (46.57±0.27 fl) while the Sudanese breed of horses had the least (45.66±0.70 fl). As for the non-clinically diseased horses, WA Barb breed of horses had the highest value (48.30±0.31fl) while Argentine horses had the lowest MCV value (45.98±0.90 fl) (Table 4.4).

In terms of MCH for the clinically diseased horses, WA Barb breed of horses had the highest value 16.78±0.13pg and least by Sudanese breed of horses with 16.26±0.27 pg. As for the non-clinically diseased horses, WA Barb breed had the highest value (17.10±0.36 pg) while, the Sudanese breed of horses had the least value of 16.57±0.60 pg(Table 4.4).

With regard to the MCHC for clinically diseased horses, Argentine breed of horses had the highest MCHC (35.95±0.42 g⁄dl)while the least value was obtainedin the WA Barb breed of horses with 35.68±0.30 g⁄dl value. As for those with non-clinical disease, Sudanese horses had the highest value (36.80±0.50g⁄dl) for MCHC value while Argentine breed of horses had the least (35.14±0.32 g⁄dl) (Table 4.5).

Table 4.2: Mean (±SEM) haematological values in clinically and non -clinically bran diseased horses in the three selected Local Government Areas in Kaduna State.

Parameter	**Horse group**	**Igabi**	**Zaria**	**Sabon Gari**	**Ref.value (Ememe *et***

					al **2015)**
WBC(×10^9⁄L)	Nc	7.50 ±0.79	9.20 ±1.17	7.30 ±1.24	5.4 -14.3
	C	6.37 ±0.37	8.79 ±0.65	7.74 ±0.73	
LYM(×10^9⁄L)	Nc	2.88 ±0.86	2.43 ±0.09	2.02±0.28	
	C	2.43 ±0.87	2.74 ±0.37	2.68 ±0.43	
NEU(×10^9⁄L)	Nc	2.80 ±0.37	3.00 ±0.00	3.20 ±0.37	
	C	3.50 ±0.33	2.92 ±0.29	2.60 ±0.31	
GRAN (%)	Nc	0.00 ±0.00	45.00 ±0.00	53.50 ±10.50	
	C	0.00 ±0.00	56.07 ±2.28	55.27 ±2.42	
RBC (×10^6⁄µl)	Nc	5.74 ±0.19	6.07 ±0.30	5.99 ±0.34	6.8-12.9
	C	5.97 ±0.30	6.55 ±0.26	6.71 ±0.29	
Hg (g⁄dl)	Nc	10.86 ±0.63	10.98 ±0.71	10.94 ±0.78	11.0-19.0
	C	9.46 ±0.56	10.94 ±0.48	10.96 ±0.58	
PCV (%)	Nc	25.06 ±0.45	28.64 ±1.68	29.78 ±3.10	32-55
	C	26.71 ±1.35	30.78 ±1.17	32.14 ±1.64	
MCV (fl)	Nc	44.68 ±0.79	46.26 ±1.08	48.02 ±0.56	37-58.5
	C	46.59 ±0.69	46.26 ±0.38	45.95 ±0.49	
MCH (pg)	Nc	16.56 ±0.34	16.98 ±0.21	19.90 ±0.42	12.3-19.7
	C	16.54 ±0.11	16.83 ±0.14	16.47 ±0.22	
MCHC(g⁄dl)	Nc	35.08 ±0.47	35.28 ±0.40	35.66 ±0.50	31.0-38.6
	C	35.67 ±0.38	36.08 ±0.38	35.73 ±0.35	

Key: WBC=White blood cell, LYM= Lymphocytes, NEU= Neutrophils, GRAN=Granulocytes, RBC= Reb blood cell, Hg= Haemoglobin, PCV=Pack cell volume, MCV= Mean corpuscular volume, MCH= Mean Corpuscular Hemoglobin, MCHC=Mean Corpuscular Hemoglobin Concentration, Nc=Non-clinically diseased, C=clinically diseased. Means are not statistically significant ($p > 0.05$).

Table 4.3: Mean (±SEM) haematological values in clinically and non-clinically bran diseased horses based on sex

Parameter	Horse Group	Male	Female	Ref.value (Ememe *et al.*, 2015)
WBC($\times 10^9$/L)	C	7.76± 0.53	7.56 ±0.54	5.4 -14.3
	Nc	5.94 ±0.58	8.87 ±0.75	
LYM($\times 10^9$/L)	C	2.70 ±0.23	2.46 ±0.21	
	Nc	2.18 ±0.12	2.24 ±0.18	
NEU($\times 10^9$/L)	C	3.10 ±0.25	2.90 ±0.28	
	Nc	2.80 ±0.20	3.11± 0.26	
GRAN (%)	C	53.17 ±2.26	59.42 ±1.88	
	Nc	43.00 ±0.00	54.50 ±9.50	
RBC ($\times 10^6$/µl)	C	6.30 ±0.25	6.55 ±0.22	6.8-12.9
	Nc	6.12 ±0.32	5.73 ±0.16	
Hg (g/dl)	C	10.38± 0.46	10.58 ±0.46	11.0-19.0
	Nc	11.28 ±0.58	10.68 ±0.56	
PCV (%)	C	29.48 ±1.29	30.46 ±1.14	32-55
	Nc	30.98 ±3.16	25.59 ±1.33	
MCV (fl)	C	46.19 ±0.38	46.33 ±0.48	37-58.5
	Nc	46.50 ±0.90	46.64 ±0.88	
MCH (pg)	C	16.71 ±0.15	16.52 ±0.12	12.3-19.7
	Nc	16.50 ±0.51	16.98 ±0.14	
MCHC(g/dl)	C	35.90 ±0.28	35.75± 0.33	31.0-38.6
	Nc	35.92 ±0.52	35.01± 0.27	

Key: WBC=White blood cell, LYM= Lymphocytes, NEU= Neutrophils, GRAN=Granulocytes, RBC= Reb blood cell, Hg= Haemoglobin, PCV=Pack cell volume, MCV= Mean corpuscular volume, MCH= Mean Corpuscular Hemoglobin, MCHC=Mean Corpuscular Hemoglobin Concentration, Nc=Non-clinically diseased, C=clinically diseased. Means are not statistically significant ($p > 0.05$).

Table 4.4: Mean (±SEM) haematological values in clinically diseased and non -clinically bran diseased horses based on age.

