JOURNAL OF VETERINARY AND APPLIED SCIENCES
2014 VOL. 4(1): 43 - 47
Manuscript No. JVAS/2014/032; Received: 17/09/2013; Accepted: 18/07/2014
Published by: Faculty of Veterinary Medicine, University of Nigeria, Nsukka, Nigeria

A CASE OF NUTRITIONAL DEGENERATIVE CARDIOMYOPATHY IN THE DOG

*** Gloria Daniel-Igwe**

[1]Department of Veterinary Pathology, College of Veterinary Medicine,
Michael Okpara University of Agriculture, Umudike, Abia State, Nigeria

==========

ABSTRACT

Seven pure bred Alsatian dogs acquired at 2 months of age were maintained on a diet of predominantly rice, beans, milk and occasionally ground beef without the addition of red palm oil. The dogs remained apparently healthy until they were 6 months old when one of them collapsed and died suddenly during exercise. A few days thereafter, the remaining six dogs showed signs of anorexia, emaciation, muscular weakness and paresis and later hyperpnoea with nasal discharge. The urine was dark brown and all the dogs died within two weeks of manifesting these clinical signs. Postmortem examination of the dead dogs revealed cardiac enlargement, myocardial degeneration and necrosis with calcification especially in the papillary muscles, consolidation of the right lung, spleen and liver enlargement with ulceration of the duodenal mucosa. Based on the history, the manifested clinical signs and the gross and histopathologic lesions seen at post mortem, it was concluded that the dogs suffered from nutritional deficiency of vitamin E/Selenium which resulted in degenerative cardiomyopathy and consequent congestive heart failure and death.

***Keywords*:** Nutrition, Deficiency, Degenerative, Cardiomyopathy, Dog.

==========

INTRODUCTION

Nutritional myopathies are common in both domestic and wild animals with malnutrition being common in their cause and degeneration of cardiac and skeletal muscles in their manifestations [1]. Azoturia has been reported in horses after a few days of rest on full feed and followed by resumption of exercise. The large masses of skeletal muscles are affected while the kidneys suffer injury with degeneration and necrosis of the proximal convoluted tubular epithelium. The lesions are caused by accumulating lactic acid which irritates or stimulates the muscle to extreme contraction with lack of oxygen and blood leading to necrosis [2].

Capture myopathy follows severe physical exertion associated with capture involving the musculature. In such cases, the clinical signs of convulsions, opisthotonus and torticollis are usually observed before paralysis. The lesions consist of sharply demarcated zones of haemorrhage with pale, soft gelatinous muscle, usually bilateral, with dark brown swollen kidneys. Haemorrhages occur in the adrenal cortex.

Correspondence: E-mail: gloria.danieligwe@gmail.com; **Tel:** +234 8064817749
ISSN: 2315 - 6856

The syndrome is seen mostly in capture and transport of wild animals with severe physical exertion [3, 4]. In addition, certain plants including coyotillo, coffee senna, gossypol and vetch are known to cause myodegeneration and necrosis [2, 4].

As a result of the bad economic conditions in Nigeria, many owners do not feed their dogs with ready-made balanced dog diets. In its place they feed these dogs with human food items which may not provide all the essential nutrients the animals require for healthy growth.

This paper describes a case of suspected nutritional degenerative cardiomyopathy in seven pure bred Alsatian dogs that died at the age of six months following their maintenance and feeding on diet of rice, beans, milk and ground beef without the addition of red palm oil (*Eleasis guineansis*).

CASE HISTORY

Seven pure breed Alsatian dogs were acquired at the age of two months by a private dog breeder in Umuahia, Abia State, Nigeria. They were housed in a fly-proof kernel with concrete floor that was regularly washed with disinfectants. The dogs received all necessary vaccinations against the common canine diseases in the environment: leptospirosis, infectious canine hepatitis (ICH), canine parvovirus (CPV), canine distemper (CD) and anti–rabies vaccine (ARV). The feeding consisted mainly of boiled rice and beans, fed either separately or in combination. Milk and ground meat were occasionally served with the main diet. Red palm oil was never added to the mix of boiled food, neither was a separate sauce prepared with red palm oil for eating the boiled beans and rice as practiced by humans. Water was given *ad libitum*.

