Retrospective study of the survival of cats with acquired chronic renal insufficiency offered different commercial diets

E. A. Plantinga, H. Everts, A. M. C. Kastelein, A. C. Beynen

A retrospective study was carried out on the efficacy of seven commercial diets designed to be fed to cats with chronic renal failure. The median survival time of 175 cats that received conventional diets was seven months, whereas the median survival time of 146 cats given one of the seven diets was 16 months. The cats on the most effective of the diets had a median survival time of 23 months and those on the least effective diet had a median survival time of 12 months. The composition of the seven diets was comparable, except that the most effective diet had a particularly high content of eicosapentaenoic acid.

CHRONIC renal failure is a common clinical condition in cats, and the prevalence of impaired kidney function increases with age (Burkholder 2000), reaching 30 per cent in cats over 15 years old (Krawiec and Gelberg 1989). The condition is characterised by an irreversible and progressive loss of kidney function, leading to azotaemia, uraemia and clinical signs associated with the kidneys' decreasing ability to perform their normal functions. Affected cats have a poor prognosis because the renal dysfunction generally progresses to its end stage (Polzin and Osborne 1995).

Nutritional management is essential in the treatment of chronic renal failure. In cats with induced renal failure, a restriction of phosphorus intake, as the only dietary variable, has been shown to reduce the extent of lesions observed in the kidneys postmortem (Ross and others 1982). It has been suggested that further dietary changes, such as reductions in the intakes of protein and sodium, increases in potassium, and a moderate base excess may contribute to reducing the progression of the condition (Plantinga and Beynen 2004). In dogs with experimental renal disease, the feeding of fish oil instead of either safflower oil or beef tallow diminished the rate of decrease in renal function (Brown and others 2000). It has also been suggested that the eicosapentaenoic acid (EPA) in fish oil alters eicosanoid metabolism and results in an increase in the production of vasodilatory mediators and a reduction in the production of proinflammatory eicosanoids, these changes being beneficial for renal function (Barchelli and Pollak 1985). Arachidonic acid is a competitive inhibitor of the conversion of EPA into eicosanoids (Siess and others 1980, Fischer and Weber 1983). It can be hypothesised that diets for the management of chronic renal failure in cats should be rich in EPA and satisfy their minimum requirement for arachidonic acid (Plantinga and Beynen 2004). Both of these fatty acids are essential for cats because cats lack the enzymatic activity to produce adequate amounts of them (Rivers and others 1975).

The use of specially designed diets to manage cats with chronic renal failure is now standard in veterinary practice. Elliott and others (2000) have shown that affected cats fed a special diet instead of a normal cat food survived longer, the median survival times being 20-8 and 8-7 months, respectively. On the market there is a wide variety of kidney diets that were prescribed.

From the reports selected, the following variables were extracted and classified: clinic code, breed, age, sex, plasma urea and creatinine concentrations at diagnosis, the use of a kidney diet (0 No, 1 Yes), the type of kidney diet (1 to 7), the use of an angiotensin-converting enzyme (ACE) inhibitor (0 No, 1 Yes) and the survival time after diagnosis (months).

The kidney diets were as follows: 1 Hill’s KD wet, 2 Hill’s KD dry, 3 Leo FKW, 4 Leo FKD, 5 Royal Canin renal programme dry, 6 Waltham low phosphorus/protein dry, 7 Waltham low phosphorus/protein dry. The cats were classified according to the positions of the kidney diets that were prescribed.

No record was kept of the cats' daily food intake. The use of the owner's choice not to feed a kidney diet. The diet had to be consumed for at least 75 per cent of the cat's survival time. No record was kept of the cats' daily food intake. The use of an ACE inhibitor was considered as a possible confounding factor.

Statistical analysis

The data were analysed by using the statistical package GENSTAT 5 (Payne and others 1993). The initial model contained all the variables and the survival time as the dependent variable; each variable was removed successively and then added again to the model, to identify variables that significantly affected the variance in survival time. The procedure...
was carried out either with all the kidney diets pooled (kidney diet 0 or 1) or with specified brands (kidney diet 0 or 1 to 7), and single and multiple regression analyses were carried out with the selected variables. Kaplan-Meier survival curves were constructed and the mean and median survival times were calculated.

RESULTS

After data processing, 321 reports were considered suitable. Table 3 shows the characteristics of the cats. Table 4 shows the types of diet offered in relation to the age and initial plasma urea and creatinine concentrations of the cats.

In the statistical model with the normal diet and all the kidney diets pooled, the variables clinic code, breed, age, sex, and the use of an ACE inhibitor did not significantly affect survival time, but diet, plasma creatinine and plasma urea did have a significant effect. The regression models were: survival time (months) = (22·00 – 0·454) x plasma urea (mmol/l) (R^2 adj=0·064, P<0·001); survival time (months) = (22·86 – 0·027) x plasma creatinine (µmol/l) (R^2 adj=0·102, P<0·001); and survival time (months) = (8·483 + 10·50) x (kidney diet = 1) (R^2 adj=0·229, P<0·001). The Kaplan-Meier survival curves for the last model are given in Fig 1. After combining the three significant predictors, the regression model with the highest predictive value was: survival time (months) = (15·47 + 9·51) x (kidney diet = 1 – 0·339) x urea (R^2 adj=0·283, P<0·001).

In further analyses, the brand of kidney diet was specified. Again, the variables clinic code, breed, age, sex and the use of an ACE inhibitor had no significant effect on survival time, but the variables plasma creatinine, plasma urea and the type of kidney diet did have a significant effect. Table 5 shows the estimated prolongation of the mean and median survival times by each kidney diet when compared with no dietary intervention. The incorporation of the variables plasma urea and creatinine into the model increased the percentage of variance accounted for, but did not affect the prolongation of survival time. The Kaplan-Meier survival curves for each type of kidney diet are shown in Fig 2.