Parameter	Age	1-5years	6-10years	11-15years	16-20years	Ref.value (Ememe *et al*., 2015)
WBC($\times 10^9$/L)	C	6.73 ±0.73	8.24 ±0.63	8.08 ±0.77	6.48 ±0.10	5.4 -14.3
	Nc	7.40 ±1.99	8.90 ±1.45	6.35 ±0.13	9.14 ±1.10	
LYM($\times 10^9$/L)	C	2.51 ±0.35	2.78 ±0.28	2.37 ±0.32	2.43 ±0.15	
	Nc	2.25 ±0.25	1.80 ±0.80	2.25 ±0.15	2.34 ±0.05	
NEU($\times 10^9$/L)	C	3.55 ±0.41	2.81 ±0.28	2.73 ±0.27	3.00 ±0.82	
	Nc	3.33 ±0.33	3.33 ±0.33	2.50 ±0.29	3.00 ±0.32	
GRAN (%)	C	53.50 ±3.08	55.87 ±2.39	57.71 ±3.50	0.00 ±0.00	
	Nc	44.00 ±1.00	54.50 ±9.50	0.00 ±0.00	0.00 ±0.00	
RBC($\times 10^6$/µl)	C	6.44 ±0.33	6.76 ±0.27	5.97 ±0.35	6.08 ±0.19	6.8-12.9
	Nc	6.67 ±0.36	6.16 ±0.32	5.66 ±0.23	5.56 ±0.12	
Hg (g/dl)	C	10.97 ±0.58	11.11± 0.51	9.33 ±0.54	9.43 ±1.24	11.0-19.0
	Nc	11.43 ±0.43	11.63 ±0.79	11.80 ±0.78	9.50 ±0.39	
PCV (%)	C	28.44 ±1.81	31.48 ±1.34	29.36 ±1.88	28.80 ±1.56	32-55
	Nc	32.77 ±4.60	29.17 ±1.61	28.88 ±2.23	23.22 ±1.56	
MCV (fl)	C	45.90 ±0.62	46.23 ±0.46	46.58 ±0.56	46.50 ±1.46	37-58.5
	Nc	46.17 ±0.87	48.30 ±0.32	47.05 ±0.72	45.68 ±1.54	
MCH (pg)	C	16.65 ±0.21	16.63 ±0.18	16.49± 0.13	16.83 ±0.21	12.3-19.7
	Nc	16.97 ±0.23	17.10 ±0.36	16.48 ±0.65	16.82 ±0.12	
MCHC (g/dl)	C	36.28 ±0.44	35.96 ±0.27	35.28± 0.55	35.50 ±0.34	31.0-38.6
	Nc	35.30 ±0.58	35.47 ±0.75	35.48 ±0.78	35.18 ±0.38	

Key: WBC=White blood cell, LYM= Lymphocytes, NEU= Neutrophils, GRAN=Granulocytes, RBC= Reb blood cell, Hg= Haemoglobin, PCV=Pack cell volume, MCV= Mean corpuscular volume, MCH= Mean Corpuscular Hemoglobin, MCHC=Mean Corpuscular Hemoglobin Concentration, Nc=Non-clinically diseased, C=clinically diseased. Means are not statistically significant ($p > 0.05$).

Table 4.5: Mean (±SEM) haematological values in clinically and non -clinically bran diseased horses based on breed.

Parameter	Breed	Argentine	W/A Barb	Sudanese	Ref.value (Ememe *et al.*, 2015)
WBC($\times 10^9$/L)	C	6.76 ±0.49	8.17 ±0.61	8.20 ±0.92	5.4 -14.3
	Nc	8.06 ±0.74	8.90 ±1.45	6.93 ±1.97	
LYM($\times 10^9$/L)	C	2.50 ±0.11	2.58 ±0.35	2.84 ±0.56	
	Nc	2.36 ±0.06	1.80 ±0.80	2.00 ±0.00	
NEU($\times 10^9$/L)	C	3.56 ±0.28	2.50 ±0.24	2.90 ±0.41	
	Nc	3.00 ±0.24	3.33 ±0.33	2.67 ±0.33	
GRAN (%)	C	56.50 ±6.50	54.06 ±2.32	58.40 ±2.28	
	Nc	0.00 ±0.00	54.50 ±9.50	44.00 ±1.00	
RBC($\times 10^6$/µl)	C	5.95 ±0.27	6.79 ±0.24	6.51 ±0.37	6.8-12.9
	Nc	5.67 ±0.12	6.16 ±0.32	6.48 ±0.54	
Hg (g/dl)	C	9.74 ±0.52	10.69 ±0.45	11.26 ±0.77	11.0-19.0
	Nc	10.56 ±0.55	11.63 ±0.79	11.33 ±0.52	
PCV (%)	C	26.90 ±1.18	32.31 ±1.31	30.57 ±1.81	32-55
	Nc	24.61 ±1.05	29.17 ±1.61	36.13 ±2.58	
MCV (fl)	C	46.28 ±0.64	46.57 ±0.27	45.66 ±0.70	37-58.5
	Nc	45.98 ±0.90	48.30 ±0.31	47.10 ±0.50	
MCH (pg)	C	16.66 ±0.12	16.78 ±0.13	16.26 0±.27	12.3-19.7
	Nc	16.80 ±0.23	17.10 ±0.36	16.57 ±0.60	
MCHC(g/dl)	C	35.95 ±0.42	35.68 ±0.30	35.91 ±0.39	31.0-38.6
	Nc	35.14 ±0.32	35.47 ±0.75	36.80 ±0.50	

Key: WBC=White blood cell, LYM= Lymphocytes, NEU= Neutrophils, GRAN=Granulocytes, RBC= Reb blood cell, Hg= Haemoglobin, PCV=Pack cell volume, MCV= Mean corpuscular volume, MCH= Mean Corpuscular Hemoglobin, MCHC=Mean Corpuscular Hemoglobin Concentration, Nc=Non-clinically diseased, C=clinically diseased. Means are not statistically significant ($p > 0.05$).

4.4 Faecal Calcium and Phosphorous Levels in Sampled Horses

The faecal calcium and phosphorus levels are presented in Figure 4.10 and Appendix XII. The faecal calcium value was highest in horses in Igabi LGA with a value of 75.89±6.55 mg/kg for the non-clinically diseased horses and 82.85 ± 4.17 mg/kg for the clinically diseased horses. Similarly, the faecal phosphorous values for the clinically diseased horses was highest in horses in Igabi LGA with a value of 2801.76 ± 212.51 mg/kg while the lowest phosphorus level of 2554.5 ± 207.38 mg/kg was recorded in Sabon Gari LGA.

4.4.1Faecal calcium and phosphorous levels in sampled horses by sex

The faecal calcium and phosphorus levels by sex shows the calcium level was higher inclinically diseased females (64.69 ±6.66 mg/kg)than their male counterparts(57.22± 8.19 mg/kg) while, for the non-clinically diseased horses, the males had higher value (76.03± 16.17 mg/kg)than what was seen in the females with (73.82± 8.88 mg/kg). However, the faecal phosphorus levels were higher in males for clinically diseased (2705.64± 172.38 mg/kg) while the females had the higher value of (2501.11± 205.30 mg/kg) for non-clinically diseased horses (Fig 4.11; Appendix XIII).