The dogs were taken out daily for exercise that usually lasted a few hours. This practice started from the age of two months when the dogs were acquired. The dogs remained in apparently healthy condition until the age of six months when one of the dogs collapsed and died after such exercise. The regular exercise stopped after the death of the first dog. Four days later, the remaining six dogs became clinically sick and died within two weeks of the manifestation of clinical signs.

MATERIALS AND METHODS

At death all animals were subjected to a thorough postmortem examination. Tissue samples were preserved in 10% neutral buffered formalsaline solution for histopathology. The tissues were dehydrated in graded concentrations of ethanol, cleared in xylene, embedded in paraffin wax, sectioned at 5 microns thickness and stained with haematoxylin and oesin (H&E) stain and with Von Kossa (12). Smears/swabs of tissue fluids from the liver, kidney, spleen and lymph nodes were subjected to microbial isolation tests following standard microbiological procedures (14).

RESULTS

Clinical Manifestations

The clinical signs observed in the dogs included muscular weakness, with reluctance to walk or jump. Their respiration was labored and became more marked with the exercise. The gait was unsteady with droopiness of the head when forced to walk. The abdominal wall was flaccid. There was a moist cough. The dogs either refused to eat or vomited soon after eating. Some of the dogs developed oedema of the dependent parts of the body: limbs, neck and ventral abdomen. As the condition progressed the dogs showed extreme emaciation and depression with purulent nasal discharge. Pustular dermatitis developed in the inter-digital clefts of all limbs and the hairs pulled off easily. The urine was dark brown in colour. The dogs usually died within two weeks of the manifestation of such clinical signs.

Gross Pathological Lesions

The animals were grossly emaciated with subcutaneous edema of the ventral abdomen. There was no transudation into the abdominal cavity. The skeletal muscles were pale and edematous. The heart was

enlarged especially in the ventricles with pale areas in the myocardium intermixed with normal-looking areas especially in the papillary muscles (Fig. 1). The lungs were edematous with scattered lobular consolidation in the right antero-ventral parts (Fig. 1). The trachea contained frothy fluid. The bronchial lymph nodes were swollen, red and edematous on cut surface, while the liver was congested. The duodenal mucosa was eroded.

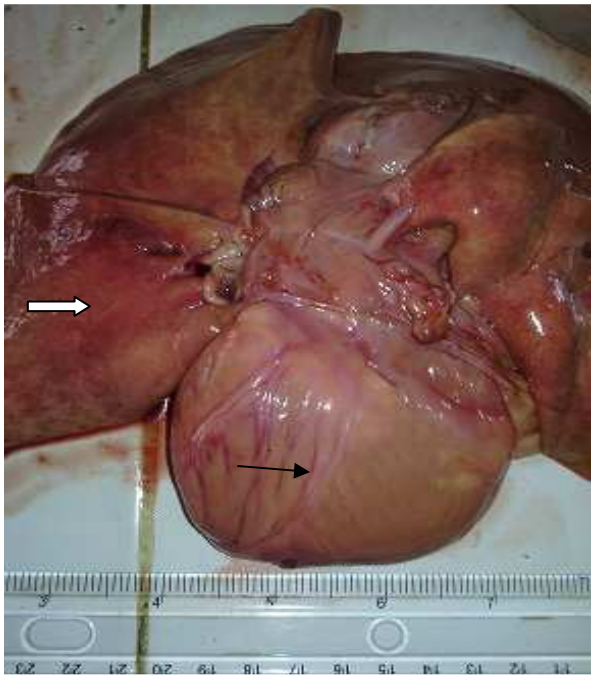

Fig. 1. Gross photograph showing the enlarged heart (black arrow) and edematous lungs with areas of consolidation (white arrow).

