

Bovine Fatty Liver Syndrome

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DESCRIPTION

Bovine fatty liver syndrome is a metabolic disease of heavily lactating dairy cows. The ailment is sometimes called "fat cow syndrome", however, a cow need not be visibly overweight at calving time to be affected by fatty liver.¹ Cows with fatty liver syndrome are more likely to suffer from periparturient problems such as milk fever, ketosis, mastitis and metritis. They tend to respond poorly to therapy and they often relapse. Their milk production is often reduced, they may have reproductive disturbances, and they may die.

Factors involved in the pathogenesis of fatty liver syndrome include feeding excess energy to cows late in lactation and/or during the dry period, mobilization of large amounts of stored fat to support high milk production in early lactation, reduced feed intake by cows which are too fat, and the supply of protein available for fat mobilization. There may also be an inherited tendency to develop fatty liver.

When fatty acids are mobilized faster than they can be broken down and utilized by the animal, they are deposited in skeletal muscle fibers and in vital organs including liver, kidney, heart muscle and adrenal gland. If insufficient dietary protein is available for maintenance of the high rate of fatty acid mobilization, then skeletal muscle proteins will be scavenged and used for this purpose.¹

Hepatic function can be severely impaired by fatty infiltration of the liver. One of many results of this impairment of liver function is a drop in serum albumin levels. Low serum albumin and reduced liver metabolism of sex steroids have been shown to be associated

with reduced fertility in cows suffering from fatty liver syndrome.² Fatty infiltration of other vital organs is thought to increase the cow's susceptibility to infectious and metabolic diseases.³

INCIDENCE AND PREVALENCE

Fatty liver is usually a herd problem.⁴ It is commonly seen in high producing dairy cows, especially those which are loose-housed and fed free choice. It is unusual to see this condition in stanchioned cows which are fed on an individual basis. Anywhere from 50 to 90 percent of the cows in a problem herd may be affected, and mortality among these cows may reach 25 percent.⁴

The economic losses associated with fatty liver syndrome can be considerable, especially where a large number of cows are affected. Losses may be obvious, as with cows that die, or more insidious. Sources of economic loss include:

- mortality
- decreased milk production
- impaired reproductive ability
- veterinary care for periparturient problems, infectious diseases, reproductive failures, etc.
- feed dollars wasted by overfeeding heifers and dry cows.

CLINICAL DESCRIPTION

The fatty liver cow may initially be presented with one of many diseases of periparturient and lactating cows. Conditions such as milk fever, mastitis, metritis and retained placenta all occur more often in fatty liver cows. These ailments have a tendency to be more severe and less responsive to treatment in these cows. These are seen clinically as the milk fever cow that stays down, the ketotic

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cow that doesn't respond, the chronic and recurrent mastitis cows, and the repeat breeder that defies all treatment. They often relapse or go from one problem to another, their milk production drops off, and they frequently die.

While the particular cow in question may no longer be visibly obese at the time she is seen clinically, observation of the herd may reveal other animals, particularly dry and almost-dry cows, which are overweight.

DIAGNOSIS

Diagnosis of the fatty liver cow is usually difficult. Knowledge of a herd problem, assessment of management practices, and observation of the physical condition of other cows in the herd, especially dry cows, may suggest the possibility of fatty liver disease.

Useful laboratory findings include persistent ketonuria, increased BSP retention time, increased SDH and SGOT levels, and low serum albumin levels. Hyperglycemia with glucose intolerance, hypokalemia, and lowered serum glucocorticoid levels are all common laboratory findings.⁶

Further information can be obtained by performing a percutaneous liver biopsy. Several samples may be taken and submitted for histopathological evaluation, and the tissue may be assayed for lipid content using copper sulfate solutions of two different specific gravities, 1.025 and 1.055, and water. Based on the bouyancy of the sample in each of the three media, lipid content may be assessed as >34%, 25–34%, 13–25%, or <13%.⁷

Unfortunately, many diagnoses are made at post-mortem; either the cow succumbs to some secondary problem, or she is culled for being a downer, a chronically sick cow, or for being open too long.

TREATMENT

Treating the cow with clinically severe fatty liver syndrome is almost invariably unrewarding. As with most ailments for which there is no definitive cure, numerous treatment regimes have been proposed. The approach to treatment will be divided into three categories for purposes of discussion: treatment during a crisis, treating the diseased liver, and prevention of future cases.

