SUSPECTED AVOCADO (PERSEA AMERICANA) POISONING IN GOATS

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ABSTRACT

A herd of 15 Cameroon goats was suspected of having been poisoned by eating leaves of the Fuerte variety of avocado pear (*Persea americana*). Two of the affected goats were examined clinically, while necropsies were carried out on 3 of the 4 that had died. The most significant clinical findings were tachycardia, hyperpnoea and evidence of lung oedema. At necropsy severe lung oedema, hydrothorax and hydropericardium were present. Severe myocardial degeneration, necrosis and fibrosis were the major histopathological findings.

Key words: Avocado poisoning, Persea americana (Fuerte strain), goats, cardiomyopathy

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Stadler P.; Van Rensburg I.B.J.; Naudé T.W. Suspected avocado (Persea americana) poisoning in goats. Journal of the South African Veterinary Association (1991) 62 No. 4, 186-188 (En.) Department of Medicine, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onder-stepoort, Republic of South Africa.

Although the avocado pear (Persea americana) is eaten with impunity by man, several outbreaks of toxicity in animals, ingesting particularly the leaves of various varieties, have been reported. Not all commercial varieties are toxic to animals³⁷. Leaves of the Fuerte and Nabal strains killed rabbits within 24 h, whereas the Mexicola strain had no detrimental effect. Leaves, bark, seeds and fruit have been reported to be toxic to cattle, horses, goats, rabbits and canaries. In cattle, goats and horses, the primary clinical sign mentioned is a noninfective mastitis. More recently4, sheep dosed with fresh avocado leaves of the Fuerte variety, developed cardiomyopathy. The ability of the Gautemalan variety to cause severe mastitis in goats has been reported¹². The involvement of Gautemalan avocados ((P. americana (Anaheim variety)) in a field outbreak, was confirmed through dosing experiments. Although the animals developed severe non-infective mastitis both in the field and experimentally, the condition was not lethal and all the animals recovered. Neck and brisket oedema and coughing, however, was noted in the field cases. Apart from severe mammary gland pathology and oedema, and haemorrhage in the supramammary lymphnodes of one goat, no lesions were found in the organs of goats experimentally intoxicated². Unfortunately the hearts were not examined histologically. In this experiment, *P. americana* var. *Guatemala* proved to be toxic, but the Mexican variety was not.

The toxicity to cage birds of the fruit of both a common Guatemalan variety, Hass, and the very popular hybrid between the Mexican and Gautemalan varieties, Fuerte, was recently confirmed⁶. Budgerigars were highly susceptible to intoxication with both varieties. As little as 1,6 g given over a period of 4 h to budgerigars with an average mass of 36,25 g (dosage approximately 45 g kg⁻¹) was lethal within 36 h. Canaries were less susceptible and mortality was only with the Hass variety. Subcutaneous oedema, hydropericardium and generalised congestion were the most prominent findings and it was concluded that cardiac failure might have played a role in the death of the birds. Both these varieties are very popular in South Africa.

During August 1988, sudden deaths occurred in a herd of 15 Cameroon

dwarf goats on a small-holding near Onderstepoort. They were kept with a few head of cattle in a camp of approximately 0,5 ha. The goats were fed dry lucerne hay, commercial sheep pellets, yellow maize and had access to a commercial lick. They were in a fair condition. The camp in which they were kept was severely overgrazed and the animals craved green feed. Apart from bluegum trees (Eucalyptus spp.), which had been depleted of leaves as high as the animals could reach, no plants of toxicological significance could be found in this camp. Periodically the animals were fed green garden refuse. Amongst others, avocado tree cuttings from the owner's own garden had been fed, without any deleterious effects. However, 6 d before the first animal died (Day 0), the owner started feeding them the foliage of one specific avocado tree that was being cut down in a garden in the vicinity. This was fed for approximately 10 d. On Day 6 the first goat died and over the next 2 d, 2 more died. The carcasses of these latter goats, (Goats 1 and 2), were submitted for autopsies on Day 9.

The same day, 2 animals, Goat 3 (a young ewe approximately 9 months old) and Goat 4 (a heavily pregnant ewe, approximately 14 months old) were admitted to the Department of Medicine, Faculty of Veterinary Science, University of Pretoria for examination and treatment. The owner had observed that the animals tended to lie down, were slightly bloated and showed openmouthed breathing.

The remaining avocado branches Persea americana Mill. cf. var. drymifolia (Schechtend. & Cham.) S.F.Blake i.e. the Fuerte strain of avocado pear, were removed from the camp 15 d after their introduction. The leaves of this particular strain are anisescented when crushed and this characteristic has been retained by the dry specimen for over a year.

A total of 4 animals out of 15 were lost, the last one being Goat 4 which succumbed 16 d after the first deaths occurred.

On admission, both goats were clinically examined. Goat 3 had a temperature of $39,2^{\circ}$ C, a heart rate of 140 min⁻¹ and a respiratory rate of 30 min⁻¹. The intensity of the respiratory

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Fig. Myocardial necrosis and granular degeneration showing contraction bands and myolysis in goats with suspected avocado poisoning. HE X400

sounds increased and she coughed occasionally. Goat 4, which was heavily pregnant, had a temperature of 39,5°C, a heart rate of 180 min⁻¹ and a respiratory rate of 60 min⁻¹. The intensity of her respiratory sounds also increased and she tended to lie down. Both had a decreased appetite, but no other abnormalities were found.

