DESCRIPTION

Fatty liver hemorrhagic syndrome (FLHS) is a noninfectious disease characterized by excessive accumulation of fat in the liver and abdominal cavity, causing liver rupture, hemorrhage and sudden death of hens. Death is caused by internal bleeding.

The condition is mostly confined to caged egg-type layers and can result in significant economic losses not only from bird mortality but also from decreased egg production in affected flocks. An epidemiology survey in Queensland showed 40% of caged hen mortality was related to FLHS, the same survey showed that FLHS can occur in cage-free housing systems, but to a much lesser extent than in cage systems. Total mortality due to fatty liver usually does not exceed 5%.

PATHOLOGY

Hens in affected flocks are generally obese (overweight by 20% or more) and experience a sudden drop in egg production. Dead birds typically have paleness of the head, wattles, comb, and skin. Necropsy reveals livers that are enlarged, pale, soft, and engorged with fat. The excess fat in the livers makes them yellow, soft, and friable. The affected liver has lost structural integrity leading to a susceptibility for rupture and hemorrhage. The liver may easily break apart when handled. Large blood clots are found within the abdomen. The origin of the blood clot is from a rupture of the liver capsule. In other instances, the liver is yellow, greasy, and soft. There are generally abundant abdominal and intestinal (mesenteric) fat reserves present.

HISTOPATHOLOGY

Microscopic examination of liver tissue shows liver cells (hepatocytes) that are grossly distended with fat. There may be hemorrhages present. Fat within the hepatocytes are seen as clear spaces (vacuoles) within the cytoplasm of the hepatocytes. The accumulation of fat within the liver weakens the integrity of blood vessels, leading to hemorrhage.

There is an association between FLHS and poor egg shell quality. One important function of the liver is the activation of Vitamin D into its metabolically active form. Serum calcium levels in hens with FLHS was elevated, suggesting interference with the formation of active Vitamin D (1, 25 (OH2) D3) which is vital in the egg shell formation process.
POSSIBLE CAUSES OF FLHS

The exact cause of FLHS is unclear but several factors have been associated with the condition. Factors related to nutrition, genetics, environment and hormonal influence have been proposed. It is suspected that combinations of these factors are needed to cause FLHS²,¹⁰,¹¹.

BODY WEIGHT AND ENERGY BALANCE

When FLHS was first recognized, high-energy density diets were introduced to the industry worldwide and were considered to have contributed to increased prevalence of the disease. Since then, studies have indicated that high-energy diets, especially maize or wheat-based diets, produce higher incidences of FLHS¹²,¹⁴,¹⁵. Feeding high-energy diets to high-producing hens in cages where exercise is restricted was associated with the development of fatty livers and FLHS²,¹⁶.

Layers consuming dietary energy in excess of their requirements will store the energy as body fat. The liver processes the energy into fatty acids to be stored as abdominal fat. FLHS occurs when birds are in a positive energy balance for a long period of time. This relationship is independent of source of energy (i.e. fat or carbohydrate source)⁹. Absolute energy intake is of less relevance than the amount of excess energy available for fat synthesis in the liver. The balance between hepatic synthesis and secretion of lipids is the key point that regulates hepatic and extra hepatic fat deposition in the laying hen¹⁷. In other words, the layer must not be fed energy in excess of requirements, as feeding an excess of energy for an extended period of time leads to obesity and increases fat infiltration of the liver.

Layers housed in cages have lower energy expenditure due to less activity. Less active birds will lower their maintenance energy requirement, resulting in a surplus of energy to be stored as fat. This might explain the higher incidence of FLHS in flocks housed in cages compared to those housed in alternative systems.

ESTROGEN PREDISPOSES THE FLOCK TO FLHS

The hormone estrogen is associated with sexual maturity and stimulates the liver to store more fat for egg yolk synthesis. When the bird comes into egg production, the liver size increases dramatically in response to estrogen levels. A combination of positive energy balance and influence of estrogen predisposes the laying bird to develop FLHS. In force feeding studies, liver hemorrhaging due to FLHS was markedly increased when excess energy intake was combined with exogenous estrogen treatments¹⁸. This emphasizes the need to avoid excess energy intake, especially in the early laying period when birds are in a highly productive state.