Treatment During A Crisis

Treat the cow for the mastitis, milk fever, or other secondary problem in the usual ways, while confirming the diagnosis of fatty liver. Therapy should be quite vigorous, and usually must be prolonged—seven to ten or more days of active treatment. Glucose plays a key role in this therapy and should be administered in large amounts—10% or 50% dextrose IV, via a slow drip if possible, at a rate of 60 grams per hour, or the equivalent dose two or three times daily.⁶ Insulin and potassium chloride may be administered to fight cellular glucose intolerance. Protamine zinc insulin (150–200 units) is administered subcutaneously every 36–48 hours.⁶ Adrenocorticotrophic hormone, used to stimulate endogenous corticosteroid release, is preferable to administering exogenous corticosteroids. Intramuscular doses of ACTH are given as follows: 600 units on day 1, 400 units on days 2 and 3, none on day 4, and 200 units on day 5. Propylene glycol, 1–2 pints per day orally, as a glucose precursor, is given with the ACTH treatment.⁴

A variety of vitamins, lipotrophs, amino acids, minerals, appetite stimulants and other nutrient and non-nutrient substances have been tried and recommended for the treatment of a fatty liver crisis, but their efficacy is not proven. Choline chloride has been recommended at a dose of 50–100 grams per day for three days to encourage removal of fat from the liver. Vitamin E and selenium may have value as antioxidants. Oral cobalt sulfate solution and/or B complex vitamins are commonly used to stimulate the appetite.⁴

Antimicrobial drugs are indicated to help prevent secondary infections during recovery, since the fatty liver cow is more susceptible to infectious disease. It might be prudent, however, to select those antimicrobial agents which are less dependent on liver activation or elimination.

Treating The Diseased Liver

As with so many diseases where drug therapy is unrewarding, there are numerous proposed treatments for bovine fatty liver disease. The severely affected liver has only limited ability to regain its normal function. When treating cows with suspected or confirmed liver disease, the practitioner must take care not to over-tax the liver with drugs.⁸

A shortage of highly unsaturated phospholipids has been shown experimentally to result in fatty liver disease. Choline is the cofactor required for formation of these essential phospholipids.⁶ Lipotropic agents such as choline and methionine may be used in an attempt to remove fat from liver cells, slow down fat deposition, or prevent further damage. The suggested dose for choline in adult dairy cows is 1–8 grams and methionine, 20–30 grams. There is no proof that the use of lipotropic agents is of any value when there is no proven deficiency of the given agent, except for choline, which has a definite role in the clinical management of fatty liver syndrome.⁹ ACTH appears to be helpful in the control of fatty liver syndrome.⁸ However, while endogenous or exogenous corticosteroids help reduce fat stores and increase gluconeogenesis and are beneficial to the fatty liver cow, they also tend to antagonize the effects of insulin, decrease cellular utilization of glucose, and increase protein catabolism.¹⁰ An inverse relationship has been shown to exist between plasma corticosteroid levels and milk production, but the effect on milk production of artificially increased endogenous or exogenous corticosteroid levels is not well defined.¹¹

Vitamin K is important in the management of animals with liver disease. The normal liver stores vitamin K, which may become significantly depleted in liver disease. B-complex vitamins are sometimes used in cases of fatty liver disease to supply cofactors of metabolism and to help stimulate the animal's appetite. Vitamin E and selenium are sometimes used, as their anti-oxidant effects help protect the liver.

A high-energy, low-protein ration is essential during the treatment of fatty liver disease. The cow may have to be force-fed. If she survives, she should have access to hay and/or pasture while she recuperates.⁶ Oral potassium supplementation may be needed if body stores of potassium have been depleted by the illness.

PREVENTION

Once the existence of a fat metabolism problem has been established in a herd, the best "cure" is the prevention of future cases. This can best be handled by modifying the way in which cows are fed, as well as what they are fed.

Division of the herd into feeding groups is very important. Bred heifers, dry cows, high producing cows, and late lactation cows should be fed in separate groups. In most cases, the separation will be physical, in the form of fences and pens, but in some dairies it may involve the use of newer feeding systems, such as those which are controlled by a signaling unit worn around the cow's neck.

The details of dairy cow nutrition are beyond the scope of this paper, but the following tips may be helpful.

Ideally, heifers should be bred at 15 months of age to calve at 24 months. If heifers remain open for too long, they tend to become fat. While bred heifers are still growing and need a good plane of nutrition, they should never be allowed to become fat. A ration containing about 10% crude protein and consisting mainly of forages, with little or no grain or silage, is sufficient for pregnant heifers.

A 60 day dry period is considered optimum for best efficiency and milk production. The dry period is the time for cows to replenish their body stores and to prepare themselves for the next lactation. Longer dry periods are inefficient and can result in cows gaining excess weight. If there are overweight cows in the herd, this is the best time to attempt to thin them down. Dry cows can be maintained on relatively low-energy, low-protein forages until the last few weeks of pregnancy, when they should be started on a concentrate mix with very low calcium levels to allow the rumen to adjust to grain feeding and to help prevent milk fever.

