

CESTRUM POISONING IN A YOUNG HORSE — A CASE REPORT

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INTOXICATION D'UN POULAIN PAR *CESTRUM*: ETUDE D'UN CAS

Résumé

On a signalé un cas d'intoxication aiguë d'un poulain par *Cestrum aurantiicum*. Il y avait chez l'animal des changements dus à l'intoxication dans le foie, les reins, les muscles du squelette et le système nerveux central. Il y avait également des changements hématologiques et biochimiques contrairement aux cas d'intoxication par *Cestrum diurnum* observés chez des chevaux. La plante qui est d'habitude utilisée pour faire des clôtures semble provoquer très souvent la mort soudaine des chevaux et des bovins. Il faudrait donc entreprendre d'autres études pour connaître l'agent responsable, les symptômes, les changements hématologiques, biochimiques et pathologiques chez les chevaux nourris de cette plante.

Summary

A case is reported of acute *Cestrum aurantiicum* poisoning in a young horse in which there were signs due to toxic changes in the liver, kidneys, skeletal muscles and the central nervous system. There were also hematological and biochemical alterations unlike those reported for *Cestrum diurnum* poisoning in horses. The plant, which is commonly used for hedges in this area is proposed to be a cause of many sudden deaths in horses and cattle. Consequently, further studies are warranted to determine the active agent, anticipated clinical signs, hematological, biochemical and pathological alterations in horses fed on this plant.

INTRODUCTION

Cestrum plant has previously been reported to grow abundantly in Limuru area and other regions of the former Kenya White Highlands⁽¹⁾. It was responsible for acute poisoning in cattle leading to sudden death. Other workers have implicated *C. diurnum* as the causative agent of hypercalcaemia and calcinosis in Florida horses and cattle. These had a chronic debilitating disease characterized by progressive weight loss and lameness of increasing severity. The toxic principle is a 1, 25-dihydroxy vitamin D3-glycoside which interferes with calcium and phosphorus metabolism^(2,3).

This report describes a case of acute *Cestrum* poisoning in a young horse in which there were signs of intermittent episodes of abdominal pain and lateral recumbency, and central nervous system

depression. Liver function was affected with levels of SGOT decreased to 0 for 5 days. There were signs of renal and skeletal muscle disfunction.

History

A two and a half year old colt from Tigoni, Limuru, was admitted to the large animal clinic, Department of Clinical Studies, University of Nairobi, on 13 June 1990. It had a rather protracted history, the owner later admitting that the animal fed on *Cestrum* plants that had been cut from a hedge.

Physical examination

The colt exhibited intermittent episodes of abdominal pain and lateral recumbency, anorexia, congestion and icterus, hematuria, profuse sweating and edema around the neck and the supraorbital fossae. The hind limbs were weak

and the animal continuously shifted weight. Very many ticks and tick-bite scars were present all over the body. The rectum was empty and coated with mucin.

Initial hematological examination indicated a neutrophilic leukocytosis and no hemoparasites. A fecal sample contained 200 e.p.g. of strongyle eggs.

An exploratory laparotomy was performed after induction with an intravenous injection of 3 g thiopentone and general anesthesia maintained with Halothane — oxygen mixture in a closed circle system. There were subserosal hemorrhages on a portion of the ileum and a mild twist of intestinal loop which was corrected and the incision closed routinely.

Initial signs of colic, lateral recumbency and CNS depression were still present. Almost predictably, the colt would approximate the limbs contiguously shift weight, tuck up the abdomen and go into sternal recumbency. It would then stiffly stretch its limbs while in lateral recumbency. In this state the patient would profusely sweat, pant show a rapid respiratory and cardiac rates later stabilising to normal. It would take some water and a bit of green grass while in lateral recumbency. After 3-5 hours it would stand on being assisted into sternal position and would otherwise pass for a normal horse. This it was able to do for 15-30 minutes and then go into lateral recumbency. Towards the end of the first week the colt was fully recovered save for some mental depression.

Hematology and Biochemistry

Biochemical findings from clotted blood samples collected daily for the period the colt was hospitalized are shown in Table 1. There was a persistent mature neutrophilic leukocytosis. A voided urine sample collected in the course of convalescence was found to have acidic pH(5) and contained 250 erythrocytes per decilitre thus confirming the cause of the red urine as hematuria.

