Toxic Causes of Intestinal Disease in Horses

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KEYWORDS

• Toxin • Poisoning • Enteritis • Poisonous plant • Toxic plant • Horse • Equine

KEY POINTS

- Most ingested toxins produce enterocyte damage, but these changes are overshadowed by other clinical and organ-specific lesions.
- Arsenic and mercury toxicities are characterized by hemorrhagic enteritis.
- Some plant awns cause oral and mucosal ulceration, irritation, or obstruction.
- Other plant toxins are highly cytotoxic and produce extensive gastroenteritis and colic.
- Some plant toxins produce intestinal fibrosis and colic.

INTRODUCTION

Most toxins are ingested and the gastrointestinal enterocytes are literally the first to be exposed and damaged. However, because many toxins cause extensive organ-specific damage, the gastrointestinal lesions are lost in the shadows of other lesions that have been generally accepted as characteristic of poisoning. A typical example is the microcystin. The microcystins poison horses, cattle, and various other animals including man. They are potent hepatotoxins produced by *Microcystis aeruginosa*, *M viridis*, and *M wesenbergii.* They inhibit cellular serine and threonine protein phosphatases, resulting in excessive phosphorylation of cell regulatory proteins and cytoskeleton collapse. This damage is seen as massive hepatocellular degeneration and necrosis. The disrupted hepatic vascular plate results in hemorrhage and embolization of necrotic and degenerative hepatocytes to distant vessels. Microcystins target hepatocytes as they are absorbed from the portal circulation by bile acid–like receptors. Similar receptors are responsible for microcystin absorption in the ileum where enterocytes have similar receptors. Microcystin's effect in these enterocytes produces extensive degeneration, necrosis, and loss of structure including villous blunting and collapse. However, this damage is rarely identified in poisoned animals because

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it is easily misinterpreted as autolytic change or simply overlooked. Similar intestinal lesions are often overlooked in many other poisonings and it is beyond the scope of this article to describe all the toxins that damage the gastrointestinal system or alter its function. The objectives of this article are to review select toxins and poisonous plants that primarily cause gastrointestinal disease in horses.

TOXIN-INDUCED INTESTINAL DISEASE

Heavy metals rarely poison horses and, when they do, they are generally caused by medicinal or diet misformulation (mercury, selenium), contaminated pastures (lead, zinc, arsenic), or contaminated water (cadmium). All heavy metals produce gastrointestinal lesions with colic and hemorrhagic enteritis; however, most also produce extensive neurologic disease or nephrosis, making the intestinal lesions seem minor in comparison. For example, lead, which is the most common heavy metal that poisons horses, produces severe neurologic disease with prominent laryngeal paralysis. The gastrointestinal lesions are largely overlooked. The exceptions are arsenic and inorganic mercury, which cause severe gastroenteritis with hemorrhagic diarrhea.

Arsenic poisoning is usually caused by contaminated feed, especially contaminated grain. Many poisonings occur when arsenic-containing chemicals are used as wood preservatives or as dressing on seed grains. Other intoxications occur when arsenic-containing insect dips, orchard insecticides, rodenticides, or arsenic-containing medications are inadvertently ingested or when arsenic-containing medicinals are used inappropriately.[1,2] Arsenic-induced gastroenteritis is characterized by severe necrosis, ulceration, and hemorrhage that results in dehydration, electrolyte imbalances, and acidosis. Poisoning is generally identified by associating clinical signs with a kidney arsenic concentration of greater than 10 ppm. Chronic poisoning can be best characterized using hair analysis. Toxicity is produced when arsenical salts react with cellular sulfhydryl groups on proteins and enzymes, disrupting cellular metabolism, including oxidative phosphorylation. Some arsenicals may directly inhibit oxidative phosphorylation by replacing phosphate in those reactions. Arsenical valence is critical to both site and severity of toxicity. Dose and duration determine the clinical syndrome. High doses generally produce neurologic signs with sudden death. Lesser doses produce gastroenteritis with fetid hemorrhagic diarrhea. Disease may become more severe over several days with neurologic signs and death within 5 or 6 days. Lower doses produce chronic disease affecting mostly the skin and hair, and at times producing chronic conjunctivitis with ulcers. Treatment includes removing the source of exposure and decreasing absorption using laxatives, sodium thiosulfate, or dimercaprol.

Mercury poisoning is uncommon today, because its toxicity is well-known and its use has been discontinued in most medicinal, manufacturing, and industrial processes. Rare equine poisonings have been reported to occur when organic mercuric fungicides were used as seed preservatives or inorganic mercury was used in some blistering agents. The mercury salts are corrosive, producing extensive gastrointestinal necrosis and hemorrhage along with renal tubular necrosis. When mercuric salts were used medicinally, oral ulcers were historically used to monitor “effectiveness” of the purported treatment. Clinically, poisoning is characterized by diarrhea with anorexia and nervousness. There are also clinical urinalysis changes of renal failure including glycosuria, proteinuria, phospaturia, isothenuria, polyuria with granular casts, and increased urinary gamma-glutamyl transpeptidase, alkaline phosphatase, and amino aspartate transferase activities. Histologic changes include extensive renal