Histopathology

The skeletal muscle fibres showed areas of coagulative necrosis, with deep eosinophilia and myorrhexis (fragmentation). The cardiac lesions were localized in the sub-endocardial areas of the ventricles and consisted of fiber necrosis, and calcification confirmed by special staining for calcium (Fig 1). The adjacent interstitium was infiltrated by a few mononuclear leucocytes. The lymph nodes were congested and edematous with reticuloendothelial proliferation and erythrophagocytosis in the medullary sinuses. The lungs had generalized alveolar edema and congestion. The liver and spleen were congested. Laboratory tests did not reveal any microbial or parasitic agents.

DISCUSSION

In Nigeria, a combination of beans, rice, milk and meat are commonly consumed by humans. In the preparation of such diet, red palm oil (*Eleasis guineansis*) is freely added and thoroughly mixed with it during or after boiling but before it is eaten. In such diets, rice formed the source of energy while the beans, milk and meat provided the protein source. The red palm oil was added to provide fat soluble vitamins and minerals. However, in the present case, red palm oil was not used in cooking the food of these dogs. White rice, beans, milk and beef contain very low levels (1µg/g; 5µg/g; 2µg/g and 6µg/g respectively) of vitamin E (Bauernfied, 1980). On the other hand, red palm oil is a very rich source of vitamin E; containing 985µg/g [5]. The minimum requirement of the dogs is about 70µg/g of vitamin E per day [6]. Therefore, a diet of rice, beans, milk and meat without supplementation of red palm oil is not likely to provide adequate amount of vitamin E to dogs. The nutritional deficiency of vitamin E may have therefore been responsible for the condition of the dogs in the present case.

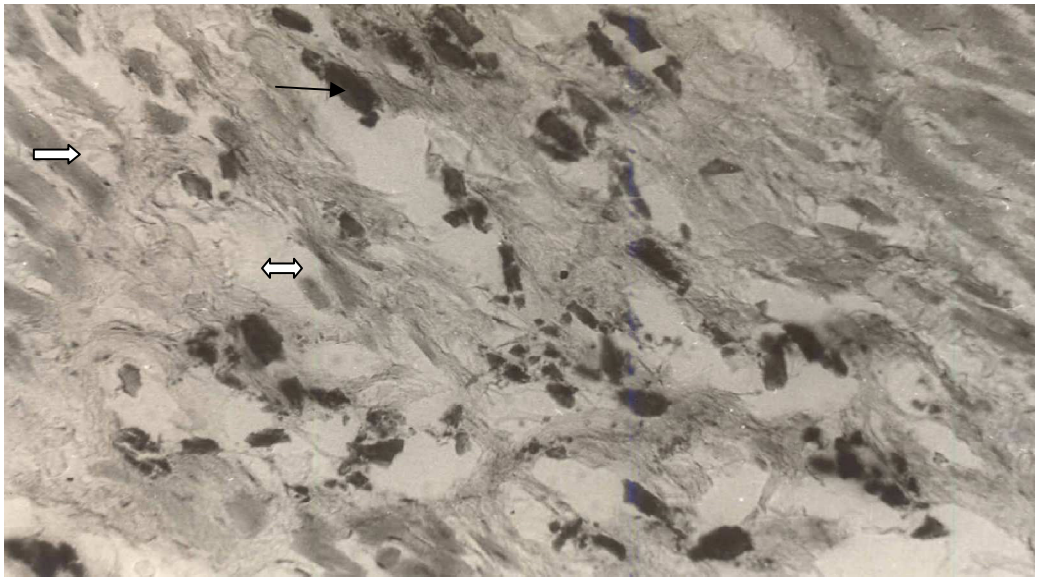

Fig 1. Photomicrograph showing hyaline degeneration (white arrow), necrosis (double arrow head) and calcification of the myocardium (black arrow), Von Kossa ×40.

Vitamin E prevents lipid peroxide formation and the autocatalytic lipid peroxidation and cell damage [7]. The clinical signs and post mortem lesions observed in the present case are consistent with those seen in dogs with vitamin E deficiency [2, 8]. Skeletal myopathy has been reported in young dogs fed evaporated milk that was not supplemented with vitamin E [9]. Necrosis of skeletal and cardiac muscles is common in young animals with vitamin E / Selenium deficiency [10]. Physical exertion is known to increase the myopathy induced by vitamin E/Selenium deficiency by increasing the *in vivo* lipid peroxidation [11]. This may account for the sudden death in the first dog that collapsed and died after an exercise. The vomiting observed may be due to the duodenal ulceration seen in most of the dogs. The dark brown urine could have resulted from myoglobinuria associated with the necrotic muscles. There was no damage to the kidneys. The moist cough and the terminal development of edema in the limbs, neck and ventral abdomen may be indicative of the enlarged and malfunctioning heart (4).