The patient died of evisceration following suture dehiscence and the pathologi-

cal diagnosis was that of acute peritonitis, bronchopneumonia and septisaemia.

Treatment

There is no specific therapy for *Cestrum* poisoning. To counteract secondary bacterial infection, 30 mls of Combiotic (Pfizer Agricultural Division New York N.Y. 10017-200,000 IU of procaine penicillin G per ml per ml and 250 mg of dihydrostreptomycin) was administered intramuscularly daily for 14 days. A metabolic stimulant — catosol 15 mls intramuscularly and Multivite orally were given to restore electrolyte imbalances. 500 mls of liquid parafin was administered orally for 3 days to aid in gut evacuation. Decubitous wounds were kept clean and an aerosol preparation of oxytetracycline and gentian violet applied.

Discussion

A case is described of acute *Cestrum* poisoning in a young horse. The eminent signs included intermittent abdominal pain and lateral recumbency, central nervous system depression, hepatic, renal and skeletal muscle dysfunction. Previous reports have described chronic, almost life-long ingestion, leading to a debilitating disease in Florida horses and characterized by progressive weight loss, lameness of increasing severity and deposition of calcium in soft tissues. In this report the calcium levels were within the normal range unlike the previous report., but agrees with the literature on enzootic calcinosis⁽⁴⁾. Phosphorus was not assayed due to lack of reagents in our laboratory, but is expected to rise in case of *cestrum* poisoning.

Available literature reported no change in the levels of alkaline phosphatase, total protein, albumin, cholesterol, uric acid, bilirubin, CPK, LDH, SGOT, BUN, glucose, chloride, erythrocytes, leukocytes hematocrite and hemoglobin⁽⁴⁾. In this case alkaline phosphatase was increased possibly due to gastrointestinal irritation. CPK was increased following prolonged recumbency, leading to skeletal muscle ischaemia, degeneration and necrosis.

Table 1: Biochemical findings of daily blood samples from the colt since admission

Day	1	2	3	4	5	6	6	7	8	9	10	11	12	Normal
AP (IU/L)	604	577	226	262	513	460	500	657	359	306	312	324	229	95-233
SGOT (IU/L)	13	0	0	0	0	1	0	15	49	167	188	206	239	200-400
CPK (IU/L)	–	–	3107	2771	0	857	–	36	170	112	71	80	101	65
Bilirubin (mg %)	6.8	3.8	3.8	2.8	4.0	3.3	5.5	–	2.9	2.7	–	–	3.2	0.2-2.0
BUN (mg %)	–	29	37	42	38	37	42	23	24	29	35	44	35	10-20
TPP (mg %)	–	6.55	5.34	6.72	6.05	6.52	6.39	6.20	7.44	7.10	6.74	7.51	6.51	5.7-7.9
Albumin (mg %)	–	2.58	2.58	2.43	2.63	2.63	2.38	2.35	2.95	2.68	2.96	3.48	2.59	2.33-3.85
A:G ratio	–	0.77	0.93	0.57	0.77	0.68	0.59	0.61	0.66	0.61	0.78	0.86	0.66	0.62-1.00
Calcium (mg %)	–	7.68	8.84	–	–	8.46	8.08	10.5	9.08	7.54	11.3	10.4	9.41	11.2-13.8

*Blood and Radostits. Sixth Ed. Bailliere Tindall, a division of Csell Ltd. 1 St. Anne's Road, Eastbourne BN21 3 UN.

AP=Alkaline Phosphatase. SGOT=serum glutamic oxaloacetate. CPK=creatinine Phosphokinase.

BUN=Blood urea nitrogen. A:G=albumin:globulin ratio.

– indicates that the test was not done on this day.

SGOT was acutely decreased to 0 for 5 days, later being restored to near normal. The cause of this decrease is unclear to us but is somehow related to the cestrum poisoning. Bilirubin was also elevated and this may have been due to starvation or liver insult.

There are differences in the clinical, hematological and biochemical manifestations of poisoning with *Cestrum diurnum* and *Cestrum aurantiacum*. Although the toxic principle in *Cestrum diurnum* and *Solanum malacoxylon* has been identified as a 1, 25-Dihydroxy vitamin D3 glycoside⁽⁵⁾, the toxic substances in *Cestrum aurantiacum* have not been fully characterized and requires further elucidation.

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