DISCUSSION

For the interpretation of this retrospective study it is important to understand its limitations. First, its validity depends on the reports received from the practitioners. Different veterinarians may diagnose and treat chronic renal failure in cats differently, and it could be suggested that the observed effect of the diets was biased by the attitude and expertise of the practitioners. However, it is reassuring that in the statistical analysis the diets was on average nine months longer. These data agree with those of Elliott and others (2000); the validity of the study may thus be acceptable.

Table 5 and Fig 2 show that the type of kidney diet influenced the survival time. Diet 3 was the most effective and diet 6 was the least effective when compared with normal cat food. The restriction of phosphorus intake is the mainstay of the treatment of cats with chronic renal failure, and a phosphorus level of 0·1 to 0·2 g/MJ in wet diets and 0·13 to 0·27 g/MJ in dry diets may be considered ideal (Plantinga and Beynen 2004). Thus, diet 5 may be too rich in phosphorus (Table 1). It has been suggested that the ideal concentrations of sodium, potassium and arachidonic acid may be 0·04 to 0·06, 0·4 to 0·6 and 0·01 to 0·02 g/MJ, respectively (Plantinga and Beynen 2004). On this basis, all the diets contain too much sodium and too much arachidonic acid, but are optimal in potassium. On
the assumption that the optimal value for the base excess is 150 to 350 mmol/kg dry matter (Plantinga and Beynen 2004), the base excess of diets 1 and 5 may be considered appropriate. The ideal content of EPA may be 0.2 to 1.0 g/MJ (Plantinga and Beynen 2004), which would imply that only diet 3 has the ideal content. The authors suggest that the efficacy of diet 3 may be explained by its high concentration of EPA. Controlled research is necessary to investigate the role of EPA in the survival of cats with chronic renal failure.

The use of an ACE inhibitor had no significant effect on the survival time of the cats, whereas there is evidence that ACE inhibitors in cats have not been reported, but these drugs are frequently prescribed for cats with chronic renal failure. The results of this study provide further evidence that commercial kidney diets can prolong the survival time of cats with chronic renal failure. The data indicate that diet 3 prolonged the survival time most; it is relatively low in phosphorus and high in potassium and arachidonic acid and EPA. The superior performance of diet 3 may be due to the complex combined effects of its constituent parts, but interpreting the results together with earlier findings, it appears that the low level of phosphorus and the high levels of potassium and EPA, particularly the latter, may be its most important characteristics. This result is clearly important for assisting manufacturers in the formulation, and veterinarians in the selection, of suitable diets for treating cats with chronic renal failure.

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References


TABLE 4: Mean ages and plasma urea and creatinine concentrations of the cats offered a normal diet and the seven kidney diets

<table>
<thead>
<tr>
<th>Diet</th>
<th>Number of cats</th>
<th>Age (years)</th>
<th>Urea (mmol/l)</th>
<th>Creatinine (µmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>175</td>
<td>15.5±2</td>
<td>20.6±4</td>
<td>277.1±1*</td>
</tr>
<tr>
<td>1</td>
<td>5</td>
<td>16±2</td>
<td>19.2±2</td>
<td>313.2±2*</td>
</tr>
<tr>
<td>2</td>
<td>56</td>
<td>14±2b</td>
<td>16.5±4</td>
<td>242.1±1*</td>
</tr>
<tr>
<td>3</td>
<td>24</td>
<td>14±2b</td>
<td>17.3±3</td>
<td>269.5±4*</td>
</tr>
<tr>
<td>4</td>
<td>9</td>
<td>15±2b</td>
<td>18.5±3</td>
<td>253.7±4*</td>
</tr>
<tr>
<td>5</td>
<td>10</td>
<td>14±2b</td>
<td>16.7±3b</td>
<td>203.2±1*</td>
</tr>
<tr>
<td>6</td>
<td>35</td>
<td>14±2b</td>
<td>20±2b</td>
<td>278.6±4*</td>
</tr>
<tr>
<td>7</td>
<td>7</td>
<td>14±2b</td>
<td>15.4±3</td>
<td>276.3±4*</td>
</tr>
</tbody>
</table>

a, b Different superscripts within the column indicate significant differences between the diets (P<0.05)

TABLE 5: Mean (sd) and median survival times of the cats offered a normal diet and the seven kidney diets

<table>
<thead>
<tr>
<th>Diet</th>
<th>Survival time (months)</th>
<th>Mean (sd)</th>
<th>Median of cats</th>
<th>Number of cats</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>8.47 (5.49)</td>
<td>7.0</td>
<td>175</td>
<td>5</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>1</td>
<td>18±8 (9.61)</td>
<td>13.5</td>
<td>5</td>
<td>&lt;0.05</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>18.1 (10.6)</td>
<td>16.0</td>
<td>56</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>29.7 (10.6)</td>
<td>25.0</td>
<td>24</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>16±8 (6.22)</td>
<td>16.0</td>
<td>9</td>
<td>&lt;0.05</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>17±4 (8.98)</td>
<td>17.0</td>
<td>10</td>
<td>&lt;0.05</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>14±0 (10.0)</td>
<td>12.0</td>
<td>35</td>
<td>&lt;0.005</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>21±9 (8.49)</td>
<td>19.0</td>
<td>7</td>
<td>&lt;0.01</td>
<td></td>
</tr>
</tbody>
</table>

* Difference versus normal diet

FIG 2: Kaplan-Meier survival curves for the seven types of kidney diet (groups 1 to 7 refer to the diet codes)

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