It is therefore suspected that the diet consisting predominantly of boiled rice, beans, milk and meat fed to the dogs without the supplementation of red palm oil, or any other source of vitamin E might have been responsible for the myocardial lesions with consequent congestive heart failure and death observed in the Alsatian dogs. It is therefore recommended that dog owners should ensure that dog diets are prepared with red palm oil or other extraneous sources of supplementary vitamin E. In addition, some animal sources of selenium example, liver should be added to dog diets prepared at home when in deficient area. But care should be taken not to pump too much of this trace mineral into the food because it can create toxicity at a high level. Never give more than 200 mg of selenium per day in larger breeds and no more than 100 mg per day for smaller dogs (13).

ACKNOWLEDGEMENT
The author is grateful to Dr. E. O. Onyekweodiri of the Department of Veterinary Pathology, College of Veterinary Medicine, Michael Okpara University of Agriculture, Umudike, Abia State, Nigeria for helpful suggestions.

REFERENCES

1. Jubb, K. V. F. and Kennedy, P. C. (1970). *Pathology of Domestic Animals*, 2nd Ed. Vol. 2 Academic Press New York Pp. 482.
2. Jones, T. C., Hunt, R. D. and King, N. W. (1996). *Veterinary Pathology*. 6th Ed. Williams and Wilkins, Philadelphia Pp. 986.
3. Hauer, G. (2001). Capture myopathy. http.//bisoncentre.com/resources/bce240/bce240/_capture_ myopathy.html. Reprinted from the *Tracker*, 5 (2).
4. McGavin, M. D. and Zachary, J. F. (2007). *Pathologic Basis of Veterinary Disease*, 4th Ed. Mosby Elsevier, pp 584 – 590.
5. Bauernfeind, J. C. (1980). *Vitamin E: A comprehensive treatise*, L. J. Machin (Ed) Marcel Dekker, New York Pp. 99.
6. Scott, M. L. (1978). *Handbook of Lipid Research*. H. F. Deluca (Ed.), Vol. 2, Plenon Press, New York. Pp. 133.
7. Combs, G. F. (jr), Noguchi, T. and Scott, M. L. (1975). Mechanisms of action of selenium and vitamin E in protection of biological membranes, Federation Proceedings. 34(11): 2090 - 2095.
8. Van Vleet, J. F. (1975). Experimentally induced vitamin E / selenium deficiency in the growing dog. *Journal of the American Veterinary Medical Association*, 166: 769 – 774.
9. Anderson, H. D., Elvehjem, C. A. and Gonce, J. E. jr. (1940). A comparison of the nutritive values of raw, pasteurized and evaporated milk for the dog. *Journal of Nutrition*, 20: 433 – 443.
10. Andrews, E. D., Hartley, W. J. and Grant, A. B. (1968). Selenium-responsive diseases of animal in New Zealand. *New Zealand Veterinary Journal*, 16(1-2): 3 – 17.
11. Brady, P. S., Brady, L. J. and Ullrey, De. E. (1979). Selenium, vitamin E and the response to swimming stress in the rat. *Journal of Nutrition*, 109: 1103 – 1109.
12. Bancroft, J. D. and Stevens, A. (1977). *Theory and Practice of Histological Techniques*. Churchill Livingstone, London, Pp 407.
13. McGuffin, D. (1999). What Are the Benefits of Selenium in Dogs? http://www.ehow.com/facts_5630732_benefits-selenium-dogs_.html Accessed 15th June, 2014.
14. Kelley, S. G. and Post, F. J. (1989). *Basic Microbiology Techniques*. 3rd ed.Star Publishing Company Belmont, California. ISBN: 9780898631166, 272Pp