Aetiology of Chronic Emaciation: Trematodiasis and Secondary/Concurrent Organ Compromise in Off-Take Cattle in Ibadan Metropolis

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ABSTRACT

Aims: The primary aim of the investigation was to determine the trend and most significant cause of chronic emaciation and cachexia in off take cattle in Ibadan metropolis; and evaluate clinico-pathologic findings.

Study Design: The study was a prospective one. In this particular study, causative factors of chronic emaciation were examined. The purposive sampling technique was used for this study. Samples were collected from 100 chronically emaciated off take cattle of different breeds, sexes and ages, not less than 2 years old.

Place and Duration of Study: The study was carried out at the Veterinary Teaching Hospital of the University of Ibadan, Nigeria, between September, 2019 and December, 2019.

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<table>
<thead>
<tr>
<th>Methodology:</th>
<th>The diagnostic protocol of complete physical examination; and comprehensive laboratory investigations such as parasitology, haematology, serum chemistry and urinalysis, etc., were followed as a minimum.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Results:</td>
<td>The study revealed a trend of aetiologies of emaciation with trematodiasis due to <em>Fasciola hepatica</em> and <em>Dicrocoelium dendriticum</em> as primary causes. Secondary and concurrent organ dysfunctions were important findings. The haematologic parameters of packed cell volume (PCV), haemoglobin (Hb) concentration, mean corpuscular volume (MCV), mean corpuscular haemoglobin concentration (MCHC), neutrophil and platelet count were statistically significant (p&lt;0.05) with differences between the mean values of emaciated cases and control subjects. The serum chemistry parameters of albumin, globulin, bilirubin, aspartate amino transferase (AST) and gamma glutamyl transferase (GGT) were statistically significant (p&lt;0.05) with differences between the mean values of emaciated cases and control subjects.</td>
</tr>
<tr>
<td>Conclusion:</td>
<td>The study established trematodiasis with secondary or concurrent organ compromise as one of the important aetiologies of chronic emaciation and cachexia in cattle in Ibadan metropolis. The increasing prevalence of dicrocoeliasis as revealed in the study should be of epidemiological and clinical relevance to livestock health institutions and large animal practitioners in northern Nigeria where these animals are sourced.</td>
</tr>
</tbody>
</table>

Keywords: Aetiologies; chronic emaciation; cachexia; trematodiasis; organ compromise; cattle; Ibadan metropolis.

1. INTRODUCTION

The clinical problem of weight loss or emaciation suggests that an individual large animal patient or a herd has lost weight over a known period of time [1].

Emaciation is not a disease but rather a symptom that can be found in many diseases either singly or in combination with other unspecified clinical signs [2]. Chronic emaciation however reflects the severity and duration of the aetiologic associated disease condition [3].

The subject of chronic emaciation is broad as there are multifactorial causes, and with a progression to syndrome of cachexia. Established causes of chronic emaciation in cattle include chronic bacterial infections such as tuberculosis [4,5], paratuberculosis (Johnne’s Disease) [6,7,18], actinomycosis, actinobacillosis, nocardiosis, corynebacteriosis [9,10,11,12,13,7,14,15,1,16] etc.; chronic inflammations such as endocarditis, lymphadenitis, pleuritis, pericarditis and enteritis etc. [7,1]; parasitic infections such as trypanosomiasis, toxoplasmosis, chronic babesiosis [17,18,19,20,21,22,23], anaplasmosis, besnoitiosis [24,18], sarcosporidiosis [24,25,26], intestinal coccidiosis [27] and chronic helminthiasis [28,18] etc.; hepatic and renal conditions associated with several causes [29,30,7,1,31]; gastro-intestinal disorders such as infiltrative diseases associated with intestinal malassimilation [18,7,1,32]; immune mediated conditions such as immune mediated haemolytic anaemia (IMHA) [33] and those associated with amyloid deposition in various organs and hyperglobulinemia, consequent upon chronic infection/inflammation or neoplasm [7]; congenital disorders such as congenital cardiac malformations and congenital renal disease, which creates physiologic inefficiencies that requires energy beyond the body's ability to supply it [1]; endocrine dysfunction such as diabetes mellitus [34]; severe tick infestation [18]; skin conditions such as dermatophilosis, dermatomyositis and mange [7,35]; neoplasms such as lymphosarcoma and carcinomas [36,37,38]; and primary undernutrition [39,1] etc.

A major cause of chronic emaciation and cachexia in cattle which has elicited interest in these researchers is hepatobiliary disease associated with trematode infection caused by *Fasciola sp.* and *Dicrocoelium sp.*

*Fasciola hepatica* and *F gigantica* are common liver flukes of cattle. The life cycle commences with the hatching of ova to miracidia under suitable environmental conditions. The miracidia are acquired by lymnaeid snails, the intermediate hosts, and undergo asexual development and multiplication through the stages of sporocysts, rediae, daughter rediae, and infective cercariae. The infective cercariae migrate onto wet herbage, encysting as metacercariae, the highly resilient infective stage of liver fluke. The infective metacercariae are acquired by cattle during

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grazing on pastures. Following ingestion, young flukes excyst in the duodenum, penetrate the intestinal wall, and enter into the peritoneal cavity, following which they invade the liver, wander as immature flukes and invade the bile duct [40,41].