4.4.2Faecal calcium and phosphorous levels in sampled horses by age

The faecal calcium and phosphorus levels by age showed that the faecal calcium level was highest in the 16-20years old group of horses in both clinically (80.47±7.28 mg/kg) and non-clinically (89.97±7.56 mg/kg) diseased horses. However, the faecal phosphorus level was highest (2901.34±258.81 mg/kg) in clinically diseased horses in the age bracket 11-15years old and 2516.33±349.46 mg/kg for non- clinically diseased horses in the 6-10years old bracket (Fig 4.12; Appendix XIV)

4.4.3Faecal calcium and phosphorous levels in sampled horses by breed

The faecal calcium and phosphorus levels by breed showed that the faecal calcium level was highest in the Argentine horses with a mean SEM of 88.84±5.54 mg/kg and 85.33± 5.19 mg/kg for both clinically diseased and non-clinically diseased horses respectively. Similarly, the faecal phosphorus levels were highest (2773.42± 195.50 mg/kg) in Argentine horses for the clinically diseased horses and highest (2750.86± 189.08 mg/kg in Sudanese horses for the non-clinically diseased horses (Fig 4.13; Appendix XV)

4.5 Level of oxalate and phytate in horse feeds

The grasses identified to be fed to the horses during the time of sampling were *Bracheria decumbens and Vossia cuspidate*, while the concentrates were pelleted processed feed, corn bran and soya bean hull (Plates IV to VIII; Appendix XX)

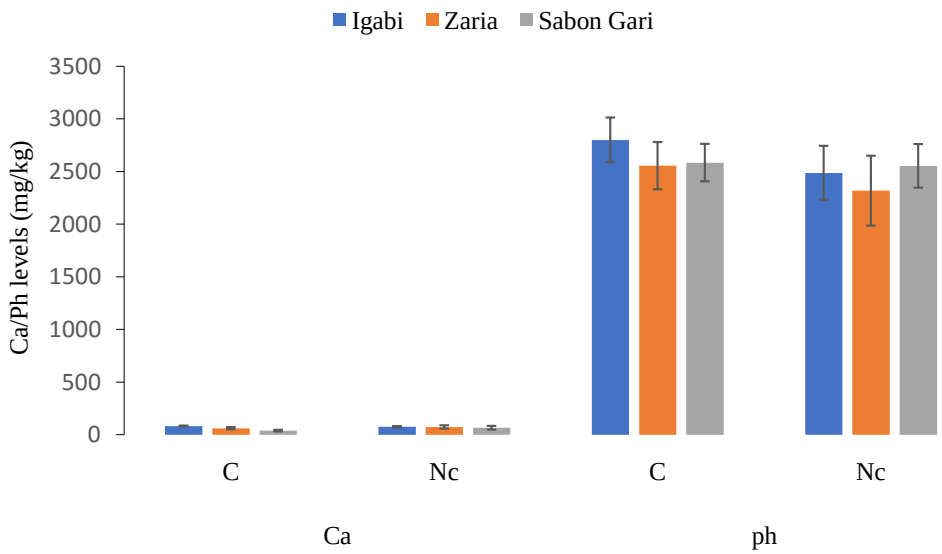


Figure 4.10: Mean (±SEM) faecal calcium and phosphorous levels in clinically and non - clinically bran diseased horses in the three selected Local Government Areas of Kaduna State.Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased,C=clinically diseased.

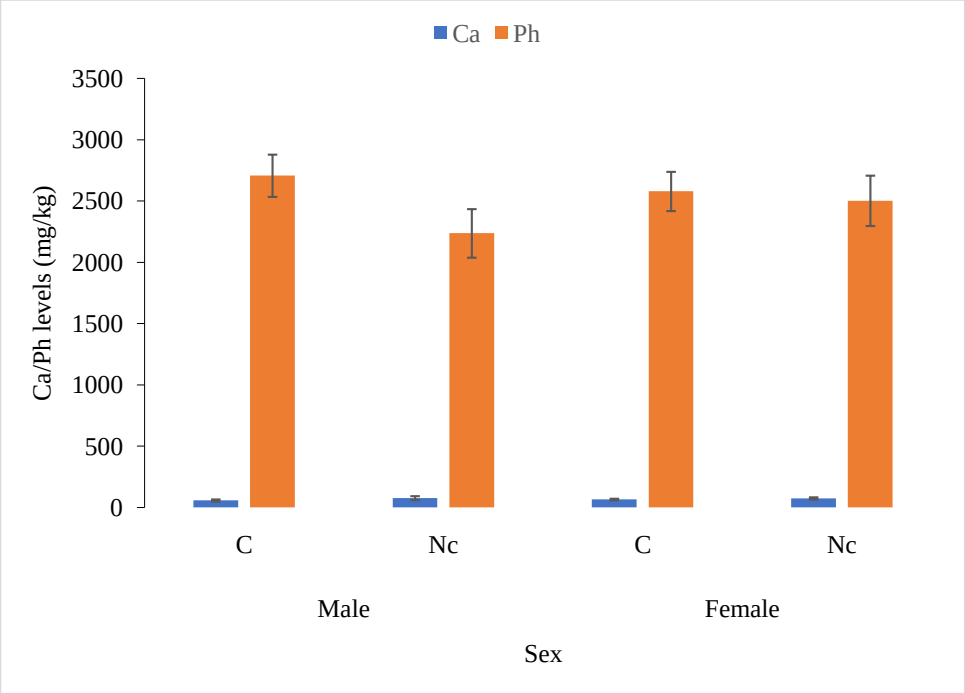


Figure 4.11: Mean (±SEM) Calcium and Phosphorous levels of faeces in clinically and non -clinically bran diseased Horses based on sex.Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased.

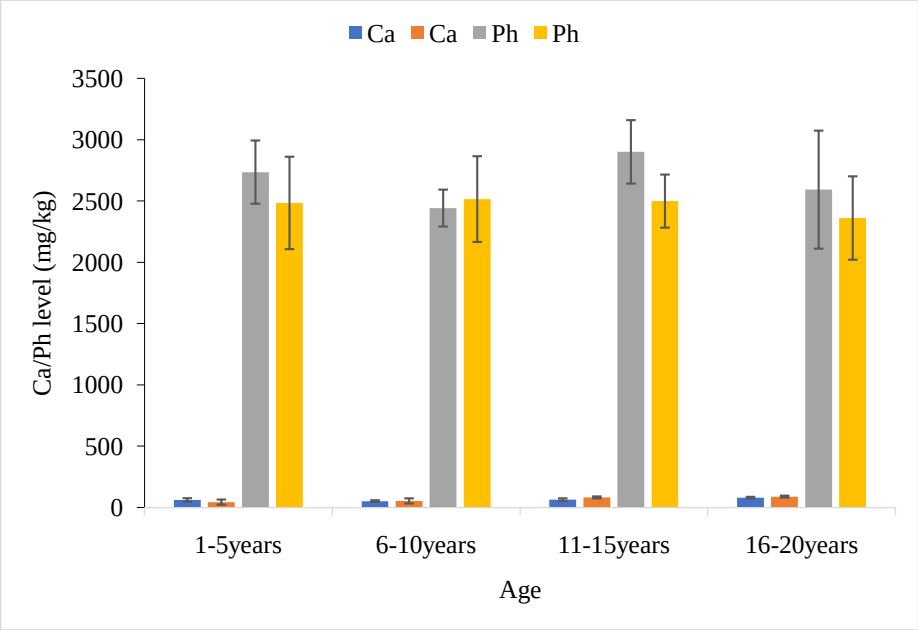


Figure 4.12: Mean (±SEM) calcium and Phosphorous levels in faeces of clinically and non -clinically brandiseased Horses based on Age.Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased

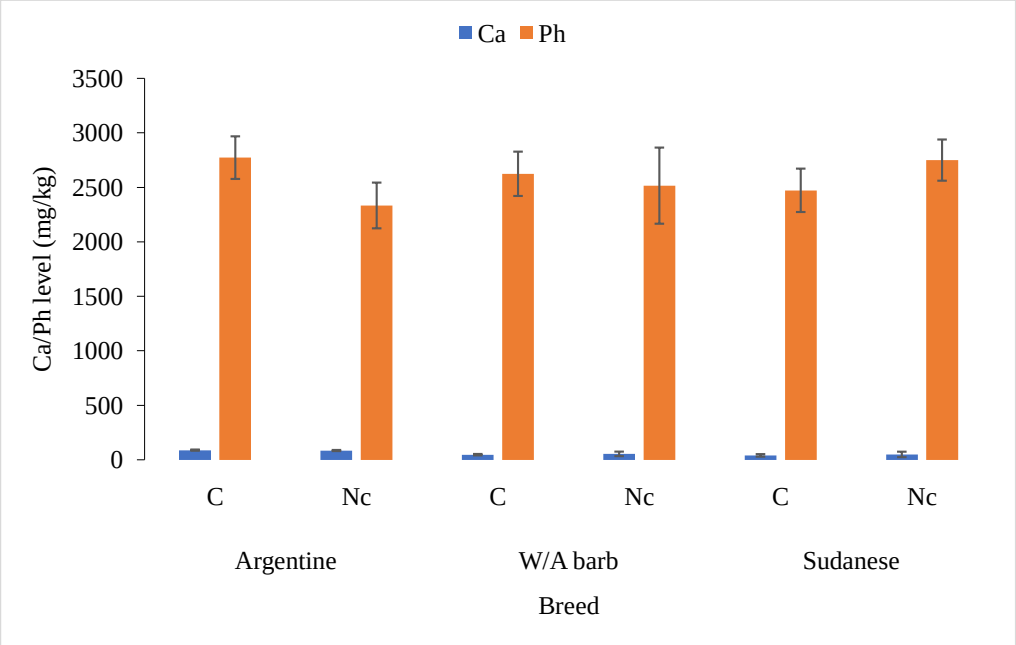


Figure 4.13: Mean (±SEM) calcium and Phosphorous levels in faeces of clinically and non -clinically bran disease based on breed.Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased

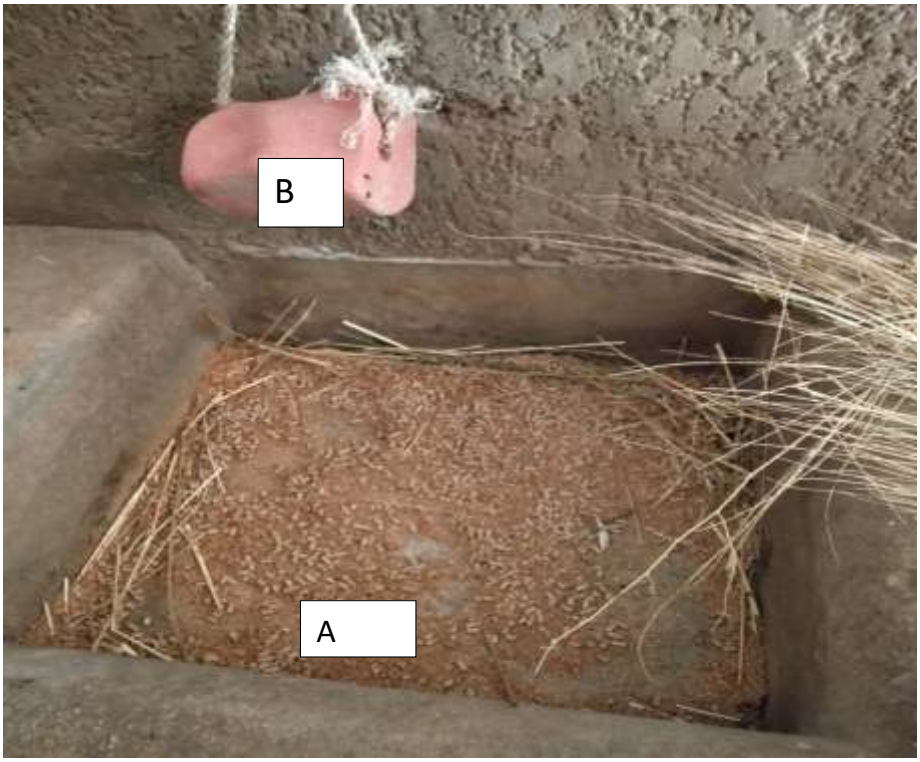


Plate IV: Pelleted feed (A) and Salt lick (B).



Plate V: *Bracheria decumbens*straw(arrow)



Plate VI: Soya bean hull (Arrow)



Plate VII: *Vossia cuspidate* (Arrow)



Plate VIII: Corn bran (Arrow).

Table 4.6: Analyses of horse feeds for oxalate and phytate in selected Local Government Areas of Kaduna state

Feed	Oxalate mg/100kg	Phytate mg/100kg
Soya bean hull	0	0.51
Corn bran	0	0.77
Vossia cuspidate	0.02	0
Bracheria decumbens	0	0

4.6 Knowledge, Attitude and Practice of Participating Horse Grooms

4.6.1 Grooms' demographic data

The demographic information of the participating grooms in the study shows the responses of horse grooms in the three sampled LGA with a total of 21 questionnaire administered with each LGA having 7 respondents. All the respondents were males. Most of them were primary school leaving certificate holders 11 (52.4%), with only 3 (14.3%) having tertiary education (Table 4.7).

From the grooms' responses it was evident that breeds of horses being kept by stables included Argentine (33.3%), West African Barb (28.6%), Sudanese (14.3%) and horses of mixed breeds (23.8%) (Table 4.7).

4.6.2 Grooms' knowledge on *Osteodystrophia fibrosa*

A total of 21 grooms participated in the questionnaire aspect of the study with 18 (85.7%) of them indicating knowing the condition*Osteodystrophia fibrosa*. Eleven (52.4%) of the grooms indicated their source of knowledge of the condition to be veterinarians while 8 (38.1%) indicated their source of knowledge to be friends. Furthermore, 15 (71.4%) of them reported signs of the condition to include swollen face while 1 (4.8%) and 3 (14.3%) others respectively indicated emaciation and other non-identified signs of the condition (Table 4.8).

4.6.3 Attitude of grooms to *Osteodystrophia fibrosa* (bran disease)

The responses of grooms on attitude towards *Osteodystrophia fibrosa* showed that 19 (90.5%) of them indicated reporting such cases to the Clinic. On further enquiry whether

they rode such horses, 6 (28.6%) of them reported riding such horses while 13 (61.9%) said they do not ride horses with the condition, 9 (42.9%) said they pitied horses with such condition and as such they did not ride them (Table4.9).