Haematological examinations demonstrated a leucocytosis due to an increase in both mature and immature neutrophils. The leucocytosis was more pronounced in Goat 4, mainly due to larger numbers of immature neutrophils. both goats were normal. Blood glucose concentrations in Goat 3 and 4 were respectively 5 and 3,8 mmol 1⁻¹.

In view of the post mortem findings on Goats 1 and 2, muscle enzyme levels were determined in Goats 3 and 4 (Table 1).

Goat 4 went into partus seven days after admission. She developed a dystocia and a live lamb was subsequently removed by means of a caesarian section under local anaesthesia. Although the ewe appeared quite strong after the operation, she died within hours and a necropsy was

 Table 1:
 Serum concentrations of muscle enzymes in goats suspectedly poisoned by avocado pear (Persea americana)

Enzyme μ <i>θ</i> '25°	Goat 3	Goat 4
Creatinine kinase	640	12750
Lactate dehydrogenase	655	5375
Hydroxybutyrate dehydrogenase	368	2705

Toxic granulation of the neutrophils and monoblasts was present in both goats

The faeces of both goats were normal. Urine could only be collected from Goat 4. This was examined by means of a reagent strip for urinalysis (Multistix, Miles Laboratories), and had a pH of 8,5, an S.G. of 1,010 and was positive for protein (4+), blood (2+) and ketones (3-4+).

A specific clinical diagnosis could not be made on the available information. The mild ketosis in the older goat was regarded as secondary and it did not cause severe clinical abnormalities. Serum creatinine concentrations in

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performed. Goat 3 made an uneventful recovery and was discharged.

In the 3 animals which were examined severe pulmonary oedema, hydrothorax, hydropericardium and a varying degree of ascites, were constant findings. Hyperaemia of the rumen wall was also seen in all the cases, while Goats 1 and 4 also showed hyperaemia of the intestinal wall. Perirenal oedema was present in Goats 2 and 4, while petechiae were present on the serosal surfaces in Goat 2, and in the pancreas of Goat 1. Moderate congestive hepato- and splenomegaly were also seen. Except in Goat 4 where the myocardium had a mild, greyishwhite mottled appearance, no specific myocardial lesions were noticed.

Light microscopical examination of sections stained with haematoxylin and eosin (HE), revealed congestion of the myocardium and multifocal areas of granular degeneration, necrosis and rarifaction of myocardial fibres in Goats 1 and 2 (Fig. 1). These lesions were present in the ventricular walls and the interventricular septa and were characterised by the presence of karyopyknosis, contraction bands, vacuolisation and lysis of the sarcoplasma. In certain areas, this latter change left empty sarcolemmal tubes. The presence of necrotic myocardial fibres were confirmed with the Modified Cardiac Necrosis staining technique⁵. In Goat 1 this lesion was most striking in the subendocardial area, while it appeared more diffuse in Goat 2. Stasis of neutrophils were present in some blood vessels, while a few myocardial fibres showed reduplication of nuclei and giant cell formation. Sections stained with the Mason's Trichrome method revealed multifocal areas of early fibrosis. In Goat 4, which had survived 7 d longer since first exposure to the avocado leaves, the fibrosis was more advanced and mature, while the acute necrosis and cellular infiltration were less striking. Other abnormalities included congestion and oedema of the lungs, centrilobular hepatic congestion and congestion of the gastrointestinal tract. A mild nephrosis characterised by severe cellular swelling was evident in all 3 cases.

Varieties of avocado vary in their toxicity to animals and it would appear that the syndromes caused by them may differ. In the outbreak reported here, a cardiac-failure syndrome was observed. The clinical and pathological findings correlate well with those reported by Grant et al4 in which the Fuerte strain of avocado was also involved. More recently, however, it was shown that mastitis was the primary lesion in cases poisoned by the Gautamalan variety12. Unfortunately, in these animals the hearts were not properly examined and as they showed subcutaneous oedema and coughing, cardiac involvement cannot be ruled out. Clinically there was no evidence of mastitis in the outbreak caused by the Fuerte variety reported on in this paper. Unfortunately, mammary tissue was not examined histologically. It would therefore appear that the primary syndrome induced by different varieties may differ.

The toxic dose of leaves in the case of avocado poisoning is unknown. From our own observations and from experimental evidence⁴, it would seem that the leaves are toxic at a relatively high dose in the case of farm animals, but that low doses of the fruit might kill cage birds⁶. It will be difficult to distinguish the macroscopical pathology from that seen in cases with heartwater. The most important differential feature is the presence of varying degrees of splenomegaly due to lymphoid hyperplasia and the presence of the causative organisms in brain smears. No *Cowdria ruminantium* organisms were detected in brain smears of any of the 3 cases.

The histopathological lesions can easily be confused with that of gousiekte, *Cotyledon* poisoning, chronic gifblaar poisoning and ionophore toxicity. The history, and proof that avocado had been eaten, will be of great value in reaching a final diagnosis. Appropriate chemical analysis should exclude ionophore poisoning.

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