HEAT STRESS

The highest incidence of FLHS occurs during the warmer time of year¹⁹,²⁰,²¹. This is likely due to the fact that higher environmental temperatures reduce energy requirements in layers and result in a more positive energy balance which, as previously discussed, is a prerequisite for the development of FLHS.

Birds rely on evaporative cooling during respiration to regulate their body temperature. Excessive fat deposition in the abdomen will obstruct normal breathing action making it more difficult to lose body heat. These birds are more prone to heat stroke and liver rupture.

Figure 3. An extreme case of fatty liver and obesity.

Figure 4. This bird died of liver hemorrhage. The blood clot is visible through the skin over the abdomen. Note the pale breast muscle devoid of blood.
MYCOTOXINS
Mycotoxins, especially aflatoxins, which may contaminate cereals, induce liver lipid accumulation and liver hemorrhages. The use of rapeseed meal in the diet increases the incidence of FLHS, because erucic acid or other toxic metabolites can affect the strength of the connective tissue in the liver, which predisposes to hepatic cell breakdown and hemorrhaging.

LIPOTROPIC NUTRIENTS
Lipotropic nutrients are feed ingredients that promote healthy liver function and the export of fat from the liver. Methionine, choline, inositol, Vitamin B12, biotin, L-tryptophan, carnitine, and selenium are essential for proper liver function and fat metabolism. Supplementation of these nutrients in the diet or in birds' drinking water has been used as a treatment for FLHS with variable success.

PREVENTION STRATEGIES
Avoid excessive body weight development during lay by avoiding surplus energy intake in birds. Feed adequate energy to sustain the birds and optimize their production but not more than that. Surplus energy will be stored as fat, increasing the metabolic burden on the liver. Maximizing energy intake in the early period of lay is essential to support productivity; however, energy requirements will reduce through lay as production reduces, and it may be necessary to reduce diet density to avoid birds gaining excessive weight. Monitoring feed intake and specifying the diets to provide adequate nutrients to the bird is critical.

Limit energy intake through the use of lower energy diets and/or change in feed management. Use of crumbed or pelleted feeds will result in greater feed intake and more energy intake compared to a mash. It is not advisable to feed either crumb or pellets to flocks which are susceptible to FLHS.

Replacement of dietary carbohydrate with supplemental fat has been shown to reduce the incidence of FLHS as long as the energy level of the diet is not increased. Supplemental fat depresses de novo fatty acid synthesis, meaning the liver has to produce less fat for the yolk reducing the metabolic ‘burden’ on the liver. One of the most important aspects in preventing FLHS is to look for signals, decreased egg production, increased body weights and mortality.

Heavier and higher producing birds have been shown to be more susceptible to FLHS. Constant monitoring of the flock for FLHS should involve routine body weight and uniformity checks of flocks throughout the laying cycle (every 30 days). As soon as the birds start showing signs of excessive body weight development, steps should be taken to reduce the energy intake in order to control the body fat percentage before it affects liver condition.

Less uniform flocks are likely to have relatively heavier individuals who are more susceptible to FLHS. Adhering to the breeder-recommended body weight profile in rear and lay is important, but target uniformity must also be achieved.

Post-mortem examination of dead hens should assess the condition of the liver and signs of excess fat deposition in the abdominal region.

Layer diets should contain adequate levels of Vitamin E (50–100 IU/kg and selenium (0.3 ppm) to ensure adequate levels of antioxidants to prevent tissue rancidity. Supplementation with lipotropic agents such as choline (500mg/kg), methionine (0.1%), and Vitamin B12 help to mobilize fat from the liver and support recovery in affected hens.

Avoid stress of any type, especially heat stress, as this condition precipitates and/or accentuates the occurrence of FLHS and so the usual management considerations for alleviating such conditions are relevant.

Calcium deficiency has also been associated with FLHS. This deficiency can be addressed through the addition of large-particle calcium and Vitamin D to the feed rations, which will allow the birds to eat more calcium without over-consuming the energy component of the feed.
REFERENCES


5. Leeson, S and J.D. Summers, 1995: Poultry Metabolic Disorders and Mycotoxins; Fatty liver hemorrhagic syndrome pp. 64.


12. Couch, J.R., 1956. Fatty liver in laying hens – a condition which may occur as a result of increased strain. Feedstuffs. 28: 46-54.