Aged adult cattle are reported to have a higher prevalence of fascioliasis than young subjects [42]. Chronic cases can be seen in all seasons but manifests primarily in late fall and winter [40].

However, the trematode *Dicrocoelium dendriticum* presents with a different life cycle. There are two intermediate hosts; the first is a snail, while the second is an ant, *Formica fusca* [41].

In trematodiasis of a chronic course caused by *Fasciola hepatica* and *F. gigantica* or giant fluke in cattle, clinical findings include anaemia, unthriftiness, submandibular edema, fibrosis or cirrhosis of the liver, enlargement of duct wall, or even cystic, and thickened fibrosed walls; however, in acute infection, the liver is enlarged, and migratory tracts can be seen [29,41]. Associated array of clinical findings of hepatic dysfunction may be consequent upon severe liver damage by flukes. Further reading: [43,7,1].

Anorexia is a major occurrence in cattle with chronic infection with *Fasciola* sp or *Dicrocoelium* sp.; and it is the primary mechanism for weight loss [18]. Weight loss results from decreased nutrient intake, when partial anorexia occurs over a long period [18]. Anorexia is usually associated with a primary disease condition (such as fascioliasis or dicrocoeliasis), and is regulated by cytokines, including interleukin (IL) - 1 and tumour necrosis factor alpha (TNF-α), released during an inflammatory response [1]. These inflammatory cytokines directly cause anorexia, increases lean body mass loss and may increase energy requirements [44,3]. Chronic emaciation or cachexia appears to be a response to increased catabolism with either normal or decreased appetite [45].

Diagnostic investigation of trematodiasis associated with chronic emaciation or cachexia should follow the conventional protocol of comprehensive history taking, careful physical examination [46,18,7,1], laboratory examination of relevant samples such as parasitology, haematology, serum chemistry and serology etc. [47,48,49,50,51,52,53,54,18,55], to provide a diagnosis and evaluation findings of the infection; and rule out other possible causes of emaciation. Faecal examination by zinc sulphate centrifugation-flotation technique usually reveals fluke ova in faeces [56].

Haematology reveals anaemia in severely infected and emaciated cattle. In liver fluke infection, serum γ-glutamyl transferase (GGT) concentrations are increased with bile duct damage especially during the late maturation period when flukes are in the bile ducts [40]. Serum liver enzymes such as sorbitol dehydrogenase (SDH), arginase, ornithine carbamoyltransferase (OCT), aspartate amino transferase (AST), isoenzyme 5 lactate dehydrogenase (LDH-5), and alkaline phosphatase may be altered in hepatic dysfunction that may also be associated with fluke infection [43,54].

Further evaluation of the liver by ultrasonography for imaging, biopsy and guidance for aspiration of bile duct to detect flukes supports diagnosis and also reveals the degree of hepatobiliary compromise [43,7,1]. Liver biopsy may be a useful procedure to determine the type of pathology, degree of hepatic fibrosis present and regenerative capabilities of the liver parenchyma [43].

An average of 450 cattle is slaughtered at the central abattoir located at Amosun in Ibadan. Post mortem records indicate partial condemnation of carcasses with organ involvement; and this is of economic significance. Besides carcass losses and reduced meat quality due to distinct organoleptic changes, it can be imagined that there would have been production losses when these animals were alive [57,58].

Relatively few studies have been carried out to investigate emaciation in cattle related to some specific disease causes such as the one carried out by Raji et al. [59] to examine pathological lesions of organs of 7812 cattle slaughtered in Zaria abattoir.

The main aim of this study therefore, was to investigate the trend of aetiologies of chronic emaciation in various breeds of cattle in Ibadan metropolis, Nigeria, using standard diagnostic protocol to evaluate clinico-pathological abnormalities.
2. MATERIALS AND METHODS

2.1 Scope and Design

The study was a prospective one. In this particular study, causative factors of chronic emaciation were examined.

The purposive sampling technique was used for this study. Thus, samples were collected from only chronically emaciated cattle presented at the central slaughter house in Ibadan metropolis.

A total number of 100 cattle of different breeds, sexes and ages not less than 2 years old and of body condition score between 1 and 3 were selected for the study. However, there were 50 animal control samples. The control animals were apparently healthy and of body condition score from 6 and above. The sample size was calculated using the statistical programme, G-Power® version 3.1.1; Germany.

