

# **Inflammatory myocardial disease and toxicity in horses – a recent update**

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Myocarditis is a focal or diffuse inflammatory reaction of the myocardium with degeneration or necrosis of myocytes, followed by an infiltration of inflammatory cells. Inflammatory myocardial disease or myocarditis is not commonly diagnosed in horses but the use of cTnI has markedly increased the sensitivity to detect myocardial disease. A wide variety of causes can result in myocarditis. The clinical signs, which are related to cardiac electrophysiological and contractile properties, and to underlying disease, are very variable. In many cases with clinical signs and increased troponin levels indicative of myocardial disease the exact aetiology remains speculative.

Aetiological factors are of infectious, toxic, nutritional, infiltrative, hyperthermic, hypoxic or idiopathic origin. Viral infection, such as Influenza, Equine Viral Arteritis virus, Equine Infectious Anaemia, African Horse Sickness, Equine Herpes Virus 1, Eastern Equine Encephalomyelitis, and bacterial infection, e.g. *Streptococcus* spp., *Staphylococcus* spp., *Salmonella*, *Clostridium*, *Borrelia burgdorferi*, piroplasmiasis, *Leptospirosis*, *Neorickettsia risticii*, and also parasites as *Strongylus vulgaris*, *Onchocerca*, have been associated with myocarditis. Myocardial inflammation may result from exposure to cardiotoxins from plants (foxglove, oleander, Adonis, Eonymus, lily of valley, yew, rhododendron,...), ionophore antibiotics (monensin, salinomycin, lasalocid), heavy metals, cantharidin toxicosis (blister beetle), snakebite envenomation,... Nutritional imbalances, such as due to vitamin E, selenium, copper and molybdenum, and ischemia and heat stroke may also result in myocarditis.

Clinical signs are very variable and range from subclinical to poor performance, exercise intolerant, syncope and sudden death. Clinical signs may be those of underlying systemic disease (fever, colic, weakness, respiratory signs, shock,...) or may be directly related to myocardial damage. Myocardial inflammation may result in enhanced ectopy or vulnerability, impaired conduction, and altered systolic or diastolic function. Enhanced ectopy results in atrial or ventricular premature beats, ventricular tachycardia or ventricular fibrillation. Enhanced or, more frequently, decreased ventricular contractile function may occur and in chronic cases myocardial fibrosis might be present.

In order to make a diagnosis, history is very important (duration of disease, possible contact with toxins, other animals involved,...). Physical exam may reveal dysrhythmias, a cardiac murmur, weak

pulse, oedema. Electrocardiography (ambulatory, exercise, 24-hour) and cardiac ultrasound are crucial to evaluate cardiac function. Cardiac glycosides may result in increased inotropy while many other toxins will produce contractile dysfunction. Complete blood count and biochemistry including cTnI must be performed. In case of suspicion of specific disease or intoxication, serum, urine samples, swabs,... should be taken for further diagnostic tests (digoxin, serology, PCR, toxicology,...). The environment should be checked for possible contact with toxins (plants, ...).

The horse should be rested for at least 6-8 weeks. Therapy aims to treat underlying disease and life-threatening dysrhythmias, improve cardiac function and treat heart failure. In case of intoxication, further access to the toxin must be avoided and activated charcoal or mineral oil should be administered by nasogastric tube in acute cases of oral intoxication. Horses must be closely monitored during the treatment. Horses that recover should always be re-examined before they are allowed to return to exercise.

Below an update will be given by describing a number of cases of myocardial injury that were encountered over recent years.

#### *Lasalocid intoxication*

Accidental lasalocid poisoning occurred on a farm with 81 horses, of which 14 started to show clinical signs after being fed a new concentrate batch. One horse died on day 20 and another on day 27. The most severe cases ( $n = 7$ ), admitted to the clinic on day 29–46, underwent cardiac examination and blood biochemical analysis, including determination of plasma cardiac troponin I (cTnI), at admission and during follow-up. On day 57–70, cardiac examination, including TDI and 2D speckle tracking, cTnI determination or both were undertaken on 72 remaining horses. Short-term effects of lasalocid intoxication included inappetance, lethargy, sweating, and muscular weakness. All 7 horses admitted to the clinic demonstrated signs of myocardial degeneration such as increased cTnI (1.39-816 ng/ml), dysrhythmia and reduced myocardial contractility. Four horses developed ataxia on day 40–50. Five horses died or were euthanized on day 30–370, 2 horses recovered fully and returned to previous athletic use. None of the 72 remaining horses exhibited clinical signs between day 57–70, but 34 had dysrhythmia and 13 had increased cTnI concentrations. After a period of rest, all these horses returned to their previous work. Lasalocid was detected in hepatic tissue of 2 necropsied horses. Lasalocid intoxication induced myocardial and neurological damage. Although uncommon, this should be included as differential diagnosis for unexplained inappetance, signs of depression, cardiomyopathy, and ataxia in horses. TDI and 2DST measurements allowed detection and quantification of LV dysfunction in horses exposed to lasalocid.

### *Atypical myopathy*

Atypical myopathy (AM) is an acute, often fatal, rhabdomyolysis in grazing horses that mainly affects skeletal muscles. Post mortem examinations have shown that myocardial damage also occurs. Limited information is available on the effect of AM on cardiac function in affected and surviving horses. Horses (n = 12) diagnosed with AM, in which cardiac ultrasound and ECG recordings was obtained, were studied. All horses underwent clinical examination, serum biochemistry, electrocardiography, and echocardiography. Four surviving horses underwent the same examinations after 2-10 weeks. All but 1 horse had increased cardiac troponin I concentrations and 10 horses had ventricular premature depolarizations (VPDs). All horses had prolonged corrected QT (QTcf) intervals on the day of admission and abnormal myocardial wall motion on echocardiography. One of the surviving horses showed VPDs and prolonged QTcf at follow-up after 10 weeks. It was concluded that AM resulted in characteristic electrocardiographic and echocardiographic changes and that it was commonly associated with increased cardiac troponin I concentrations and VPDs. In survivors, abnormal cardiac function may be found at follow-up after 10 weeks. Additional research in a larger group of horses is necessary to identify the long-term effects of AM on cardiac function.

### *Other cases*

Additional cases will be presented such as cardiac glycoside intoxication, resulting in ventricular arrhythmia, increased inotropy and/or atrial standstill, suspected iron intoxication with advanced second degree atrioventricular block, and infra-hisian block probably due to lyme disease.