Treatment Of Feline Diabetes Mellitus Using An A-Glucosidase Inhibitor And A Low Carbohydrate Diet

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SUMMARY

The purpose of this study was to determine the effect of an α-glucosidase inhibitor (acarbose), combined with a low-carbohydrate diet on the treatment of naturally occurring diabetes mellitus in cats. Eighteen client-owned cats with naturally-occurring diabetes mellitus were entered into the study. Dual-energy x-ray absorptiometry (DEXA) was performed prior to and 4 months after feeding the diet to determine total body composition, including lean body mass (LBM) and percent body fat. Each cat was fed a commercially available low-carbohydrate canned feline diet and received 12.5 mg/cat acarbose orally every 12 hours with meals. All cats received subcutaneous insulin therapy except one cat in the study group that received glipizide (5 mg BID PO). Monthly serum glucose and fructosamine concentrations were obtained, and were used to adjust insulin doses based on individual cat's requirements. Patients were later classified as responders (insulin was discontinued, n=11) and non-responders (continued to require insulin or glipizide, n=7). Responders were initially obese (>28% body fat) and non-responders had significantly less body fat than responders (<28 % body fat). Serum fructosamine and glucose concentrations decreased significantly in both responder and non-responder groups over the course of four months of therapy. Better results were observed in responder cats, for which exogenous insulin therapy was discontinued, glycemic parameters improved, and body fat decreased. In non-responders, median insulin requirements decreased and glycemic parameters improved significantly, despite continued insulin dependence. The use a low-carbohydrate diet with acarbose was an effective means of decreasing exogenous insulin dependence and improving glycemic control in a series of client-owned cats with naturally-occurring diabetes mellitus.

RESULTS

In the acarbose/diet group, 11 cats were classified as responders, and 7 cats as non-responders. In the diet control group, 4 cats were classified as responders and 2 as non-responders. The total daily median insulin dose in non-responders decreased from 10 U/cat/day (5 units/dose) to 2U/cat/day (1 unit/dose) in both the diet and control groups.

The validated DEXA technique had a coefficient of variation for total body weight, LBM and percent body fat of 0.3%, 2.7%, and 3.8%, respectively. Overall, body weight was 5.7±1.2 kg for both groups at the start of the study and 5.9±1.2 kg after 4 months of therapy. No significant
difference in LBM (3.85±0.67 kg) was observed between responders (3.71±0.69 kg) and non-responders (4.08±0.62 kg) at the start of the study. However, LBM (0.26±0.32 kg) was significantly increased in both responders and non-responders in both study and control groups after four months of therapy (p=0.0006).

At the onset of the study, responders had a significantly (p < 0.0001) higher percent body fat (39±6.7%) compared with non-responders (19.6±17.2±6.85.1%). Following the four months of therapy, all responders showed an increase in LBM (271±393 g) and a decrease in body fat (3.51±3.7%). All non-responder cats also exhibited an increase in LBM (247±240 g) with therapy. However, the non-responders also showed an increase in body fat (56.3±0.3%). Although all responders showed a decrease in body fat and all non-responders showed an increase in body fat, the change was not statistically significant over the course of therapy.

The combination of a high dietary carbohydrate load, neutering, decreased exercise, and insulin resistance leads to the development of obesity in cats, and can contribute to the eventual development of type 2 diabetes mellitus (Nelson et al, 1990; Kirk et al, 1993; Biourge et al, 1997; Fettman et al, 1997; Link and Rand, 1998). A decrease in dietary carbohydrate load favors insulin secretion by the feline pancreas (Kettlehut et al, 1978; Kienzel, 1993; Kirk et al, 1993; Kitamura et al, 1999). This effect was best observed in obese patients who likely had a higher functional pancreatic reserve.

A frequent observation in this series of cases was a worsening of long-term glycemic control with owner non-compliance and addition of small amounts of high-carbohydrate food (mostly dry formulations). This further supports the contention that the benefits observed were associated primarily with a change to the low-carbohydrate diet and not solely by continued use of exogenous insulin in these patients. Since the responder group discontinued insulin therapy, exogenous insulin could not have been the cause of improved diabetic regulation in these cats and their improvement could only have been a result of the change in diet. Previous studies have shown that diabetic cats may respond (30% discontinued insulin and all cats showed a 50% decrease in insulin requirements) to low dietary carbohydrate therapy alone after treatment of glucose toxicity with exogenous insulin (Anderson et al, 2000).

The effect of a low-carbohydrate, protein-replete diet on body composition was particularly enlightening. The best response was seen in obese cats exhibiting a decrease in percentage of body fat and an increase in lean body mass. This effect might have been missed if body weight alone had been measured during the dietary period. However, it became apparent that increases in body weight were associated with an increase in LBM rather than an increase in adipose tissue. Our findings of improved glycemic control and loss of insulin dependence in obese diabetic cats that lost adipose tissue during consumption of a low-carbohydrate diet is also consistent with earlier findings in humans. Human Type 2 diabetics who gain lean body mass also experience improved glycemic control when consuming low-carbohydrate,
high-protein diets (Gougeon et al, 2000). Cats that gain weight experience a decrease in insulin responsiveness that is reclaimed upon subsequent loss of body fat (Fettman et al, 1998).

In summary, obese cats receiving a low carbohydrate canned diet showed a loss of insulin dependence and improvement in glycemic control after 4 months of therapy. Cats with low body fat also improved glycemic parameters but continued to require insulin at much lower dosage than prior to the dietary intervention. The addition of acarbose did not seem to affect the number cats that continued to require exogenous insulin or the improvement in glycemic control.