2.2 Sampling

The various samples for which data were derived include; the animal, blood, serum, faeces, urine, skin scrapings and fluid aspirates. These samples including the cattle were collected from the central slaughter house in Ibadan metropolis; and within 5 km radius from laboratory where investigations were carried out. The samples were collected over a period of 3 months. Consent to conduct physical examination and collect various samples from the animals were obtained from the owners after explaining the purpose of the study. Subject data was captured on a preprinted form.

Each animal was subjected to a thorough physical examination; and basic parameters including temperature, respiratory rate, pulse and body condition score were taken and recorded. Abnormal systemic findings observed were noted.

Blood samples were taken from all the animals; for parasitology and haematologic evaluation. The sampling procedure was as described by Baker and Silverton [48].

Serum samples were retrieved from blood samples collected from all the animals for chemistry evaluation. The sampling procedure was as described by Baker and Silverton [48].

Faeces of animals were collected for various examinations including parasitology, cytology and occult blood examination. The sampling procedure was as described by Baron [60]; and CDC [61].

Urine of each animal was collected for urinalysis by stroking the escutcheon in the female, and by massaging of the preputial orifice in the male. The voided urine was collected into a clear tube for urinalysis and sediment examination as described in Merck literature [18].

Scrappings of the skin of animals with lesions were carried out for dermatologic investigations. These were done using sharp scalpel blades to scrape the skin near and around observed lesions, following drops of glycerine oil; the scrapings including hairs and scales were transferred into clean glass slides as described in Merck literature [18].

2.3 Procedure

2.3.1 Epidemiological data sourcing

Various data were obtained from responses of cattle merchants and physical observation. History related to subjects such as sources, environmental conditions, husbandry practices and abnormal symptoms were sought by oral questioning of cattle merchants. The age, sex and breed of the animals were noted from physical observation. Breeds of all the cattle were identified using morphological features which distinguishes them such as horn shape, body size and coat color as described by Kugonza et al. [62]; and Terefe et al. [63]. Age was determined using the technique of dentition as described by Lasisi et al. [64].

2.3.2 Physical examination

Each animal was subjected to a thorough physical examination beginning with a distant and followed by a close examination as described by Radostits et al. [7]. Abnormalities observed related to animal's condition were recorded.

2.3.3 Laboratory investigations

2.3.3.1 Blood smear examination (thin films)

A clean slide was wiped with alcohol and a very small drop of blood placed near the end of the slide. Another clean and disinfected slide
was used to spread the drop of blood along the sample slide to form a thin smear. The slide was air dried, fixed briefly in methanol and stained with giemsa stain. The slide was then examined under the microscope (magnification up to 1000 x) to identify protozoan organisms (e.g. Babesia Sp, Trypanosoma Sp) and Rickettsial organisms (e.g. Anaplasma bovis). This method was as described in Merck literature [18].

2.3.3.2 Buffy coat examination

This concentration technique was used for trypanosome identification as described in Merck literature [18]. Blood was taken up from one end of the haematocrit tube, and the other open end was sealed with a burner. The sealed tube containing heparinized blood was placed in the groove of the rotor plate with the sealed end outwards; and spun at a high speed 12,000 rpm for 5 minutes. After centrifugation the tube was removed and buffy coat examined for the presence of trypanosomes by making a smear of this area. The slide was air dried, fixed in methanol and stained with giemsa stain before examining under microscope (100 x).

2.3.3.3 Haematology

Haematologic procedure as described by Baker and Silverton [48] include procedures to determine packed cell volume, haemoglobin level, red cell count, red cell indices, white cell count, platelet count; and blood glucose and ketone body concentration.

The PCV was determined using a microhaematocrit tube, a specifically designed centrifuge and a reader as described by Baker and Silverton [48].

Haemoglobin concentration was determined by the cyanmethaemoglobin method for measuring haemoglobin concentration in test blood samples as described by Baker and Silverton [48].

Red cell count of blood sampled from each animal was performed by visual method using the improved Neubauer counting chamber as described by Baker and Silverton [48].

White cell count of sampled blood was achieved by visual method using the improved Neubauer counting chamber as described by Baker and Silverton [48].

Platelet Count was performed by visual method using the improved Neubauer counting chamber as described by Baker and Silverton [48].

Blood glucose concentrations of test samples were determined using manual technique of Trinders glucose oxidase method (using phenol and 4 - Aminophenazone) as described by Baker and Silverton [48].

2.3.3.4 Serum biochemical analysis

Serum total proteins, albumins and globulin fractions were determined with the manual biuret method as described by Baker and Silverton [48]. All stages were carried out to the point at which the colour was developed with the biuret reagent, at a temperature above 25°C to avoid crystallization of sodium sulphate. This was achieved by precipitating the globulins in a centrifuge tube placed inside wider centrifuge tube containing water warmed to about 30°C and centrifuging both tubes.

