



HEPATIC LIPIDOSIS IN REPTILES

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Hepatic lipidosis is a clinical condition commonly diagnosed in reptiles, particularly in turtles. In fact, it is a metabolic condition rather than a true disease. The factors influencing such condition are the following (Divers & Cooper 2000):

- 1) Fat or too plentiful diets (associated to obesity and increased abdominal fat bodies). Diagnosed in badly fed crocodiles, lizards and turtles (Maccolini 2010).
- 2) Reduced activity. Absence of exercise. Diagnosed in monitors and turtles (Stahl 2003).
- 3) Physiologic disorder. Lack of hibernation. Lack of reproduction. Diagnosed in tortoises and iguanas.
- 4) Chronic hypoxemia and stress. It decreases the glycaemia and increases glucagon. Both factors favour fat release into blood and storage of triglycerides in liver cells (Ware, 1998). Diagnosed particularly in invasive turtles grown wild.

In domestic reptiles, the condition is diagnosed usually when it is progressing and there is high degree of hepatic malfunction. Depending on the species, some reptiles tolerated high degree of lipidosis (turtles) and other are very sensitive (chameleons, monitors). Thus, the diseased reptile show weakness, depression, jaundice and oral mucous membranes paleness, low muscular tone and even hepatic encephalopathy signs. As the disease progresses, development of hepatitis and/or maldigestion are favoured. Diarrhoea (sometimes foul-smelling) and/or biliverdinuria (green dropping eliminated through cloacae due to abnormal elimination of biliverdin that stains faeces and urine) may be found. Biliverdin is the majority bile acid in reptiles, since they lack the biliverdin reductase enzyme required to produce bilirubin.

Diagnostic:

Haematology. Decreased plasmatic proteins, increased bile acids and increased cholesterol, AST, ALT, LDH, alkaline phosphatase and creatin kinase. Glucose levels vary a lot in this condition, increasing or decreasing depending on the case; they are rarely within the normal range. The increased biliverdin (although it is rarely analyzed) is interpreted when observing the stained dropping.

Imaging. The radiology allows observing hepatomegalia in a few species of saurian, and particularly in chameleons. It is virtually impossible to observe it in tortoises. The ultrasonography

simply gives an indirect value of lipidosis, since it is not possible to identify the fat liver by ultrasounds, and specially the various degrees (from asymptomatic to pathologic). The ultrasonography allows ruling out whether the liver takes up the whole coelomic cavity, being the stomach completely covered by the left lobe. Hepatomegalia in such cases is suggestive, although not pathognomonic of lipidosis. Computerized tomography may help in the diagnostic of hepatomegalia and structural change of the liver, although nowadays its use is very limited (Gumpenberg et al. 2011).

Liver biopsy. This is the technique of choice and allows classifying the condition, as well as performing light and electronic microscopy and microbiological cultures if needed. Various approaches are feasible, generally the left side of the animal, since the right lobe has the gallbladder and the hepatic vein, which cannot be damaged. It can be performed by endoscopy (minimally invasive), ultrasonography-guided or by means of conventional surgery. It can be conducted by the biopsy punch technique, simple guillotine or serial guillotine (overlap suture) although given the small size of the liver in such animals, the two first choices are the most usual. In such biopsies, the poor condition of the liver must be considered when administering the anaesthesia.

Necropsy: The large size and weight of the liver attracts attention. The colour is always abnormal and ranges from pale brown to deep yellow. The liver loses the characteristic tension and is friable and soft (Martínez Silvestre & Ramis 2001).

Histology. The excessive collection of lipids in hepatocytes can be categorized in three types of fatty degeneration: 1 (minimal), 2 (moderate) and 3 (high) (Divers 2001). Level 3 uses to take place with other pathologic conditions (even pancreatic fatty degeneration) and it is symptomatic. Level 1 uses to be asymptomatic.

With the usual haematoxylin/eosin staining, variable vacuolization of the hepatocyte, nuclear peripheral marginalization and increased cell size are observed. Sometimes, Küpffer cells are also affected, and inflammatory cells can also be found to some extent spread in the liver parenchyma.

Treatment

It is essential to provide appropriate fluid-therapy, giving hypotonic fluids (glucosaline serum or mixture of 50% Ringer with physiologic serum) at 10 mg/kg/day.

Gastric tube may be needed in cases of extended anorexia, thus allowing not just feeding the animal but also administering the treatment.

Feeding with nutritional support calculated on the basis of the energetic requirements (daily consumption of Kcal) in order to stimulate the systemic lipolysis. Make sure you are providing vitamins B and C (accelerated consumption has been described in cases of lipidosis).

Supplement with amino acids such as carnitine, choline and methionine.

Stimulate lipolysis and metabolic consumption with thyroxine (20 µg every 2 days, PO).

Enhance the protein anabolism with anabolic steroids (Simpson 2006) (nandrolone, 0.5 to 5 mg/kg IM).

Administer hepatic protectors such as silymarin. 30 to 40 mg/kg up to three times a day PO. It is usually used in liver and gallbladder diseases. Used in tortoises. Mainly used to control inflammation and limitation of toxins uptake (Simpson 2006). Therefore, silymarin is not necessarily useful in hepatic lipidosis.

Should nausea and vomit appear, administer metronidazole 150 mg/kg, two doses PO.

If hepatic encephalopathy is suspected, administer lactulose (1 to 5 ml/kg once a day, PO), since it decreases blood ammonium levels, although its effect on the hepatic lipidosis is not yet known.

Ursodeoxycholic acid has been proposed as hepatobiliar stabilized, although it has not yet been tested efficiently in reptiles (Simpson 2006).

The antibiotic of choice in reptiles showing this conditions is ceftazidime (20 mg/kg IM every 72h for 2 to 3 weeks).

The prognosis depends on the degree of disease at the diagnostic.

Therapeutic precautions

In case of hepatic lipidosis, the following precautions must be considered:

1) Avoid hepatotoxic agents use in reptiles:

- Antibiotics :Doxycycline, Clindamycin, Erythromycin
- Antivirals: Aciclovir (alternative with A-5021)
- Antifungals: Itraconazole, ketoconazole.
- Antiparasitic: Metronidazole, Dichlorvos, Tiabendazol, Ivermectin
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2) From certain level of degeneration due to hepatic lipidosis, drugs metabolism and storage of minerals, vitamins or oligoelements are affected (Everaert & Vercauteren 2013), which are commonly used in herpetological clinic, which will give rise to unexpected results. Thus, in case of risk of advanced lipidosis, anaesthetics such as ketamine, tiletamine, halothane and acepromazine, and antibiotics such as enrofloxacin or pentoxifylline should be avoided,

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