Laboratory, clinical and pathological aspects of intoxication by bufotoxin in a canine – case report

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Abstract: Toxicosis caused by Bufotoxins has worldwide occurrence, frequently in rural areas and they are an emergency in clinical routine of small animals. The poisoning has clinical manifestations predominantly cardio toxic, evolving rapidly and can lead to death in a few hours. This report aims to describe an intoxication case by toad venom in a canine in Pelotas municipality, Rio Grande do Sul state, Brazil. For being an urban environment and the occasional presence of toads, there are no reports of accidents per Bufotoxins in this region. Furthermore, after contacting the venom, the dog had a grave neurological condition after contacting the venom, and then a severe depression. Although using all the therapeutic resources, the patient died within 36 hours. The results of hematological tests, liver and kidney blood biochemistry, and abdominal ultrasound and anatomo pathological findings have confirmed the severity of intoxication.

Keywords: Intoxication, toad, Bufo toad, toad toxin, zootoxins, bufotoxicosis

INTRODUCTION
Accidents per Bufotoxins are a toxicological emergency in a clinical routine of small animals. Several active principles of toxic nature compose the toxin, causing acute intoxication, severe clinical profile and rapidly evolving, irreversible organic lesions and high mortality. Poisonings occur accidentally, preferably with young animals and in rural areas. The toxicosis occurs worldwide, with higher prevalence in regions populated by genus Bufo toads. It is reported the existence in Brazil of 849 identified species and almost 500 species are endemic [1]. However, very few reports are found about the prevalence of these intoxications in Brazil, and no report was found about this toxicosis in the region where this study was conducted.

CASE REPORT
A male canine undefined breed, 7 months old, 22 kg, arrived for attendance at Hospital of Veterinary Clinics, Pelotas, Brazil, about 12 hours after killing and snapped up one toad Bufo genus. The patient fed preferentially with commercial feed, was vaccinated with polyvalent vaccine and dewormed periodically to control endo and ectoparasites. On clinical examination was observed body temperature = 37.5 ° C, good general condition, normal hydration, congested mucous membranes, lymph nodes unchanged, ptyalism, normal heart and respiratory rate, restlessness, mydriasis, soreness, generalized tremors, motor in coordination and difficulty in remaining standing. At evaluation he has not shown abdominal pain or lesions in the oral cavity. After the blood and urine collect for laboratory analysis, it was made an intravenous administration of diazepam (0.5 mg / kg), promethazine subcutaneously (1 mg / kg) and fluid therapy with Ringer Lactate solution and furosemide (2mg/kg). About two hours after the patient showed again agitation and sensitivity to light and noise. After 12 hours of treatment the patient presented cyanotic lesions on the tongue, heart and respiratory rate decreased, cardiac arrhythmia and severe depression. The death occurred about 36 hours after contact of the patient with bufotoxin. It was found in the left posterior limb an irritative hyperemic and alopecic cutaneous lesion. For the helping in the conduct of the patient, hemogram was requested, which was done in the Clinical Laboratory of UFPEL. Concerning the results observed in hematological analysis, no changes were detected in the erythrocyte indexes. In total plasma protein and fibrinogen dosage the indexes were within the normality parameters. In the leucogram, it was detected a mild leukocytosis, by segmented neutrophils without the presence of a left shift. The blood biochemistry has not shown changes in levels of ALT, alkaline phosphatase, albumin, creatinine and urea. The physical, chemical and urinary sediment tests showed no alterations. The ultrasonography examination of the abdominal cavity revealed liver hypochojenicity. A histological analysis found the presence of periportal and centrilobular...
hepatocellular necrosis and congestion of hepatic sinusoid.

**DISCUSSION**

Toxicoses by Bufotoxins are frequent in young canines and usually occur at night and in rural areas, when the toads come out of their hiding places [2]. Intoxications are accidental and occur due to the release of active toxins stored in the parathyroid glands of toads. Bufo genus to be compressed in the mouth of the predator [3, 4]. Intoxications are rare in urban areas, especially in the coverage area of our study, where are rarely found these amphibians. In our report we deal with a young patient, domiciled in closed residence in the urban area of Pelotas, Brazil, south region in Rio Grande do Sul state, Brazil. At nightfall it was seen by the owner that the dog had killed and carried in his mouth a Bufo toad. These intoxications are characterized by grave cardio toxic condition due to inhibition of the sodium-potassium pump of the cells of the heart muscle [5], neurologic, respiratory and digestive signs [6], and the degree of toxicity varies according to the specie of Bufo involved in the toxicosis [7]. Local symptoms due to contact of the toxin with the skin and mucous membranes are also reported [8]. In this study there was a prevalence of neurological signs as described previously [9], in addition to an initial necrotic process of the tongue 24 hours after intoxication. The absence of epidemiological casuistry in the region, associated with a clinical condition predominantly neurological, acute and with a rapid evolution can conduct the veterinarian to suspect chemical intoxications, iatrogenic or some infectious diseases, unless the history reveals the contact of the patient with these amphibians. Undoubtedly, the information of the patient's contact with the toad was decisive in diagnosis and therapy. The hyper acute character of intoxication, the severity of symptoms and the rapid development of toxicosis lead us to believe that this is a toxin potentially toxic. The leucogram results are consistent with acute toxic context established. Although the pathology has proven severe liver damage, the results of serum biochemical analyzes did not show alterations in liver enzymes. We believe that at the time of blood collect was not enough time to register such alterations, although the damage had already occurred. However, it can be seen by examining abdominal ultrasound the occurrence of diffuse liver damage, presenting hypo echoic, and suggesting acute hepatitis with necrosis areas, which can occur in cases of poisoning [10]. Moreover, mesenteric lymph nodes with increased volume were displayed. The absence of alterations in the creatinine and urea levels are expected, since there is not nephro toxicity reports in the literature [11] and were not found in this organ lesions by anatomic-pathologic examination.

**CONCLUSION**

Bufotoxins accidents are rare in urban centers, especially among domestic canines. The complexity of components and degree of toxicity of these toxins cause acute intoxication, grave clinical condition with rapidly evolving, and can lead to death. The absence of clinical case reports in the studied region underestimates the prevalence and importance of this toxicosis, which requires rapid diagnosis and treatment, in order to minimize the damage from intoxication and prevent the patient's death.

**REFERENCES**

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