Serum urea concentration was determined using the urease method which uses the Berthelot reaction as described by Baker and Silverton [48].

Serum creatinine concentration was determined with the manual method in samples collected as described by Baker and Silverton [48]. The method is the Jaffe reaction which produces a red colour with an alkaline picrate solution.

Serum gamma glutamyl transferase (GGT) concentration was determined using a commercial colorimetric assay kit [65]. GGT activity is determined by a coupled enzyme assay, in which the GGT transfers the γ-glutamyl group from the substrate L-γ-Glutamyl-p-nitroanilide, liberating the chromogen p-nitroanilide (pNA) at 418 nm, proportional to the GGT present. One unit of GGT is the amount of enzyme that will generate 1.0 μmole of pNA per minute at 37°C.

Serum aspartate aminotransferase (AST) concentration was determined with the use of a kinetic method as described by Baker and Silverton [48]. AST catalyzes the transfer of the amino group aspartate to 2-oxoglutarate to yield oxaloacetate and glutamate. The oxaloacetate formed in the first reaction is then reacted with reduced nicotinamide adenine dinucleotide (NADH) in the presence of malate
dehydrogenase (MDH) to form nicotinamide adenine dinucleotide (NAD). AST is determined by measuring the rate of oxidation of NAD at 340 nm.

Serum creatine kinase (CK) concentration was determined using a commercial colorimetric assay kit [66]. In this assay, creatine kinase activity is determined by a coupled enzyme reaction resulting in the production of reduced nicotinamide adenine dinucleotide phosphate (NADPH), measured at 340 nm, proportionate to the CK activity present in the serum sample.

Serum bilirubin concentration in test samples was determined using the method of Powell (1944) as described by Baker and Silverton [48]. The 'accelerating agent', urea-sodium benzoate solution cause bilirubin to react with the diazo reagent at low dilution without precipitating proteins; and the resulting colour when compared with a suitable standard gave a measure of total bilirubin in serum.

2.3.3.5 Urinalysis

The procedures for urinalysis in the study carried out include; physical examination of urine, use of colorimetric test pads for several semi quantitative chemical evaluation and microscopic examination of urine sediment. These procedures were as described in Merck literature [18] and by Parrah et al. [67].

2.3.3.6 Faecal microscopy

Faecal microscopy was done to investigate for metazoa, protozoa and blood cells as described in literatures of Merck [18] and CDC [61].

The technique of zinc-sulphate centrifugation – flotation was used to investigate for the presence of cysts of protozoa and helminth ova in faeces. Investigation for blood cells (leukocytes and red blood cells) in faeces was carried out using Gram staining technique.

2.3.3.7 Faecal occult blood test

The Haemoccult slid test (smith – kline diagnostics) was used to investigate for the presence of occult blood in faeces. It is a guaiac based test as described by Baron [60]. One side of a guaiac – impregnated paper was smeared with faeces and a few drops of developer solution (stabilized peroxide reagent) were added to the opposite side of the paper. Appearance of a blue color within 30 seconds was considered a positive test.

2.4 Method of Data Analysis

Both qualitative and quantitative forms of analysis were used for the study to analyze data obtained. All parameters were subjected to a two tail analysis of variance (t-test) to determine the level of their significance as previously described by Norman and Bailey [68] using Microsoft excel of 2010 version.

3. RESULTS

Results of laboratory investigation revealed as follows:

3.1 Haematology

Macrocytic hypochromic anaemia; thrombocytopenia; and relative neutrophilia and lymphopenia

3.2 Serum Chemistry

Elevated AST and GGT (18 out of 18 subjects); elevated CK (4 out of 18 subjects); hypoproteinemia; hypoalbuminemia; hyperglobulinemia; reduced BUN and creatine concentrations; and lower mark glucose concentration.

3.3 Urinalysis

Numerous hyaline cast (12 subjects); and Phosphate crystals (3 out of 18 subjects).

3.4 Faecal Microscopy

Dicrocoelium dendriticum (4 subjects); Dicrocoelium dendriticum + Strongyle worm/Toxocara sp (2 subjects); Dicrocoelium dendriticum + Eimeria sp (3 subjects); Dicrocoelium dendriticum + Paramphistomum sp (3 subjects); Fasciola hepatica (2 subjects); Fasciola hepatica + Eimeria sp (1 subject); Fasciola hepatica + Paramphistomum sp (3 subjects).

3.5 Faecal Cytology

Significant presence of leukocytes in 12 out of 18 subjects with concurrent gastro-intestinal parasitic infections.
3.6 Faecal Occult Blood Test

Positive for all 18 subjects.

3.6.1 Trend of aetiologies of chronic emaciation/cachexia

The trend of aetiologies of chronic emaciation and cachexia in off take cattle in Ibadan metropolis are summarized in Table 1.

