Onderstepoort Journal of Veterinary Research, 61:107–109 (1994)

RESEARCH COMMUNICATION

Putative avocado toxicity in two dogs

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ABSTRACT

BUORO, I.B.J., NYAMWANGE, S.B., CHAI, D. & MUNYUA, S.M. 1994. Putative avocado toxicity in two dogs. *Onderstepoort Journal of Veterinary Research*, 61:107–109

Two dogs were seen at the University Veterinary Teaching Hospital, Nairobi, Kenya, both having histories of dyspnoea, progressively enlarging abdomens, anasarca, ascites, pleural and pericardial effusion, and pulmonary oedema. One of the dogs had a mild neutrophilic leucocytosis, elevated levels of alkaline phosphatase, alanine aminotransferase, lactate dehydrogenase and proteinuria. Histopathological examination of the myocardium revealed some damage to myocytes and a mononuclear cellular infiltration involving the myocardium, liver and kidneys. The two dogs had a fondness for avocado fruits and, as the presenting syndrome is identical to that seen in goats, sheep and horses poisoned by avocados, a comparison is made and the probable manifestation of this poisoning presented.

INTRODUCTION

Canine myocardial disorders constitute only a small percentage of cardiovascular disease in the specie (Fox 1989). Myocarditis (inflammation of myocytes, interstitium or vascular structures) may be acute or chronic, with involved animals presenting as asymptomatic or with signs of congestive heart failure. Aetiological diagnosis of myocarditis is always difficult, being often implied through consideration of history and extracardiac manifestations of the disease process (Fox 1989). Documented extraneous causes of secondary cardiomyopathy/heart disease in the dog are numerous, but can be divided into infectious, parasitic and physical agents, and metabolic diseases and toxins. Included in the first two categories are canine parvovirus, adenovirus of canine distemper, trypanosoma species, hepatozoon canis, several bacterial species and agents of deep mycoses, while in the last category one may find chemotherapeutic agents such as doxorubicin (adriamycin), heavy metals such as lead, and several toxic ornamental plants including *Nerium oleander*, lily-of-the-valley and foxglove (Robinson & Maxie 1985; Fox 1989).

Although certain varieties of avocados are reportedly toxic to the dog (Ohme 1983), no clinical case has been described to date. The fruit, seeds and leaves of certain varieties of avocado trees can be toxic to cattle, goats, horses, rabbits, canaries and fish. Sheep and goats that are poisoned by the Fuerte strain present with a cardiac-failure syndrome (Grant, Basson, Booker, Hofherr & Anthonissen 1991; Sani, Atwell & Seawright 1991; Stadler, Van Rensburg & Naudé 1991). It is therefore interesting that the two

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Received 18 November—Editor

farm dogs that had developed a taste for avocados (Fuerte variety) presented with identical syndromes.

HISTORY

A 3-year old Alsatian bitch (dog 1) and a 4-year-old mongrel bitch (dog 2) were seen at separate farm houses about 30 km from the University Veterinary Teaching Hospital (VTH). At the time of the visit, dog 2 had been dead for about 12 h. Both had histories of poor appetite, exercise intolerance, constipation, a gradually enlarging abdomen and dyspnoea. Both the owners of the dogs had moved to their farms during the preceding year.

MATERIALS AND METHODS

Dog 1 was clinically examined and had ear-capillary blood taken for haemoparasite evaluation. Cephalic venous blood was taken for routine haematology and biochemistry (serum-protein profile, alanine amino transferase, alkaline phosphatase, lactate dehydrogenase and serum-urea nitrogen). An additional 5 mℓ of blood was obtained for *Ehrlichia canis* culture. Urine and abdominal fluids were collected for urine analysis. Because of the dyspnoea, only a lead-II electrocardiogram was taken, with the bitch in a standing position. The bitch was then medicated with furosemide (Lasix, Hoechst Ag-Vet) (3 mg/kg i/v B.I.D.) and digoxin (0,02 mg/m^2 body surface area B.I.D.). The following day the bitch was transferred to the VTH where lateral and ventrodorsal radiographs were taken. The bitch died that afternoon, a necropsy was performed and tissue was obtained from the interventricular septum, the ventricular walls, lungs, liver and kidneys for routine histopathological examination. Samples were also obtained from the cardiac muscle for bacteriology (ox-blood agar; aerobic and anaerobic). For dog 2, an on-farm necropsy was performed, but as decomposition was setting in, no tissue was taken for histopathology.