On question bothering about the nature of the feeds being fed, they were feeding their horses, 13 (61.9%) grooms answered positively while 6 (28.6%) others reported not bothering on the type of feed they fed their horses. (Table 4.9)

4.6.4Practices of grooms with respect to bran disease

The study enquired on whether grooms were doing anything to prevent their horses from coming down with Bran disease. Fifteen (71.4%) of the grooms indicated doing everything possible to prevent their horses coming down with the condition. In such cases 6 (28.6%) of the grooms indicated changing the feeds while 1 (47.6%) of the grooms indicated medicating them along with providing the horses with supplements. Yet another 8 (38.1%) of the grooms said they would give such horses bone meal as a means of controlling the condition. Three (14.3%) other grooms indicated using other methods for the control of the condition. (Table 4.10).

Table 4.7: Grooms' demographic information

LGA	No. G	Educational status			Breed of horses under grooms No. (%)			
		Primary	Secondary	Tertiary	Argentine	WA Barb	Sudanese	Mixed
Igabi	7(33.3%)	3 (42.9%)	2 (28.6%)	2 (28.6%)	7(100%)	0(0%)	0(0%)	0(0%)
Zaria	7(33.3%)	5 (79.4%)	2 (28.6%)	0	0(0%)	4(57.1%)	1(14.3%)	2(28.6%)
SG	7(33.3%)	3(42.9%)	3(42.9%)	1 (14.3%)	0(0%)	2(28.6%)	2(28.6%)	3(42.9%)
Total	**21(100%)**	**11(52.4%)**	**7(33.3%)**	**3(42.9%)**	**7(33.3%)**	**6(28.6%)**	**3(14.3%)**	**5(23.8%)**

Key:WA Barb= West African Barb No. G= No of Grooms SG=Sabon Gari

Table 4.8: Knowledge of grooms on *Osteodystrophia fibrosa*in three Local Government Areas of Kaduna State, Nigeria

LGA	NR	Knowledge of bran disease			Source of knowledge			Recognition of signs of bran disease			
		Yes	No	NoR	Vet	Friends	NoR	SF	Emaciation	Others	NoR
Igabi	7(100%)	7(100%)	0	0	7(100%)	0	0	4(57.1%)	0	3(42.9%)	0
Zaria	7(100%)	7(100%)	0	0	4(57.1%)	3(42.9%)	0	6(85.7%)	0	0	0
SG	7(100%)	4(66.7%)	2(33.3%)	1	0	5(100%)	2	5(100%)	0	0	2
Total	21(100%)	18(90%)	2(10%)	1	11(57.9%)	8(42,1%)	2	15(78.9%)	1(5.3%)	3(15.8%)	2

Key: NR: Number of respondents, NoR: No response, SG: Sabon Gari, SF: Swollen face

Table 4.9: Attitude of grooms to *Osteodystrophia fibrosa* in three Local Government Areas of Kaduna State, Nigeria

LGA	**No. Grooms**	**Action if horse develops disease**		**Riding horse with Bran disease**			**Not riding horse with Bran disease**			**Bothering about feed quality**		
		RTC	No R	Yes	No	No R	Pity	Others	No R	Yes	No	No R
Igabi	7	7(100%)	0	1(14.3%)	6(85.7%)	0	4(57.1%)	2(28.6%)	0	7(100%)	0	0
Zaria	7	7(100%)	0	2(28.6%)	5(71.4%)	0	5(71.4%)	1(14.3%)	0	3(42.9%)	4(57.1%)	0
SG	7	5(100%)	0	3(60.0%)	2(40.0%)	0	0	2(40.0%)	0	3(60.0%)	2(40.0%)	0
Total	**21**	**19 (90.5%)**	**2 (9.5%)**	**6 (28.6%)**	**13 (61.9%)**	**2 (9.5%)**	**9 (42.9%)**	**5 (23.8%)**	**7 (33.3)**	**13 (61.9%)**	**6 (28.6%)**	

Key: No R: No response, SG: Sabon Gari, RTC: Report to clinic

Comment [SS1]: Area needing improvement

Table 4.10: Grooms' practices with regard to *Osteodystrophia fibrosa* in three Local Government Areas of Kaduna State, Nigeria

LGA	No. Grooms	Actions to prevent Bran disease			Actions for prevention			Action for control			
		Yes	No	No response	Change feed	Medication/Supplement	No response	Calcium supplement	Bone meal	Others	No response
Igabi	7	4(57.1%)	3(42.9%)	0	0	4(57.1%)	3	2(28.6%)	2(28.6%)	3(42.9%)	0
Sabon Gari	7	7(100%)	0	0	4(57.1%)	3(42.9%)	0	3(50%)	3(50%)	0	1
Zaria	7	4(80.0%)	0	3	2(40.0%)	3(60.0%)	2	2(40%)	3(60%)	0	2
Total	**21**	**15 (71.4%)**	**3 (14.3%)**	**3 (14.3)**	**6 (28.6%)**	**10 (47.6%)**	**5 (23.8%)**	**7 (33.3%)**	**8 (38.1%)**	**3 (14.3%)**	**3 (14.3%)**

CHAPTER FIVE

5.0 DISCUSSION

A number of stables were encountered along with horses ofdifferent breeds that included Argentine, West African Barb and Sudanese. Other studies have shown that horses were greatly put into use as previously reported by Mshelia (2012), Musa (2013) and Baba (2016). The vital parameters (respiratory rate, pulse rate and rectal temperature) of the horses under were within normal ranges when compared with the figures stated by Sambo(2015) and Saidu *et al.* (2018). This seems to indicate that *Osteodystrophia fibrosa* does not significantly influence vital parameters of affected horses.This has been similarly observed by workers like Freestone and Seahorn (1993). It could be that the condition, being non-infectious, does not affect these parameters. There is scanty information on the condition in literature as to allow for good comparism, especially with regard to vital parameters as to guide clinicians on what to expect in clinical cases in the field.

The difference in calcium levels of the clinically diseased horses wasstatistically significant ($P<0.05$) when compared to non-clinically diseased horses. This could be as a result of the type of feed being fed to the horses along with the general management practice in the stables under study. Swartzmann *et al.*(1978) reported that feeds high in oxalate tend to result in net losses in calcium and phosphorus levels and are excreted through faeces. Furthermore, the grooms, in a lot of cases feed their horses with feeds that wereof low quality and this is especially seen during the dry season when feeds are scanty. However, in a situation where horse owners provide salt lick to their horses may tend to alleviate the condition, if present.

The parathyroid hormone levels were also statistically significant ($p<0.05$) between clinically diseased and non-clinically diseased horses.This may bedue to the fact that PTH is responsible for the minute-to-minute regulation of extracellular calcium (Ca^{2+}) concentrations. The relationship between serum Ca^{2+} concentrations and PTH is inverse and sigmoidal in the horse (Aguilera-Tejero *et al.*, 1998; Toribio *et al.*, 2001). This enables the parathyroid gland to respond rapidly to minimal changes in Ca^{2+} concentrations.

The difference in the calcium and phosphorous levels was not statisticallydifferent ($p>0.05$) based on the sex of the horses in this study. However,the diseased male and female horses had slightly lower calcium levels. The PTH levels were also higher in the diseased male and female horses but that of the male was higher. It could be that the male horses were more active, judging by their roles in polo and racing as suggested by Baba (2019).