3.7 Statistical Data Analysis

Statistical data analysis was performed with STATA 12, using unpaired two-sample t test with assumption of unequal variances between the clinical cases and the control subjects. Means and standard deviations with confidence intervals for each study variable were calculated for each group. The means obtained were then compared between the two groups and against the standard reference values for each haematologic or serum biochemical values. Level of significance threshold was set at P = 0.05.

The statistical results are summarized in Tables 2 and 3.

4. DISCUSSION

In the study, trematodiasis with secondary or concurrent systemic organ involvement was diagnosed in 18 out of 100 subjects, representing 18% of the total number of chronic emaciated and cachexic cattle investigated (Please refer to Table 1 above). These include 12 emaciated subjects with trematodiasis and secondary hepatoobiliary disorder (congestive hepatopathy, chronic hepatitis, cholestasis, cirrhosis), with or without concurrent right Congestive Heart Failure (Primary Disorder); 4 emaciated subjects with trematodiasis and secondary hepatoobiliary disorder with pathogenic *Eimeria* sp infection; 2 emaciated subjects with trematodiasis and secondary hepatoobiliary disorder, with nematode infection.

*Fasciola hepatica* was detected in 6 out of the 18 emaciated subjects while *Dicrocoelium dendriticum* was detected in the remaining 12 subjects diagnosed with trematodiasis and secondary or concurrent systemic organ compromise. Mixed infection of *Fasciola hepatica* and *Paramphistomum* sp was detected in 3 subjects while mixed infection of *Dicrocoelium* sp and *Paramphistomum* sp was detected in another 3 subjects.

The general physical examination findings in this group with trematodiasis include unthrift appearance, arched back posture, emaciation, mucopurulent ocular discharges, ruffled hair coat, weakness, dullness, pale mucous membranes, tachycardia, dyspnea, tachypnea, diarrhea and dehydration, patchy erythema of the skin, ascites and distended abdomen.

However, 4 out of the 18 subjects in this group presented obvious signs of cardiopulmonary compromise such as distended jugular vein, increased jugular pulse, abduced forelimbs, extended neck and flared nostril, cyanosis, cardiac murmur, edema and ascites, breathing difficulty and tachycardia. These findings may have been associated with right Congestive Heart Failure as a concurrent condition in these subjects. The findings of abducted forelimbs, extended forelimbs, flared nostril, cyanosis, abnormal breathing and tachycardia were suggestive of reduced oxygen diffusion-perfusion in the lungs; while the finding of a right systolic cardiac murmur may be associated with a cardiac abnormality such as endocarditis, tricuspid valve insufficiency, right ventricular myocarditis/myopathy, myocardial infarction or pulmonic stenosis, pulmonary hypertension/cor pulmonale etc. [7].

The finding of arched back posture generally in the group may be due to abdominal pain associated with pressure on the liver capsule from parenchymal swelling or directly from the lesions [7]. The findings of patchy erythema of the skin and subcutaneous edema were possibly due to hypersensitization associated with phylloerythrin retention, consequent upon compromised hepatic function [43,7]. Ascites as determined by the presence of abdominal fluid waves may have resulted from portal hypertension caused by venous blockage and protein leakage into the peritoneal cavity; and low albumin concentrations [43,7,1]. Low albumin concentrations and reduced oncotic pressure may be reasons for the observed edema in the emaciated subjects [43]. Congestive Heart Failure may have contributed to hepatic congestion and ascites for the 4 subjects diagnosed with concurrent right Congestive Heart Failure [69]. In mixed infection of trematode and pathogenic *Eimeria* sp, the faeces appeared loose and dark in colour, while in mixed infection of trematode and nematode (*Strongyle worm/Toxocara* sp), the faeces appeared loose and normal in colour.
Table 1. Frequency distribution of aetiologies of chronic emaciation and cachexia of off take cattle in Ibadan

