INTRODUCTION

Nutritional supplements are commonly given by their owner to their pets and equine athletes alike. Natural substances are often considered more benign than medications despite little or no factual basis for that belief. The first presumptive case of equine microcystin toxicity due to a commercial supplement underscores the potential risk of supplement use and highlights this differential diagnosis for fulminant equine hepatic failure.

BLUE-GREEN ALGAE SUPPLEMENTS

Reported equine deaths attributed to algal blooms date back to 1878, but the recent use of blue-green algae (Aphanizomenon flos-aquae) supplements may put more horses at potential risk of ingestion of the deadly hepatotoxin as well as others. A. flos aquae can actually produce a multitude of potentially lethal toxins including anatoxin-a, saxitoxin, cylindrospermopsin, and BMAA22 as well as co-exist with Microcystis aeruginosa, which can produce microcystin. Microcystin associated liver necrosis has been documented in humans, mice, rats, guinea pigs, sheep, cattle and dogs. Most microcystin toxicoses result from contaminated water consumption; however, important exceptions exist including recent water contamination in a dialysis center that led to twenty-six human deaths from liver failure.

In the United States A. flos aquae are harvested during algal blooms in an open environment making inadvertent collection of M. aeruginosa, a microcystin-producing blue-green algae, and microcystin a real concern. In 1996 85/87 blue green algae supplements derived from this lake were contaminated with microcystin and 72% of those samples exceeded the Oregon Health Department 1 µg/g limit (reaching up to 16.4ug/g) a limit based on the World Health Organization recommendation on drinking water for people. A case of canine hepatic failure was attributed to high levels of microcystin detected in an A. flos aquae supplement harvested from Upper Klamath Lake in Oregon.
MICROCYSTIN

No established toxicity data for microcystin exists in horses, and there is wide species variation in toxicity depending on species affected and route of administration. For example, the oral LD50 for MC-LR in mice is 10.9 mg/kg versus the intraperitoneal LD50 of 43 µg/kg.\textsuperscript{12,13} No established treatment is available in any species, and fulminant hepatic failure and death is a common fate across most species. The toxin is absorbed in the small intestine, enters portal circulation and is rapidly cleared from the blood. The toxin is not cell permeant and carrier-mediated transport results in accumulation in hepatocytes ultimately leading to hepatic structural breakdown.\textsuperscript{14-17}

DIFFERENTIAL DIAGNOSIS

Several toxic and infectious causes of active hepatocellular necrosis as well as cholestasis more commonly affect the horse than microcystin and include Theiler’s disease, pyrrolizidine alkaloid toxicosis, bacterial cholangiohepatitis with or without cholelithiasis, chronic active hepatitis, haemochromatosis, Fall Panicum (\textit{Panicum dichotomiflorum}), mycotoxins, Alsike clover, or poisonous mushroom (ex. \textit{Amanita verna}). Biopsy and histopathological analysis is needed to differentiate between these differentials.

CASE EXAMPLE

The equine case presented had recently started a new container of a commercial \textit{A. flos aquae} supplement. The horse initially showed signs consistent with mild colic, ileus, and obtundation that despite medical management progressed to fulminant hepatic failure and hepatic encephalopathy leading to euthanasia. Both ante-mortem biopsy and post-mortem histopathological changes were consistent with those caused by microcystin. Analysis of the feed supplement revealed detectable levels of microcystin.

SUMMARY

Microcystin should be considered as a differential diagnosis of fulminant hepatic failure in the horse especially when supplements (\textit{e.g. A. flos aquae}) may increase the likelihood of exposure and ingestion.
References:


