Fat Cow Syndrome

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ABSTRACT

The objective of this paper is to characterize the fat cow syndrome. This condition refers to a combination of metabolic, digestive, infectious, and reproductive conditions which affects the obese periparturient cow. The condition develops primarily due to faulty feed management which permits excessive consumption of unbalanced diets. The syndrome is frequently a herd problem characterized by a high morbidity and mortality due to an increase in disease in periparturient cows. Clinical signs include depression, anorexia, ketonuria, marked decrease in production, progressive debilitation, weakness, nervous signs, and an elevation in temperature due to infectious disease. The obesity is generalized throughout the body with extensive fatty metamorphosis in the liver. Histological changes are primarily in the liver and kidney. Treatment of the condition consists of feeding a balanced diet, symptomatic treatment, and good supportive care. The condition can be prevented by feeding a balanced diet according to nutrient requirements of the National Research Council.

INTRODUCTION

Cattle on many farms are essentially fed high energy concentrates, and corn silage or hay crop forages free choice in early lactation. When concentrates are not restricted after peak lactation and conception and excessive corn silage or hay crop forages during the dry period, cows may become obese due to an excessive intake of a high energy diet. At parturition these fat cows appear to be more susceptible to metabolic conditions such as milk fever and ketosis, digestive disorders such as displaced abomasum, infectious diseases such as mastitis and salmonellosis, and reproductive conditions such as retained fetal membranes and metritis. These obese cows are more susceptible to the added stress of disease and frequently die despite treatment. This combination of disease conditions in the obese periparturient cow has been termed the fat cow syndrome. The objective of this paper is to characterize this imprecise syndrome and outline methods of treatment and prevention based on experiences in the Michigan State University Veterinary Clinic. There is a need for additional research on this syndrome.

OCCURRENCE

The fat cow syndrome occurs sporadically, depending on feed management practices. It is most frequent in loose housing where cattle in all stages of lactation including dry cows occasionally are fed and managed in one group. The time required for the condition to develop is variable, depending on the initial condition of the cows and the amount of high energy feed available for free choice consumption. It often occurs approximately 1 yr after cattle are moved from a stanchion barn into expanded loose housing facility where all cattle are maintained temporarily in one group until there are sufficient cows to fill the barn. Lead feeding at parturition and failure to restrict concentrate intake after peak lactation predisposes cows to obesity. Cows with long dry periods due to low production or infertility also are predisposed to becoming excessively fat. The fat cow syndrome sometimes is observed following a herd infertility problem.

This condition affects periparturient cows of all ages. It has been observed in primaparous heifers when they were fed high energy feeds free choice in the same group as the milking herd for a time prior to parturition. There is no known breed predisposition to the syndrome.

INCIDENCE

The fat cow syndrome results from a herd feeding and management problem. Since the cows are being fed and managed in a group, a
The majority of the dry cows are usually too fat and are predisposed to periparturient diseases. As a result, the morbidity may vary from 50 to 90% in the fresh cows while a majority of the affected frequently die after being treated for one or more periparturient diseases for 2 days to 2 wk. Six of 10 cows died of postpartum complications after consuming a liberal concentrate, high corn silage diet free choice for approximately 1 yr (9). Three cows were lost for other reasons, resulting on one survivor on this diet. A 25% mortality occurred after 120 parturitions during 4 mo in a 600 cow herd (10).

ETIOLOGY

The primary etiological agent is the excessive intake of an unbalanced diet frequently deficient in one or more nutrients according to requirements of the National Research Council (NRC). The exact role played by each of these factors in precipitating the fat cow syndrome needs to be documented. Feeding excessive quantities of concentrate during late lactation combined with free choice feeding of corn silage and/or hay crop forages during both late lactation and dry period are the usual causative factors. Lead concentrate feeding prior to parturition increased both milk fever and mastitis which are in the fat cow syndrome (6). The excessive consumption of an unbalanced diet during the late lactation and dry period appears to predispose periparturient cows to various metabolic, digestive, infectious, and reproductive conditions and frequently results in an unfavorable response to treatment.