<table>
<thead>
<tr>
<th>S/N</th>
<th>Disease/Disorder diagnosed</th>
<th>Frequency (no of cattle)</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td><strong>Endoparasitic Infections Without Systemic Organ Compromise (70%)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Subjects with single nematode infection such as Capillaria sp, Strongyle, Toxocara vitulorum and Trichuris sp</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>Subjects with mixed infection of single nematode and Eimeria sp</td>
<td>18</td>
<td>18</td>
</tr>
<tr>
<td></td>
<td>Subjects with mixed infection of nematode sp with or without Eimeria sp including Capillaria sp, Strongyle, Toxocara vitulorum and Trichuris sp</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Subjects with single infection of Eimeria sp</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>Subjects with mixed infection of nematode and Paramphistomum sp</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Subjects with mixed infections involving nematode (Capillaria sp), cestode (Moniezia sp) and pathogenic Eimeria sp</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Subjects with single infection of Paramphistomum sp</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Subjects with single infection of Trypanosoma congolense</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Subjects with infection of Trypanosoma congolense and concurrent infection with nematodes</td>
<td>4</td>
<td>4</td>
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<tr>
<td></td>
<td><strong>Subtotal</strong></td>
<td>70</td>
<td>70</td>
</tr>
<tr>
<td>2.</td>
<td><strong>Endoparasitic Infection with Secondary/Concurrent Organ Disorders (22%)</strong></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Trematodiasis with Secondary Hepatobiliary Disorder (Congestive Hepatopathy, Chronic Hepatitis, Cholestasis, Cirrhosis); with or without Concurrent Right Congestive Heart Failure (Primary Disorder)</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>Trematodiasis/Secondary Hepatobiliary Disorder with Nematodiasis (Strongyle worm/Toxocara sp Infection)</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Trematodiasis/Secondary Hepatobiliary Disorder with Coccidiosis</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Trypanosomiasis (Trypanosoma brucei infection) with Secondary Cardiac Disorder (Right Congestive Heart Failure Possibly Associated with Ventricular Myocarditis/Myopathy or Myocardial Infarction/Concurrent Infection with Nematode)</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Helminthiasis, Concurrently with Urinary Tract Infection</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td><strong>Subtotal</strong></td>
<td>22</td>
<td>22</td>
</tr>
<tr>
<td>3.</td>
<td><strong>Others (8%)</strong></td>
<td></td>
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<tr>
<td></td>
<td>Distinct Primary Organ Disorders:</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Cardiac Disorders Associated with Right Congestive Heart Failure Possibly Due to Endocarditis, Tricuspid Valve Insufficiency, Ventricular Myocarditis/Myopathy, Myocardial Infarction or Pulmonic Stenosis, etc.)</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Granulomatous Disease (Tuberculosis, Corynebacteriosis, Nocardiosis, Mycotic Infection etc.)/ Intestinal Infiltrative Disease</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Inflammatory Muscle Disease (Sarcocystosis?)</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Nutritional Deficiencies (Protein-Calorie Malnutrition/Vitamin B12 Deficiency)</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td><strong>Subtotal</strong></td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td><strong>Total</strong></td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>
### Table 2. Haematology

<table>
<thead>
<tr>
<th>S/N</th>
<th>Parameter</th>
<th>Cases (n = 100)</th>
<th>Control (n = 50)</th>
<th>P-value</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.</td>
<td>PCV (%)</td>
<td>20.39 ± 2.66</td>
<td>37.94 ± 2.53</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Hb (g/dL)</td>
<td>5.80 ± 0.83</td>
<td>12.37 ± 0.88</td>
<td>&lt;0.001*</td>
<td>8 – 15</td>
</tr>
<tr>
<td>3.</td>
<td>RBC (x 10^6/μL)</td>
<td>3.23 ± 0.44</td>
<td>8.28 ± 0.52</td>
<td>&lt;0.001*</td>
<td>5 – 10</td>
</tr>
<tr>
<td>4.</td>
<td>MCV (FL)</td>
<td>63.23 ± 1.57</td>
<td>45.85 ± 1.18</td>
<td>&lt;0.001*</td>
<td>40 – 60</td>
</tr>
<tr>
<td>5.</td>
<td>MCHC (g/dL)</td>
<td>28.42 ± 0.98</td>
<td>32.61 ± 0.67</td>
<td>&lt;0.001*</td>
<td>30 – 36</td>
</tr>
<tr>
<td>6.</td>
<td>Platelet (x10^3/μL)</td>
<td>78.56 ± 12.22</td>
<td>277.22 ± 49.67</td>
<td>&lt;0.001*</td>
<td>100 – 800</td>
</tr>
<tr>
<td>7.</td>
<td>WBC (x10^3/μL)</td>
<td>5.26 ± 1.19</td>
<td>6.46 ± 0.49</td>
<td>0.002*</td>
<td>4 – 12</td>
</tr>
<tr>
<td>8.</td>
<td>Neutrophil (%)</td>
<td>44.89 ± 6.09</td>
<td>28.11 ± 2.45</td>
<td>&lt;0.001*</td>
<td>15 – 33</td>
</tr>
<tr>
<td>9.</td>
<td>Band Neutrophil (%)</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td>Eosinophil (%)</td>
<td>1.89 ± 1.08</td>
<td>1.89 ± 1.13</td>
<td>1.000</td>
<td>0 – 0.1</td>
</tr>
<tr>
<td>11.</td>
<td>Basophil (%)</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>--</td>
<td>0 – 2</td>
</tr>
<tr>
<td>12.</td>
<td>Monocyte (%)</td>
<td>1.5 ± 0.62</td>
<td>1.9 ± 0.76</td>
<td>0.130</td>
<td>0 – 8</td>
</tr>
<tr>
<td>13.</td>
<td>Lymphocyte (%)</td>
<td>51.72 ± 5.92</td>
<td>68.11 ± 2.27</td>
<td>&lt;0.001*</td>
<td>45-75</td>
</tr>
</tbody>
</table>