The West African Barb and Sudanese breeds of horses had lower calcium levels and higher parathyroid hormone levels in the diseased horses and this could also be ascribed to the fact that, these breeds were not adequately fed when compared with the Argentine breed of horses that were managed and owned by more organized farms ($p<0.05$).

Based on age group, the clinically diseased horses within the age brackets of 1-5years and 6-10years had slightly lower calcium levels. This could be associated with the fact that the younger horses were most susceptible to the condition with more pronounced lesions compared to the adults. This may be because of their need for more calcium requirement for growth development. This agrees with the report of Ospina *et al.* (2014) who reported that the condition is seen more in horses of 2 to 8years of age. It could also be that the owners were not particular in caring for them as they might have been more interested in

feeding theolder ones that could be used immediatelyfor racing or polo as Baba (2019) reported horse owners in Kano Metropolis being more particular in feeding those horses in active service.

The parathyroid hormone levels for the clinically diseased horses of age groups 1-5years, 6-10years, and 11-15years were high. This could bedue to thedecrease in calcium level. It was reported by Steward (2010) that increasing age does not affect the condition.

The haematological parameters as seen in this study were within normal ranges,there was no significant difference between the clinically and non-clinically diseased horses as the values were within the normal ranges. It could be that this condition may not haveeffect on blood parameters at the subclinical stage. In view of the fact that there is limited information in the literature of the condition.

The faecal phosphorous levels of the horses in the three LGAs sampled by sex, breed and age were mostly high in relation to the calcium levels of all the sampled horses. This could be attributed to the feed they were fed on. The feeds identified which were fed to the horses, especially in the Zaria and Sabon Gari LGAs had high levels of oxalate and phytate which could bind the calcium leading to the manifestation of the condition. This could be associated with the time of harvest of the grasses and high level of bran being fed to the horses.

From the study the grooms had primary, secondary and tertiary education. Thus, their ability to recognize horse diseases is expected. Other researchers had also reported similar educational status by horse grooms (Baba, 2016, 2019; Edeh, 2018). It is expected that horse grooms with these levels of educationwould be in a position to handle disease

situations better than uneducated ones. Their knowledge of the condition is expected along with their ability to proffer solutions to the condition of the horses as seen in this study. Theirmentioning parents and friends as sources of knowledge on the condition means there is a strong linkage in traditional oral knowledge of doing things as reported by other workers (Kaltungo, 2013; Baba, 2016).Furthermore,Saidu *et al*. (2018) reported that clinicians in Veterinary practice in Nigeria found it better to interact with educated clients than illiterate ones. This could also explain how they were able to recognize the condition and even its clinical manifestations. Their mentioning Veterinarians as one of their sources of knowledge of the condition is understandable as Kaltungo (2013), Buhari (2014) and Baba (2016) all reported similar findings during their respective studies on Brucella.

The ability of the grooms to report the case to veterinarians and also considered the quality of the feed being fed to the horses showed they had good attitude towards the condition. They also said they tried to control the condition by supplementing the feed using calcium supplements and bone meal. The study also observed that some of the stables gave salt lick as supplement.This showed their good knowledge, attitude and practice on the condition but there was paucity of reports and proper management practice from the field in Nigeria. Other workers have variously reported livestock and horse owners to have good knowledge of animal diseases. For example, Baba (2016; 2019) reported horse owners being fully aware of a number of horse diseases and even referred their sources of knowledge on horse diseases to veterinarians, friends, and parents as seen in this study. Similarly, Kaltungo (2013, 2018), in studies to determine the epidemiology of brucellosis in small ruminants, reported small ruminants' owners having good knowledge of diseases and even using

traditional medicine in the treatment of the disease. Baba (2019) similarly reported horse owners and grooms using traditional plants in treating horse diseases.

The attitude of the grooms in this study was seen to reflect their educational status as riding a horse with the condition could adversely affect the horse. The practice of the grooms in preventing the occurrence of the condition could be due to their levels of educational.

CHAPTER SIX

6.0 CONCLUSIONS AND RECOMMENDATIONS

6.1 Conclusions

1. *Osteodystrophia fibrosa*was seen to occur in the study area at 74.6% occurrence rate.
2. The calcium and phosphorus levels in *Osteodystrophia fibrosa* clinically diseased and non-clinically diseased horses was statistically significant ($p<0.05$).
3. The parathyroid hormone of clinically diseased horses and non-clinically diseased horses was statistically significant ($p<0.05$)
4. The oxalate and phytate levels in horse feeds were found to be 0.02 and 0.51, 0.77m/100kg respectively.
5. The faecal calcium level was highest 75.89±6.55 mg/kg in horses in Igabi LGA for the non-clinically diseased horses and 82.85 ± 4.17 mg/kg for the clinically diseased horse while the faecal phosphorous value for the clinically diseased horses was also highest 2801.76 ± 212.51 mg/kg in horses in Igabi LGA
6. The grooms very good knowledge, attitude and practice of *Osteodystrophia fibrosa*but the condition was still present in the area because of the quality of feeds available to them and also due to cost of feed.

6.2 Recommendations

1. There is the need to do more work by conducting wider study in determining the role of calcium, phosphorus, parathyroid hormone, haematological values as well as the clinical signs characteristic of the disease in clinically diseased horses as to enable its use for clinical diagnosis of the condition.
2. There is the need to examine more horse feeds with a view to determine their oxalate and phytate levels in order to ascertain their roles in the causation of the condition in Nigeria.
3. There is the need to conduct feeding trials with known feeds containing oxalate and phytate as to confirm their roles in the causation of the condition.
4. There is the need to conduct a nationwide study as to determine the prevalence of the condition in Nigeria as well as to understand horse owners and groom's knowledge, attitudeand practices with regard to the condition.
5. There is the need to conduct year-round study with a view to determine the seasonal occurrence of the conditionin Nigeria.

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Appendices

Appendix I: QUESTIONNAIRE

Department of Veterinary Medicine. Ahmadu Bello University, Zaria. Novermber,2019.

Researcher: Dr. Rukaya Altine NASIRU, MSc. Equine Medicine.

Date: _____

Questionnaire No: _____

SECTION A: Biodata of Horse owners/Grooms

1. L.G.A: _____
2. Ward:_____
3. Name of Owner (optional) _____
4. Educational level: primary ( ) Secondary ( ) Tertiary ( )
5. Stable size: _____
6. Breed of horse _____
7. Sex: Male ( ) Female ( )

SECTION B: Knowledge

8. Do you know about osteodstrophia fibrosa (kumarmari)? YES ( ) NO ( )

9. How did you get to known about the disease A) veterinarian ( ) B) Extension workers ( ) C) Radio D) Tv ( ) E) Friends ( )

How do you recognize the disease? A) Swollen face ( ) B) lameness ( ) C) Emmaciation ( ) D) Others (specify) _____