PATHOGENESIS

The manifestations of fat cow syndrome appear to be due primarily to the extensive fatty metamorphosis of the liver. The development of a fatty liver is due to one or more of the following pathogenic mechanisms: increased hepatic lipogenesis, increased mobilization of free fatty acids (FFA) from adipose tissue, decreased hepatic oxidation of fatty acids, and an impaired triglyceride secretory mechanism (11). Choline facilitates the transportation of fatty acids from the liver to fat depots and enhances the oxidation of long chain fatty acids by the liver (2). The role that choline deficiency plays in the development of the fatty liver in the obese periparturient cow needs further evaluation.

CLINICAL SIGNS

The general signs include extreme obesity with a body weight of 680 to 820 kg for Holstein cows, decreased resistance to infection, and an increased number of periparturient diseases. Specific signs in the periparturient cow include depression, anorexia, ketonuria, marked decrease in milk production, progressive debilitation, weakness, and an elevated temperature due to an associated infectious disease. Occasionally these cows exhibit vague signs of a central nervous system disturbance which include tremors and extension of the head and neck with an odd stare. The response to treatment is frequently poor resulting in death or development of the downer cow syndrome.

These cows frequently have one or more of the following conditions: milk fever, ketosis, displaced abomasum, indigestion, retained fetal membranes, metritis, mastitis, or salmonellosis. Signs associated with one or more of these diseases are usually present. When recovery from the fat cow syndrome does occur, there is frequently a delay in the onset of postpartum estrus and conception due to retained fetal membranes and metritis, and a marked loss in weight in severely affected cows.

CLINICAL PATHOLOGY

The white blood cell count is typically below 3000 per mm$^3$ with a marked increase in band neutrophils relative to segmented cells in animals with the fat cow syndrome. The urea nitrogen of blood may be elevated about 20 mg/100 ml which is an indication of impaired renal function possibly due to a decrease in renal perfusion. Hemoglobin is frequently in the high normal range of 13.0 g per 100 ml due to a decreased fluid intake or an impaired ability of the digestive tract to absorb water.

Glucocorticoid is typically below the normal of 13.0 to 20.0 ng/ml in the parturient cow (12). Blood glucose is below 40 mg per 100 ml accompanied by ketonemia and ketonuria in many cows due to ketosis. However, glucose may be normal or above in cows with ketosis secondary to other diseases. In fact, glucose was reported to exceed 100 mg per 100 ml when the sample was collected at the time of parturi-
tion, during parturient paresis, or at other times when the cow was stressed severely (13).

The FFA's are elevated above the normal of 7 mg per 100 ml while triglyceride is depressed below 5.0 mg per 100 ml. The increase in FFA's is due to accelerated fat mobilization as a result of a negative energy balance (7). The FFA's increase when glucose decreases (1). The lowered triglyceride is possibly due to impaired fat mobilization and lipoprotein formation and release as a result of extensive fatty metamorphosis in the liver (11).

Two enzyme tests specific for liver function are ornithine carbamyl transferase (OCT) and sorbitol dehydrogenase (SDH). Results of these tests usually are elevated above 200 and 500 sigma units/ml for OCT and SDH, respectively. The bromosulphophalein (BSP) is the liver function test of choice. The results of this test in cows with fatty livers frequently exceed the normal half-time of 3 min by three to five times.

Blood electrolyte in fat cows is usually low normal to slightly below normal unless a condition such as milk fever is present to account for a more severe depression of serum calcium.

Necropsy

The obesity is generalized throughout the body except in cows which have been sick for 1 to 2 wk where external body condition may appear relatively normal due to the chronic emaciation. Cows which die both acutely and chronically have large deposits of internal fat located around the heart, kidney, mediastinum, pelvic canal, and in the omentum. The internal organ affected most severely is the liver which is enlarged and swollen with round edges and a pale color due to the intensive fatty metamorphosis. A portion of the liver placed in water will float due to the extensive fatty infiltration. Metritis and mastitis are common findings along with myorrhexis in downer cows which have been struggling to get up. Other pathologic findings are variable depending on associated diseases.

Primary histological changes are a fatty infiltration of the liver and kidney along with other changes associated with concurrent diseases. In the liver the hepatocytes around the central vein contain a large single fat vacuole while those at the periphery of the lobule contain smaller fat vacuoles. The epithelial cells of the kidney tubules also contain fat vacuoles. They are most prominent in those tubules at the corticomedullary junction and in the medulla.