While it is true that lactating cows require large amounts of energy and protein for milk production, the concept of 'challenge feeding' is sometimes carried too far. On the other hand, this is definitely not the time for an overweight cow to be losing weight, since this could precipitate a fatty liver crisis. She should be maintained and handled carefully until she dries off, if possible. As an example, a 1500 pound Holstein cow producing about 90 pounds of milk per day with 3.5% butterfat would require 3.8 kilograms of total protein and 17 kilograms of total digestible nutrients daily.¹² This is quite a bit of nutrition to pack into the weight of feed that a cow is capable of consuming in one day. Some work with a pencil will prove that while it is difficult, but not impossible, to fatten a lactating cow, it can be difficult not to fatten a dry cow.

NECROPSY FINDINGS

The definitive gross lesion which is seen in cows with fatty liver syndrome is the greasy, yellow-tinted liver, which is enlarged and has rounded margins. Other organs which may be similarly infiltrated include the kidney and adrenal gland. The amount of functional tissue in the adrenal gland may be reduced. In addition, the cow will usually show lesions associated with some complicating problem such as mastitis, metritis, and muscle damage from being down for prolonged periods of time.

Microscopic lesions consist primarily of diffuse fatty infiltration of the liver, kidney, heart muscle, adrenal gland, and skeletal muscle fibers. Fat globules are seen within the epithelial cells of the liver and the renal tubules, and between the cells of the myocardium.

CONCLUSIONS

Bovine fatty liver syndrome is an economically significant problem in dairy cows, and is primarily the result of management systems that allow certain cows to become obese. The disease is difficult and expensive to treat, and the response to therapy is marginal. The dairy practitioner should be alert to the signs and conditions that suggest a fatty liver problem, and should be prepared to offer help and advice to the dairyman which will help him reduce the incidence of fatty liver in his herd.

Diagnostic procedures are now available and practical for use in detecting cows with fatty infiltration of the liver, and can be used as a guide to therapy for prevention of a crisis. The importance of having a sound understanding of dairy cow feeding and nutrition

cannot be over-emphasized. Prevention is the key to reduction or elimination of losses associated with fatty liver disease.

REFERENCES

1. Roberts CJ, Reid IM, Rowlands GJ, and Patterson A.: A fat mobilization syndrome in dairy cows in early lactation. *Vet. Record* 108:1, pp. 7-9. 1981.
2. Reid IM, Roberts CJ, and Manston R: Reduced fertility associated with fatty liver in high-yielding dairy cows. *Vet. Sci. Comm.*, No. 3, pp. 231-236. 1979.
3. Reid IM: Incidence and severity of fatty liver in dairy cows. *Vet Record* 107:12, pp. 281-84. 1980.
4. Bracken FK: Diseases affecting liver function, in Amstutz, HE (ed.) *Bovine Medicine and Surgery* 2nd ed. vol. 2. Santa Barbara, Ca., Am. Vet. Pub. Inc. 1980. Pp. 689-91.
5. Kronfeld DS: Homeostatic disorders associated with lactation, in Amstutz, HE (ed.) *Bovine Medicine and Surgery* 2nd ed. vol. 1. Santa Barbara, Ca., Am. Vet. Pub. Inc. 1980. Pp. 562-64.
6. Morrow DA: Fat cow syndrome, in Howard, JL (ed.) *Current Vet Therapy Food Animal Practice* Philadelphia, WB Saunders. 1981. Pp. 365-6.
7. Herdt TH, Goeders L, Leisman JS, Emery RS: Test for estimation of bovine hepatic lipid content. *JAVMA* 182:9 953-55. 1983.
8. Grunder HD: The advantages and disadvantages of circulatory and liver protection therapy of cattle. *Bov. Practitioner* 14, pp. 83-91.
9. Jenkins, WL: Drugs affecting gastrointestinal functions, in Booth NH and McDonald, LE (eds.) *Veterinary Pharmacology and Therapeutics* 5th ed. Ames, Iowa, ISU Press 1982. Pp. 604-6.
10. McDonald, LE: Hormones influencing metabolism, in Booth, NH and McDonald, LE (eds.) *Veterinary Pharmacology and Therapeutics* 5th ed. Ames, Iowa, ISU Press 1982. Pp. 553-4.
11. Breves G, Brendler H, Harmeyer J, and Farries E: Effect of feeding and production on the function of the adrenal cortex in milking cows. In Giesecke, Dirksen, and Stangassinger (eds.), *Metabolic disorders of Farm Animals, Proc. IVth Int'l Conf. on Production Disease in Farm Animals, Munchen, Germany, Oct. 1980.*
12. Jurgens, MH: *Animal Feeding and Nutrition* 4th ed. Dubuque, Ia., Kendall-Hunt Pub. Co. 1978. Pp. 293-305.