*Interpretation: P-values with asterisks indicates the mean differences in the haematological parameters between the clinical cases and the control group is statistically significant (P < 0.05)*

### Table 3. Serum chemistry

<table>
<thead>
<tr>
<th>S/N</th>
<th>Parameter</th>
<th>Cases (n = 100)</th>
<th>Control (n = 50)</th>
<th>P-value</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.</td>
<td>Total Proteins (g/dL)</td>
<td>6.21 ± 0.55</td>
<td>7.03 ± 0.22</td>
<td>&lt;0.001*</td>
<td>6.7 – 7.5</td>
</tr>
<tr>
<td>2.</td>
<td>Albumin (g/dL)</td>
<td>2.04 ± 0.26</td>
<td>3.33 ± 0.17</td>
<td>&lt;0.001*</td>
<td>2.5 – 3.8</td>
</tr>
<tr>
<td>3.</td>
<td>Globulin (g/dL)</td>
<td>4.17 ± 0.41</td>
<td>3.70 ± 0.17</td>
<td>&lt;0.001*</td>
<td>3.0 – 3.5</td>
</tr>
<tr>
<td>4.</td>
<td>AST (μL)</td>
<td>191.67 ± 17.31</td>
<td>87.00 ± 13.03</td>
<td>&lt;0.001*</td>
<td>60 – 125</td>
</tr>
<tr>
<td>5.</td>
<td>CK (μL)</td>
<td>179.11 ± 144.06</td>
<td>118.83 ± 40.70</td>
<td>0.082</td>
<td>0 – 350</td>
</tr>
<tr>
<td>6.</td>
<td>GGT (μL)</td>
<td>33.06 ± 5.95</td>
<td>9.72 ± 1.41</td>
<td>&lt;0.001*</td>
<td>6 – 17.4</td>
</tr>
<tr>
<td>7.</td>
<td>Bilirubin (mg/dL)</td>
<td>1.46 ± 0.22</td>
<td>0.82 ± 0.19</td>
<td>&lt;0.001*</td>
<td>0.5 – 2.2</td>
</tr>
<tr>
<td>8.</td>
<td>Creatinine (mg/dL)</td>
<td>0.75 ± 0.12</td>
<td>0.96 ± 1.09</td>
<td>&lt;0.001*</td>
<td>0 – 1.6</td>
</tr>
<tr>
<td>9.</td>
<td>BUN (mg/dL)</td>
<td>9.70 ± 1.35</td>
<td>17.39 ± 0.60</td>
<td>&lt;0.001*</td>
<td>10 – 25</td>
</tr>
<tr>
<td>10.</td>
<td>Glucose (mg/dL)</td>
<td>50.28 ± 4.43</td>
<td>73.72 ± 5.69</td>
<td>&lt;0.001*</td>
<td>40-100</td>
</tr>
</tbody>
</table>

*Interpretation: P-values with asterisks indicate the mean differences in the serum chemistries between the clinical cases and the control group is statistically significant (P < 0.05)*
Haematologic findings include macrocytic hypochromic anaemia with lowered red cell indices, which may have been due to liver haemorrhage as a result of immature, wandering flukes that destroyed liver tissue, and also as a consequence of trauma and feeding activity of adult flukes within the bile ducts [43,54]; thrombocytopenia which may have resulted from sequestration of thrombocytes associated with splenic enlargement, and possibly from increased platelet consumption due to liver haemorrhage [54,18]; and relative lymphopenia and neutrophilia which may have been associated with endogenous stress due to inflammation and haemorrhage [70]. In subjects with concurrent infections with *Eimeria* sp or *Paramphistomum* sp or nematodes, anaemia was severe.

Serum chemistry findings include elevated aspartate amino transferase (AST) (a nonspecific enzyme) and gamma glutamyl transferase (GGT) for 14 subjects without concurrent Congestive Heart Failure; and serum creatine kinase levels were within the normal range for the specie. An elevated AST with a normal CK is suggestive of exclusion of muscle related disorder (cardiac and skeletal), and presence of liver pathology such as may have been caused by flukes in these subjects [43,54,7,1]. However, AST and CK were both elevated for the other 4 subjects with concurrent Congestive Heart Failure, suggestive of muscle disorder (possibly cardiac abnormality) [54,7,1] as part of the systemic disease condition. The damaging effects of Adult flukes residing in the bile ducts include cholecystitis, thickened duct wall and fibrosis etc.; and this may have resulted in enzyme (GGT) leakage from the biliary epithelium with increased serum concentration [43,54]. This finding is significant, and provides the basis for diagnosis of hepatobiliary disorder in the evaluation [43].