Diagnosis

The diagnosis of the fat cow syndrome is based on a history of excessive energy intake. Obese cows and the presence of one or more periparturient conditions such as milk fever, ketosis, displaced abomasum, retained fetal membranes, and/or mastitis are diagnostic. Since a number of clinical signs may be in this syndrome, it is important to conduct a careful and complete physical examination in establishing a diagnosis. The diagnosis frequently may be confirmed by an unfavorable response to conventional treatments effective in treating the same conditions in cows in normal physical condition. The prognosis is guarded to poor.

Treatment

The obese cow does not respond as favorably to periparturient disease therapy as the cow in normal body condition. Death frequently results in several days or the downer cow syndrome may develop which responds poorly to treatment frequently resulting in death or euthanasia.

The general treatment is usually symptomatic for the periparturient conditions. For example, those cows with milk fever are treated with calcium preparations. Specific treatments include a minimum of 1000 sc of 50% dextrose daily by slow intravenous drip. The rationale for this therapy is to inhibit fat mobilization which will decrease the quantity of fatty acids presented to the liver. Choline chloride (50%) is administered orally at 50 g once daily. The dosage in the cow is based on the daily human dosage of 2 to 4 g (2). Choline is essential for phospholipid formation which is required for lipoprotein production and fat transport. Since methionine is a precursor of choline, it also should be beneficial in treating this condition (2). Broad spectrum antibiotics should be administered in therapeutic daily doses to control infection which is frequently in the form of mastitis and metritis.

The high energy diet should be replaced with
a diet balanced and fed according to NRC requirements for energy, protein, vitamins, and minerals. It should contain a source of coarse fiber such as hay. Pasture should be provided in season. If anorexia persists, rumen transplants should be initiated to restore normal protozoa and bacteria to the rumen. Rumen transplants and feeds high in readily digestible nutrients should be force fed in the amount of 11 to 21 liters twice daily until the appetite is restored.

Exercise also appears to be helpful since these cows are usually in confinement. Recent research suggests that there is benefit from forced exercise of dry cows (3).

There is a need for controlled research to document further benefits from this therapy and to evaluate the use of products such as methionine and ACTH in treating cows with the fat cow syndrome.

**PREVENTION**

The fat cow syndrome can be prevented by following recommended feed management. Cows in loose housing should be grouped by production to feed them according to NRC requirements since cows do not regulate intake according to their physiological requirements for milk production (5). Concentrate intake should be limited after peak lactation and conception while the intake of all high energy feed should be limited during the dry period. Metabolism trials show that the energetic efficiency of fat deposition during lactation is about 82% compared to 59% during the dry period (8). It is more efficient and economical to replace lipid reserves in late lactation than in the dry period. The energy intake can be limited during the dry period by feeding coarse grass hay or pasture which also will help prevent milk fever and displaced abomasum (4). If corn silage is fed, it should be limited to approximately 13.6 kg per head daily, depending on body condition. It is important that the dry cow receive a ration which is balanced for energy, protein, vitamins, and minerals to utilize nutrients to maximum efficiency and to prevent disease.

Good reproductive management to maintain a 12- to 13-mo calving interval also will help prevent cows from becoming too fat. Exercise during the dry period will help to improve muscle tone and reduce dystocia due to uterine inertia.

Challenge feeding rather than lead feeding of grain should be practiced prior to calving. This procedure consists of feeding approximately 2.3 to 4.5 kg of grain for 2 wk prior to calving to permit the rumen protozoa and bacteria to adapt to the change in diet. Larger amounts can be fed if justified by body condition. However, sudden changes in the diet at parturition should be avoided. High grain feeding prepartum depresses total dry matter intake at parturition (4). The grain should be increased at approximately .9 kg daily postpartum to challenge appetite until peak production is reached. Although not essential, it is helpful to feed 2.3 to 4.5 kg of hay daily during early lactation to increase dry matter intake and to help prevent digestive disturbances. The total ration should contain at least 40% roughage on a dry matter basis and a minimum of 15% crude fiber. High priority should be given to feeding a balanced ration to meet NRC nutrient requirements and to follow recommended management to prevent metabolic, digestive, infectious, and reproductive conditions which contribute to the fat cow syndrome in the obese periparturient cow.

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**REFERENCES**