SECTION C: Attitude

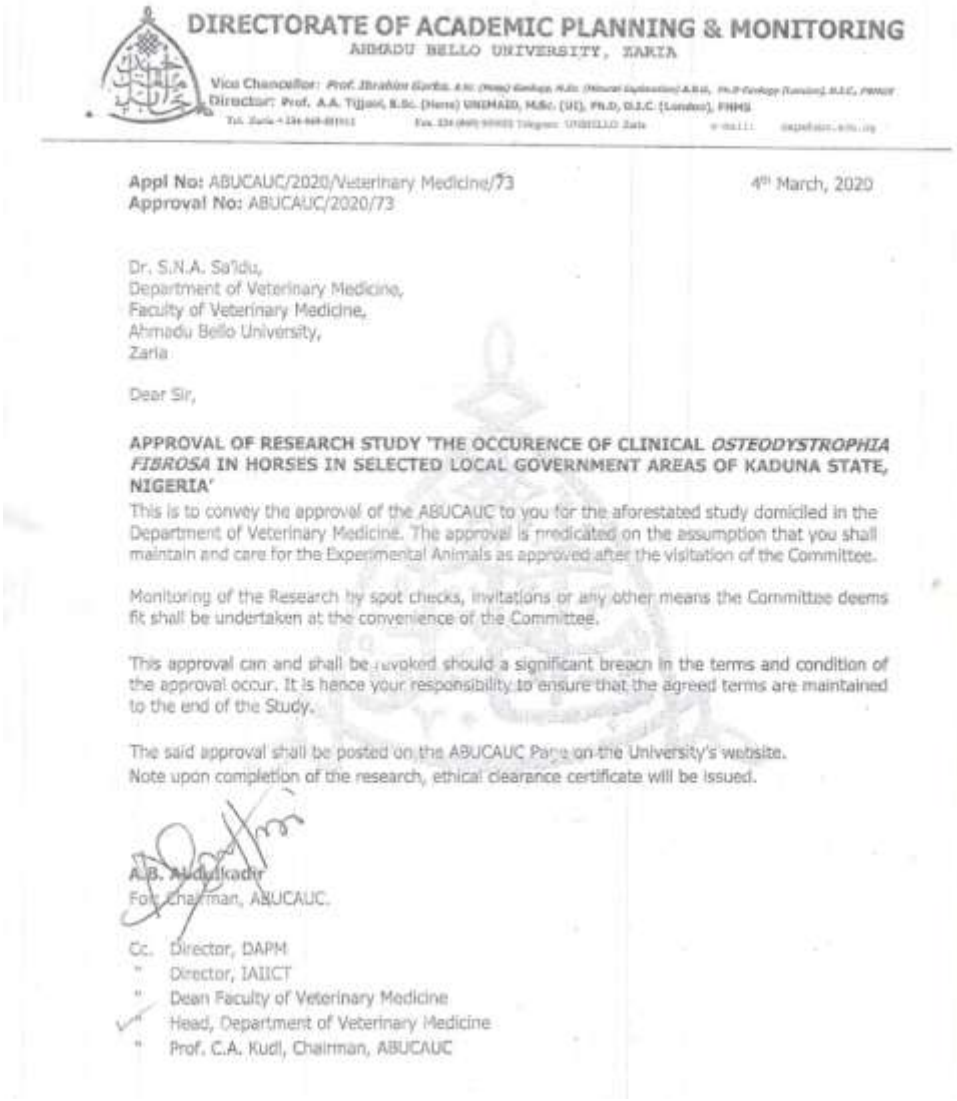
10. What do you do if your horse develops this condition A) Report to the clinic B) Use traditional medication C) Do nothing.
11. Do you ride your horse with this condition Yes ( ) No ( )
12. If NO, why A) Pity for it B) Death
13. Do you bother about the feed quality against this condition A) Yes ( ) B) No ( )

SECTION D: Practice

14. Do you do anything to prevent your horses from coming down with this condition? YES ( ) NO ( )
15. If answer YES, what do you do? A) Change feed B) Medicate horses C) Dispose sick horses D) Do nothing
16. What do you use to control the condition? A) Use calcium supplement ( ) B) Bone meal ( ) C) Good feeds ( ) D)Others (specify)_____

THANK YOU.

Appendix II: Evidence of Ethical Clearance for the Study



DIRECTORATE OF ACADEMIC PLANNING & MONITORING
AHMADU BELLO UNIVERSITY, ZARIA

Vice Chancellor: *Prof. Ibrahim Garba, B.Sc. (Hons) Geology, M.Sc. (Natural Exploration) A.B.U., Ph.D Geology (London), D.I.C., FNMGS*
Director: Prof. A.A. Tijjani, B.Sc. (Hons) UNIMAID, M.Sc. (UI), Ph.D, D.I.C (London), FNMS
Tel: Zaria +234-069-881111 Fax: 234-(069) 50003 Telegram: UNIBELLO Zaria e-mail: dapm@abu.edu.ng

Appl No: ABUCAUC/2020/Veterinary Medicine/73 4th March, 2020
Approval No: ABUCAUC/2020/73

Dr. S.N.A. Sa'idu,
Department of Veterinary Medicine,
Faculty of Veterinary Medicine,
Ahmadu Bello University,
Zaria

Dear Sir,

APPROVAL OF RESEARCH STUDY 'THE OCCURENCE OF CLINICAL *OSTEODYSTROPHIA FIBROSA* IN HORSES IN SELECTED LOCAL GOVERNMENT AREAS OF KADUNA STATE, NIGERIA'

This is to convey the approval of the ABUCAUC to you for the aforestated study domiciled in the Department of Veterinary Medicine. The approval is predicated on the assumption that you shall maintain and care for the Experimental Animals as approved after the visitation of the Committee.

Monitoring of the Research by spot checks, invitations or any other means the Committee deems fit shall be undertaken at the convenience of the Committee.

This approval can and shall be revoked should a significant breach in the terms and condition of the approval occur. It is hence your responsibility to ensure that the agreed terms are maintained to the end of the Study.

The said approval shall be posted on the ABUCAUC Page on the University's website.
Note upon completion of the research, ethical clearance certificate will be issued.

A.B. Abdulkadir
For: Chairman, ABUCAUC.

Cc. Director, DAPM
" Director, IAIICT
" Dean Faculty of Veterinary Medicine
" Head, Department of Veterinary Medicine
" Prof. C.A. Kudi, Chairman, ABUCAUC

Appendix III: Distribution of sampled horses from the study areas

	Total numbers sampled	**Male**	**Female**	**Argentine**	**Sudanese**	**W/A Barb**	**1-5years**	**6-10years**	**11-15years**	**16-20years**
Igabi	19	6	13	19	0	0	1	3	6	7
Zaria	20	14	6	5	11	4	5	8	5	2
Sabon Gari	20	8	12	1	10	9	5	10	4	3
Total	**59**	**28**	**31**	**25**	**21**	**13**	**11**	**21**	**15**	**12**

Appendix IV: Mean (±SEM) levels of calcium and phosphorous in clinically and non - clinically bran diseased horses in the three selected local government areas inKaduna state.

LGA	Ca(mmol/L)		Ph (mmol/L)	
	NC	C	NC	C
Igabi	2.66 ± 0.05	2.71 ± 0.03	1.02 ± 0.73	1.09 ± 0.05
Zaria	2.82 ± 0.02[a]	2.47 ± 0.06[b]	1.18 ± 0.05	1.23 ± 0.07
Sabon Gari	2.81 ± 0.03	2.42 ± 0.07	1.20 ± 0.11	1.26 ± 0.08

Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased.
[a,b]Means with different letters across rows are significantly different ($p < 0.05$).