Hypoproteinemia was generally observed in the subjects. Hypoalbuminemia and hyperglobulinemia were important clinical findings. Hypoproteinemia and hypoalbuminemia are expected findings with compromised liver which has reduced capacity to anabolize proteins [50]. Consequently, serum BUN and creatinine were observed to below normal [54]. Hyperglobulinemia may have been due to inflammation and acute phase proteins [54]. In subjects with concurrent infections with *Eimeria* sp or *Paramphistomum* sp or nematodes, hypoalbuminemia was severe.

Serum glucose was low normal for all the subjects possibly due to effects on glucose homeostasis and glycogen storage by the compromised liver [54]. Serum bilirubin concentrations for the subjects in this group were within normal range, though some were at the upper mark. This finding was not unexpected as in liver disease of cattle, bilirubin level is usually within normal range, and elevation above reference value is commonly associated with hemolytic conditions; and in severe diffused hepatic disease or hepatic failure [43].

The finding of hyaline casts on urine sediment examination for some of the subjects may have been due to dehydration [67], while the finding of significant phosphate crystals in urine of 3 subjects was suggestive of high alkaline urine [67].

Faecal cytology revealed inflammatory cells (leukocytes) for subjects with concurrent infections with *Eimeria* sp and nematodes; and also for mixed infection with the rumen fluke, *Paramphistomum* sp. Faecal occult blood test was positive for all the subjects in this group; this may have resulted from haemorrhage and communication with the intestinal tract; and in addition, for subjects with concurrent infection with *Eimeria* sp, from small intestinal haemorrhage [18].

Diagnosis of trematodiasis and associated infection with *Eimeria* sp or nematodes for some subjects were based on clinical findings including the detection of ova of *Fasciola hepatica*, *Dicrocoelium* sp or *Paramphistomum* sp (in mixed infection) and nematodes; and oocysts of *Eimeria* sp with zinc sulphate centrifugation-flotation technique. Results of qualitative faecal test revealed higher prevalence of fluke infection in cattle aged four years and above (13 out of 18 cases constituting about 72%); this finding is consistent with the report of higher prevalence in aged cattle by Affroze et al. [42]. However, the rate of fluke infection appeared not to have been affected by breed or sex as prevalence or severity was about the same in cases diagnosed for animals of different breeds and sexes. Seasonal influence may have resulted in the high incidence rate as animals were sampled between September and mid-December which coincides with fall and early winter periods [40]. Apart from a few with *Eimeria* sp oocysts and/or ova of nematodes there was absence of fluke ova in faeces of control animals following qualitative faecal test.
Diagnoses of secondary and concurrent systemic organ compromise were based on analyses of physical examination and laboratory findings such as haematology, serum chemistry, urinalysis, faecal cytology and occult blood analysis. The findings of macrocytic hypochromic anaemia associated with haemorrhage and elevated serum GGT associated with biliary disorder were suggestive of the presence of both liver wandering immature and duct residing mature flukes [43] for subjects in this group, which were further suggestive that these cases may have been at the sub-acute stage. Further evaluation of the liver by ultrasonography for imaging, biopsy and guidance for aspiration of bile duct to detect flukes supports diagnosis and also reveals the degree of hepatobiliary compromise [43,71,7,172]. Electrocardiography, echocardiography and measurement of serum cardiac troponin concentrations aid evaluation of cardiac disorders [73,7].

The haematologic and serum chemistry profile of the 18 emaciated and cachexic cattle with trematodiasis were clinically and statistically compared with those of 18 apparently healthy cattle or otherwise referred to as control subjects (refer to Tables 2 and 3). The haematologic parameters of PCV, Hb concentration, RBC count, MCV, MCHC, neutrophil and platelet count were statistically significant (p<0.05). These were also clinically significant due to alterations in their values either below or above reference ranges; and also at the border lines of reference ranges. The serum chemistry parameters of total proteins, albumin, globulin, glucose, creatinine, bilirubin, BUN, AST and GGT were statistically significant (p<0.05). These and parameter of serum concentration of CK were clinically significant due to alterations in their values either below or above reference ranges; and also at the border lines of reference ranges.

5. CONCLUSION

In conclusion, the study established trematodiasis with secondary or concurrent organ compromise as one of the important aetiologies of chronic emaciation and cachexia in off take cattle in Ibadan metropolis. Further investigation is however required to evaluate some of the clinico-pathological findings and relate these with existing or possible new epidemiological factors as associated with dicrocoeliasis and fascioliasis. Nonetheless, the findings of the study will be clinically and epidemiologically relevant, particularly in northern States of Nigeria where these animals are sourced with a view to reviewing their livestock health programmes. An integral part of the livestock health programme should be a broad based and periodic helminth control programme to encompass liver fluke infection.

ETHICS

Handling of all subjects for various procedures in the study was in conformity with stated guidelines of safe handling of animals as provided in the animal’s act and Council of International Organizations for Medical Sciences (CIOMS).

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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68. Source: PubMed


