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HYPERLIPIDEMIA - 3 PAGES

What is hyperlipidemia?

Lipemia, hyperlipemia and hyperlipoproteinemia all refer to increased serum lipids. These are usually triglyceride (TG) and/or cholesterol (CHL).

Hyperlipidemia is diagnosed after a 12 hour fast and blood levels exceed 500mg/dl. Generally speaking, if the initial result was not on a fasted sample, a value >500 should be rechecked on a fasted sample and if still > 500, underlying reasons for the elevated value should be explored.

Most clinical signs occur when triglycerides are >500. Clinical signs could include anorexia, vomiting, diarrhea, necrotizing pancreatitis, lipid keratopathy, arcus lipoides, stromal dystrophy, lipid in the aqueous humor, uveitis, blindness, and lipemia retinalis, cutaneous xanthomata, pruritus, and alopecia, seizures, behavioral changes, neuropathies, and cerebral atherosclerosis. These signs do not usually occur until the triglycerides are >1000. There are many patients who do not have clinical signs even when the value is > 1,000.

How does hyperlipidemia differ from hypercholesterolemia?

Hyperlipidemia produces a visible milky serum. Hyperchylomicronemia is the most common lipid disorder in companion animals. The CM's contain the highest amount of triglycerides (~90%). To test for it store a serum sample overnight at 4°C. CMs rise to form a "cream" layer at the surface.

*If the ***infra***natant is milky, an elevation in VLDLs is likely. **VLDL's** are produced in the liver from endogenously derived triglycerides. After being transported to the tissue capillaries, they are catabolized by lipoprotein lipase as are the CM's.

*When there is an increase in both CMs and VLDLs, a fat layer will form above a turbid infranatant. The presence of CM's suggests postprandial hyperlipemia, as the primary source of CM's is dietary fat.

Hypercholesterolemia does not produce visible milky serum. Hypercholesterolemia is generally caused by an increase in LDL and HDL.

Low density lipoproteins (LDL): These are responsible for transporting endogenously synthesized lipids, especially cholesterol, from the liver to the target tissues. After hydrolysis of the VLDL molecule and removal of triglyceride from the core, a short-lived intermediate density lipoprotein is formed that ultimately is processed by hepatic lipase into LDL. In humans ~70% of the cholesterol is carried within the LDL molecule, in companion animals most of the cholesterol is carried in the HDL molecule.

High density lipoproteins (HDL): These are produced in the liver, and are recognized for their ability to remove excess cholesterol from the tissues and transport it back to the liver. Newly formed HDL molecules are secreted by the liver and intestine and bind with unesterified cholesterol released from the peripheral tissue during normal cellular turnover.

Causes of Hyperlipidemia?

1) Postprandial:

In normal dogs and cats, postprandial hyperlipidemia usually persists for 6-12 hours post meal, but even with a high fat diet, the serum triglyceride levels would not be expected to exceed 500 mg/dl. Chylomicrons carry less than 10% of the total cholesterol; therefore food intake would have little impact on the cholesterol concentration seen in the serum in the 6-12 hour postprandial period.

2) Acquired or secondary: Excess concentrations of lipids in the blood stream can result from underlying diseases in which normal lipoprotein metabolism is markedly altered.

*diabetes mellitus

*Cushing's disease,

*protein losing nephropathy

*pancreatitis

*hypothyroidism

*Iatrogenic: megestrol acetate, corticosteroids

If an underlying cause for hyperlipidemia is not found, then familial (primary or idiopathic) hyperlipidemia must be suspected and lipoprotein electrophoresis and/or ultracentrifugation can be done to define the nature of the defect and determine the abundance of the different lipoprotein fractions and their content.

3) Familial or primary or idiopathic:

Idiopathic hyperlipidemia of Schnauzers **typically affects middle to older aged miniature schnauzers, though other purebred and mixed breed dogs (shelties) can be affected. An increased serum triglyceride is seen, with a mild increase in cholesterol, VLDL and visible lipemia. Etiology is unknown but lipoprotein lipase deficiency is believed to be the cause and is suspected to be inherited. Clinical signs may include abdominal pain, diarrhea, seizures and behavioral abnormalities, but many will not show any signs.**

Hypercholesterolemia has been reported in Collies and Shetland Sheepdogs.

Hyperchylomicronemia in cats is an inherited (autosomal recessive) disease described in domestic cats in New Zealand. Clinical signs have been observed after 6-9 months of age and have included peripheral neuropathies, cutaneous xanthomas, and the formation of lipid granulomas in abdominal organs. Most affected kittens will not show clinical signs other than lipemia retinalis. Both triglycerides and cholesterol are significantly elevated. Lipoprotein fractionation shows a marked increase in CM and a smaller increase in VLDL. LPL is present but inactive owing to defective binding sites.

Treating elevated triglycerides:

Commonly Used: Try First

1. Treat underlying disorders
2. Dietary levels of fat between 8-12% DM are recommended for assisting in control of disorder. Some of the diets that have fat levels in this range are Hills r/d, Iams Restricted Calorie, Purina Overweight Management, Royal Canin Calorie Control, Purina EN GastroEnteric.
3. Omega-3 fatty acids may reduce serum cholesterol and triglyceride concentrations by decreasing the synthesis of VLDL and LDL. (10-30 mg/kg PO q 24 hours)
4. We recommend **Royal Canin LF or W/D diet**

Less Commonly Used:

4. Gemfibrozil (Lopid, Parke Davis): Dosages have varied from 200 mg/d to 150-300 mg every 12 hours to 7.5 mg/kg PO q 12h. Cats :7.5-10 mg/kg every 12 hours.

   This is a fibric acid derivative that works by reducing the production of triglycerides and VLDLs; they may also increase HDLs and by doing so, they may reduce LDLs levels.

5. Chitosan is a fiber supplement made from shellfish that reportedly binds lipids in diet and decreases absorption of them. There are no real studies proving efficacy in dogs or cats.

   If using it, give it 30 minutes before a meal and give it separately from the omega fatty acids by several hours. Dosing is somewhat empirical; 1-2 tablets of the Nikken product, which are 150 mg each, has been recommended.

6. Niacin: 50-300mg/day/dog; dose can be divided into 2 daily doses. Niacin acts primarily to reduce hepatic triglyceride synthesis. Certain of the new agents, e.g., acipimox, also act to reduce adipose lipolysis. Niacin has vasodilatory effects and could cause some skin reddening which might be reduced by giving it with meals or using a "no flush" niacin product or a slow-release product. Occasionally dogs seem uncomfortable and may scratch at their face afterwards. Nicotinic acid (niacin) and the fibric acid derivatives act primarily to reduce hepatic triglyceride synthesis and VLDL production.

For Elevated Cholesterol

7. HMG CoA-reductase inhibitors or "statins" (lovastatin, simvastatin, pravastatin, fluvastatin, cerivastatin, atorvastatin) are of less value in treating increased triglycerides, but they may be effective at reducing cholesterol levels by reducing hepatic cholesterol synthesis and so up-regulating LDL-receptor activity. Adverse effects include lethargy, diarrhea, muscle pain and hepatotoxicity.

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