RESULTS

Clinical signs

Dog 1 was emaciated, it exhibited orthopnoea, and had jugular pulsation and ascites, pitting oedema involving both hind legs, and a regular, diminished-amplitude pulse of 200 bpm. Rectal temperature was 38,6 °C. The peripheral lymph nodes were of normal size. Thoracic auscultation revealed muffled heart sounds and crepitant rales. There was an increased area of cardiac dullness. Thoracic radiography revealed gross cardiomegaly, more so of the right heart. There was an increase in pulmonary radiopacity and evidence of pleural effusion. Electrocardiography established a presence of sinus tachycardia, with attenuated complexes.

Clinical pathology and necropsy findings

Dry, wet-blood smears and *Ehrlichia canis* culture results were negative for blood parasites. Total leucocyte count was 20 000 x 10^9/ℓ (normal range 6 000–17 000/ℓ) with moderate neutrophilia. Alanine amino transferase value was 120 IU/ℓ (normal range 21–102) lactic dehydrogenase value was 250 IU/ℓ (normal range 43–233). Alkaline phosphatase value was 180 IU/ℓ (normal range 20–156). Serum urea nitrogen was normal. The ascitic fluid was slightly turbid and with a pinkish tinge. Its protein content was 4,0 g/100 mℓ (normal less than 2,5 g/100 mℓ) with a total cell count of 500/μℓ. Within it were erythrocytes, neutrophils, macrophages, mesothelial cells and lymphocytes. On urine analysis there was proteinuria (75 mg/100 mℓ) and the presence of two hyaline casts. Serum-protein levels were normal.

At necropsy the carcases were found to be in poor body condition and there was anasarca involving both hind limbs, ascites, pulmonary oedema, and pleural and pericardial effusions. The hearts were flabby, particularly the right heart, which was also markedly pale. Dissection of the heart chambers and the great vessels to and from the heart revealed normal valves with the absence of any obstruction/constriction of the blood vessels. The liver and kidneys in dog 1 were congested. Microscopical examination showed degeneration of myocardial fibres, with foci of degeneration having mononuclear cellular infiltration (Fig. 1). The same type of cellular infiltrates were seen involving the liver and kidneys.

DISCUSSION

The clinical-examination results, electrocardiography, radiography and clinicopathological test results (dog 1) suggested the presence of congestive heart failure, with the right heart apparently more affected. This was confirmed at necropsy (both dogs), where the cardiac muscles were implicated. Histopathological assessment of the tissue obtained revealed damage to myocardial cells, a mostly subepicardial mononuclear cellular infiltration but with no discernable possible causative agent (Fig. 1). A similar cellular infiltration involved the kidneys, lungs and liver. Because of the medical history, ages of the dogs and relatively rapid onset of the presenting signs, the myocarditis was thought to be secondary. Documented extraneous causes of secondary cardiomyopathy/ heart disease are many, but can be divided, amongst others, into infectious parasitic, physical

FIG. 1 Photomicrograph of myocardium of the dog with congestive heart failure. Note the mostly subepicardial mononuclear cellular infiltrate

agents, metabolic diseases and toxins. The medical history, clinical presentation, tissue-bacteria-culture results, and clinicopathological and histopathological assessments would appear to suggest the first four as unlikely, leaving toxins as a probable cause of death.

