Confirmed hypoglycin A toxicosis in two gnus

Renaud B.[1], Kruse C.J.[2], François A.C.[1], Brisson L.[3], Boemer F.[4], Gustin P.[1], Votion D.M.[1]

1. Department of Functional Sciences, Pharmacology and Toxicology, Comparative veterinary Medicine, FARAH, ULiege, Belgium
2. Department of Functional Sciences, Physiology and Sport Medicine, Comparative veterinary Medicine, FARAH, ULiege, Belgium
3. Zoo de Beauval, 41110 St Aignan, France
4. Biochemical Genetics Laboratory, Human Genetics, CHU Sart Tilman, University of Liège, 4000-Liège, Belgium

Introduction

In horses, the ingestion of seeds or seedlings from *Acer pseudoplatanus* (sycamore maple) led to frequently fatal acute syndrome named atypical myopathy[1,2]. This intoxication is caused by toxins present in seeds of many *Acer* species[3]. Among the known toxins, hypoglycin A (HGA) has also caused fatalities in Père David's deer and camels kept in zoological parks[4,5].

In spring 2021, two gnus (*Connochaetes taurinus taurinus*) exposed to *A. pseudoplatanus* in their enclosure presented severe clinical signs compatible with a diagnosis of atypical myopathy. To confirm, for the first time, cases of HGA poisoning in *Bovidae*, we searched for HGA and its toxic metabolite (*i.e.*, methylenecyclopropylacetyl-carnitine; MCPA-carnitine) in blood of diseased animals as well as for biochemical alterations similar to those found in equine atypical myopathy[6].

Materials & Methods

Animals

All procedures were in accordance with national and international animal welfare guidelines. The two diseased animals were found in lateral recumbency with signs of depression and tremors.

Samples

Serum samples from the diseased animals were obtained for diagnosis while control gnus (Fig. 1) samples were selected from a bank of samples from the zoo that had been taken previously (in 2020 & 2021).

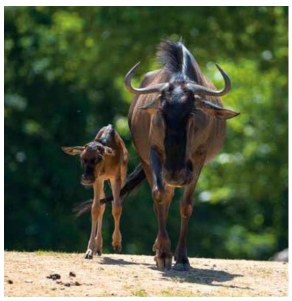

Fig. 1 – Gnus of the Beauval Park

Laboratory tests

Serum activities of creatine kinase (CK) and other biochemical parameters were determined by standard laboratory methods.

Hypoglycin A Assay

Hypoglycin A assay was performed on serum using an aTRAQ® kit for amino acid analysis of physiological fluids as previously described[7].

Methylenecyclopropylacetyl–carnitine

The separation and determination of MCPA-carnitine was done by UPLC-MS/MS as per Valberg et al. (2013)[7].

Acylcarnitines

Free carnitine and 21 acylcarnitines (C2, C3, C3DC, C4, C5, C5-OH, C5DC, C6, C8, C8:1, C10, C10:1, C10:2, C12, C12:1, C14, C14:1, C16, C16:1, C18 and C18:1 -carnitine) were quantified in serum by tandem mass spectrometry according to the method used previously[6]. This acylcarnitines profile is known to be severely modified in equids suffering from atypical myopathy.

Results

Serum activities of CK (gnu 1: 15,000 IU/L; gnu 2: 16,800 IU/L) confirmed a rhabdomyolysis syndrome. In control animals, all blood tests were within normal range.

Both diseased animals tested positive for HGA and MCPA-carnitine. All controls except one (positive for HGA) tested negative for these parameters (Table 1). The control positive for HGA was exposed to *A. pseudoplatanus* in its enclosure.

The acylcarnitines profile was severely altered with an increase in lipid metabolism intermediates as seen in equine atypical myopathy[6].

Animals	Sampling Date (dd/mm/yyyy)	HGA (µmol/L)	MCPA-carnitine (nmol/L)
Control 1	14/04/2020	None	None
Control 2	04/03/2021	None	None
Control 3	16/03/2021	None	None
Control 4	01/04/2021	0.16	None
Diseased 1	29/04/2021	0.28	0.30
Diseased 2	29/04/2021	0.67	4.67

Table 1 – Laboratory findings. Limit of quantification of quantification for HGA: 0.090 µmol/L; Limit of detection for MCPA-carnitine: 0.001 nmol/L. *See text for abbreviations*

The evolution of both diseased gnus was good with disappearance of clinical signs within 3 days after removing access to the toxic plants.

References

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Discussion and conclusion

We describe for the first time HGA poisoning in *Bovidae* exposed to *A. pseudoplatanus*. Moreover, this poisoning seems to follow a similar pathophysiological pathway to the one described in atypical myopathy with equids. The prevention of HGA poisoning in animals kept in European zoological parks is a challenge due to the ubiquitous nature of this tree and the use of other potentially toxic ornamental maples.