Appendix V: Mean (±SEM) levels parathyroid hormones in clinically and non -clinically bran diseasedhorses in the three selected local government areas in Kaduna state.

LGA	PTH (pg/ml)	
	NC	C
Igabi	38.9 ± 3.50	44.1 ± 4.47
Zaria	34.84 ± 3.58[a]	93.01 ± 20.44[b]
Sabon Gari	34.49 ± 2.35	81.01 ± 6.37

Key: PTH= Parathyroid hormone, Nc=Non-clinically diseased, C=clinically diseased. [a,b]Means with different letters across rows are significantly different ($p < 0.05$).

Appendix VI: Mean (±SEM) levels of calcium and phosphorous in clinically and non - clinically brandiseased horses based on sex.

Sex	Male		Female	
	C	NC	C	NC
Ca(mmol/L)	2.54± 0.05	2.76± 0.03	2.53± 0.05	2.26± 0.04
Ph(mmol/L)	1.20± 0.06	1.10± 0.09	1.20± 0.06	1.16± 0.07

Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased

Appendix VII: Mean (±SEM) levels of parathyroid hormones in clinically and non - clinically bran diseased horses based on sex.

Age	Male		Female	
	C	NC	C	NC
PTH	84.18± 13.80	31.99± 3.72	62.10± 6.19	37.90± 1.92

Key: PTH= Parathyroid hormone, Nc=Non-clinically diseased, C=clinically diseased

Appendix VIII: Mean (±SEM) levels of calcium and phosphorous in clinically and non - clinically brandiseased based on breed.

Breed	Argentine		W/A Barb		Sudanese	
	C	NC	C	NC	C	NC
Ca(mmol/L)	2.67± 0.04[a]	2.73± 0.04	2.48± 0.06[b]	2.84± 0.03	2.42± 0.08[b]	2.80± 0.05
Ph(mmol/L)	1.15± 0.06	1.09± 0.06	1.16± 0.07	1.33± 0.15	1.34± 0.07	1.07± 0.03

Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased.
[a,b]Means with different letters across rows are significantly different ($p < 0.05$).

Appendix IX: Mean (±SEM) levels of parathyroid hormones in clinically and non - clinically bran diseased horses based on breed.

Breed	Argentine		W/A Barb		Sudanese	
	C	NC	C	NC	C	NC
PTH	46.71± 4.90[a]	36.56± 2.72	91.72± 12.77[b]	36.59± 1.77	84.17± 22.00[b]	34.13± 4.18

Key: PTH= Parathyroid hormone, Nc=Non-clinically diseased, C=clinically diseased. [a,b]Means with different letters across rows are significantly different ($p < 0.05$).

Appendix X: Mean (±SEM) levels of calcium and phosphorous in clinically and non - clinically brandiseased horses based on age.

Age	1-5years		6-10years		11-15years		16-20years	
	C	NC	C	NC	C	NC	C	NC
Ca(mmol/L)	2.43± 0.69	2.88± 0.02	2.51± 0.06	2.84± 0.03	2.62± 0.07	2.69± 0.04	2.72± 0.06	2.75± 0.07
Ph(mmol/L)	1.23± 0.11	1.10± 0.06	1.27± 0.06	1.33± 0.15	1.08± 0.08	1.08± 0.10	1.13± 0.05	1.08± 0.07

Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased

Appendix XI: Mean (±SEM) levels of parathyroid hormones in clinically and non - clinically bran diseased horses based on age.

Age	1-5years		6-10years		11-15years		16-20years	
	C	NC	C	NC	C	NC	C	NC
PTH	60.45± 9.62[a]	34.84± 4.41	96.91± 16.66[b]	36.59± 1.77	63.01± 5.75[a]	34.86± 3.15	34.44± 1.74[c]	37.49± 4.45

Key: PTH= Parathyroid hormone, Nc=Non-clinically diseased, C=clinically diseased. [a,b,c]Means with different letters across rows are significantly different ($p < 0.05$).

Appendix XII: Mean (±SEM) calcium and phosphorous levels in faeces of clinically and non-clinically bran diseased horses in the three selected Local Government Areas in Kaduna State.

	Ca (mg/kg)		Ph (mg/kg)	
LGA	Nc	C	Nc	C
Igabi	75.89 ± 6.55	82.85 ± 4.17	2488.72 ± 256.30	2801.76 ± 212.51
Zaria	73.69 ± 15.82	62.09 ± 10.38	2319.1 ± 332.01	2556.61 ± 224.27
Sabon Gari	66.34 ± 17.70	38.88 ± 7.96	2554.5 ± 207.38	2585.84 ± 178.26

Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased

Appendix XIII: Mean (±SEM) calcium and phosphorous levels in faeces of clinically and non-clinically bran diseased horses based on sex.

	Male		Female	
	C	NC	C	NC
Ca	57.22± 8.19	76.03± 16.17	64.69 ±6.66	73.82± 8.88
Ph	2705.64± 172.38	2235.50± 197.95	2577.70± 160.01	2501.11± 205.30

Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased

Appendix XIV: Mean (±SEM) calcium and phosphorous levels in faeces of clinically and non-clinically bran diseased horses based on breed.

Biochemical element	**Argentine**		**W/A barb**		**Sudanese**	
	C	NC	C	NC	C	NC
Ca	88.84 ±5.54	85.33± 5.19	47.09± 6.65	54.42± 20.87	40.55± 11.63	49.46± 25.07
Ph	2773.42± 195.50	2334.45± 209.97	2625.10± 203.43	2516.33± 349.46	2473.50± 199.08	2750.86± 189.08

Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased

Appendix XV: Mean (±SEM) calcium and phosphorous levels in faeces of clinically and non-clinically bran diseased horses based on Age.

Biochemical element	**Age**	**1-5years**	**6-10years**	**11-15years**	**16-20years**
Ca	C	62.26± 14.33	53.03± 6.88	64.83± 10.71	80.47± 7.28
	NC	45.08± 20.83	54.42± 20.87	82.81± 8.36	89.97± 7.56
Ph	C	2736.42± 257.95	2442.98± 150.59	2901.34± 258.81	2593.15± 481.48
	NC	2484.86± 376.95	2516.33± 349.46	2499.71± 216.48	2361.84± 340.09

Key: Ca=calcium, Ph=phosphorous, Nc=Non-clinically diseased, C=clinically diseased

Appendix XVI: Horse with facial enlargement in Zaria city



Appendix XVII: Facial swelling in Igabi LGA



Appendix XVIII: A body score condition of 2/5in a horse with *Osteodystrophia fibrosa*



Appendix XIX: Horses housed in Zaria city



Appendix XX: Bags of Soyabean hull fed to horses at Sabon Gari LGA



Appendix XXI: *Osteodystrophia fibrosa* skull

Picture gotten from necropsy laboratory, Department of Pathology, Faculty of Veterinary Medicine Ahmadu Bello University Zaria



Appendix XXII: *Osteodystrophia fibrosa* skull

Picture gotten from necropsy laboratory, Department of Pathology, Faculty of Veterinary Medicine Ahmadu Bello University Zaria