Poisoning is an important cause of morbidity and mortality in dogs, and a variety of sources, including heavy metals, pesticides and drugs, are incriminated. However, as far as plant-related poisoning is concerned, relatively fewer cases are reported, this in spite of the ready accessibility of toxic plants around many households, and the proclivity of dogs to mouth or chew on foreign objects.

A possible reason for the contradiction is the suggested inadequate appreciation of the contribution of this hazard in small-animal practice by veterinarians (Ohme 1983). The list of potential poisonous plants is long, with Ohme (1983) listing a minimum of 59 ornamental plants. As an example, dogs and cats consuming fruit of the cycad or leaves of the dumbcane, ivies or philodendron get intoxicated.

Although some varieties of avocados are listed as being potentially dangerous to dogs and cats (Ohme 1983), no description of the clinical manifestation of such poisoning has as yet been presented. The fruit, seeds and leaves of some varieties of avocado trees are toxic to cattle, goats, rabbits, canaries and fish (Kingsbury 1964). Varieties (strains) of the avocados vary in their toxicity to animals.

This variation may also extend to clinical-signs/syndrome manifestation. For instance, *P. americana* (Fuerte strain) appears to be associated with a cardiac-failure syndrome in goats, sheep and horses (Stadler, Van Rensburg & Naudé 1991; Sani *et al*. 1919; Grant *et al*. 1991), while mastitis is the primary lesion when the guatemalan variety is involved (Craigmill & Eide 1984). It is therefore interesting that the two dogs seen by us, both having a fondness for avocados (*P. americana*, Fuerte strain), each had a clinical, clinicopathological and histopathological presentation identical to that seen in goats, sheep and horses consuming the fruit, leaves or seed of the same variety. This would seem to indicate a causal relationship.

In suspected cases of plant poisoning, a diagnosis often depends on the observations of the owner, as disparate factors such as the age of an animal, boredom and a change in surroundings may play a part in their occurrence. It is possible that the dogs seen by us developed an inordinate interest in the avocado fruit because of boredom and confinement to a restricted yard. Area restriction may produce increased interest in familiar plants (Ruhr 1986).

REFERENCES

CRAIGMILL, A.L., EIDE, R.N., SCHULTZ, T.A. & HEDRICK, K. 1984. Toxiciology of avocado leaves. *Veterinary and Human Toxicology,* 26:381–383.

FOX, P.R. 1989. Myocardial diseases, in *Textbook of Veterinary Internal Medicine*, 3rd ed., edited by S.J. Ettinger. Philadelphia: W.B. Saunders. 1097–1131.

GRANT, R., BASSON, P.A., BOOKER, H.H., HOFHERR, J.B. & ANTHONISSEN, M. 1991. Cardiomyopathy caused by avocado (*Persea americana* Mill). *Journal of the South African Veterinary Association*, 62:21–22.

KINGSBURY, J.M. 1964. *Poisonous plants of the United States and Canada.* Eglewood Cliffs, New Jersey: Prentice-Hall.

OHME, F.W. 1983. Toxicological problems, in *Textbook of Veterinary Internal Medicine*, 2nd ed., edited by S.J. Ettinger. Philadelphia: W.B. Saunders: 173–207.

RUHR, L.P. 1986. Ornamental toxic plants. in *Current Veterinary Therapy. IX. Small Animal Practice*, edited by R.W. Kirk. Philadelphia: W.B. Saunders: 216–220.

ROBINSON, W.F. & MAXIE, M.G. 1985. Cardiovascular system, in *Pathology of domestic animals*, 3rd ed., edited by K.V.F. Jubb, P.C. Kennedy, & N. Palmer. San Diego: Academic Press: 1–81.

SANI, Y., ATWELL, R.B. & SEAWRIGHT, A.A. 1991. The cardiotoxicity of avocado leaves. *Australian Veterinary Journal*, 68: 150–151.

STADLER, P., VAN RENSBURG, I.B.J. & NAUDÉ, T.W. 1991. *Journal of the South African Veterinary Association*, 62:186–188.