Survival time, lifespan, and quality of life in dogs with idiopathic Fanconi syndrome

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Objective—To evaluate survival time of dogs with idiopathic Fanconi syndrome.

Design—Case series.

Animals—60 dogs with idiopathic Fanconi syndrome.

Procedure—Data were collected by means of questionnaires distributed to owners and veterinarians of dogs with idiopathic Fanconi syndrome and by examination of medical records when accessible. Questionnaires and records were reviewed for criteria used in diagnosis, treatments administered, survival time, and subjective owner perceptions regarding their dogs' general condition.

Results—58 of the dogs were Basenjis. Fifty-seven dogs (95%) were reportedly managed by use of a single therapeutic regimen. Median survival time after diagnosis of Fanconi syndrome was 5.25 years; median estimated lifespan was calculated to be between 11.3 and 12.1 years. Owners of 28 of 29 (97%) dogs still alive at the time of the study subjectively assessed their dogs' general condition as good to excellent. Seizures or other neurologic dysfunction was reported for 11 dogs.

Conclusions and Clinical Relevance—Results suggest that expected lifespan for dogs with idiopathic Fanconi syndrome was not substantially reduced, compared with expected lifespan for unaffected dogs, and that affected dogs generally had a good to excellent quality of life, as subjectively assessed by their owners. What effect the treatment regimen had on survival time or lifespan could not be determined, given the small number of dogs managed with other methods. The high percentage of dogs with neurologic abnormalities was a concern, but whether this was related to Fanconi syndrome or represented a breed-related predisposition to neurologic disease could not be determined. (*J Am Vet Med Assoc* 2004;225:377–383)

Fanconi syndrome is characterized by generalized impairment of reabsorptive function of the proximal portion of the renal tubule, resulting in excessive urinary losses of water, glucose, phosphate, sodium, potassium, bicarbonate, uric acid, amino acids, and protein. Idiopathic heritable forms of the syndrome occur in humans and Basenjis.[1-4] Sporadic occurrences of Fanconi syndrome have been documented in dogs of many other breeds and in a Simmental bull.[5-11] While up to 10% of Basenjis in the United States may be affected,[4] the pattern of inheritance has not been established. Many drugs and toxins have been associated with induction of Fanconi syndrome in humans, and drug- or toxin-associated Fanconi syndrome has been documented in dogs and a horse.[2,12-14] Fanconi syndrome has also been documented in association with multiple myeloma and other dysproteinemias and with a variety of inborn errors of metabolism in humans,[2,15,16] but, to our knowledge, similar associations in animals have not been demonstrated.

With 1 exception,[10] all reported cases of idiopathic Fanconi syndrome in dogs have involved adult animals. The most common clinical signs are polyuria and polydipsia. Other variably reported clinical signs include weight loss despite a normal appetite, weakness, urinary incontinence, and poor hair coat.[8,17-21] Metabolic bone disease leading to osteomalacia or rickets has been a commonly reported finding in affected humans; however, this is not a component of the disease as typically seen in dogs.[2,9,22] Substantial variation in the type and severity of reabsorptive defects has been documented among affected dogs, with some dogs able to maintain normal serum biochemical profiles while others develop abnormalities such as hyponatremia, hypokalemia, hypophosphatemia, and hypocalcemia, which may occur alone or in combination.[7-9,17,21-23] Glucosuria is found in almost all affected dogs, with blood glucose concentration typically within reference limits.[5,6,17,20-22] Aminoaciduria is a consistent finding, although the pattern and severity of amino acid loss may be variable.[22] Hyperchloremic metabolic acidosis is common,[1,9,22-27] as is isosthenuria or hyposthenuria. Most affected dogs appear to be able to concentrate their urine when deprived of water.[17,20,21]

Although the disease may be transient in dogs with Fanconi syndrome secondary to a resolvable initiating cause, in most affected dogs the disease is chronic and persistent.[12,25,26] The long-term prognosis for affected dogs, as described in the literature, appears guarded,[28] and many dogs have been reported to die or be euthanatized shortly after the diagnosis is made.[5,6,8,17,20,22] For instance, of 21 dogs described in previous reports[5,6,8,10,17,20-22,27] for which follow-up information was available, 12 (57%) survived < 5 months after the diagnosis was made. On the other hand, 7 (33%) of these dogs survived for > 1 year after the diagnosis was made. Follow-up beyond 1.5 years was available for only 1 of these dogs, which lived for 3.5 years after the diagnosis was made.[17] To the authors' knowledge, no studies of long-term survival times or the effects of treatment on survival time for dogs with Fanconi syndrome have been published.

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Because the nature of the underlying defect responsible for development of Fanconi syndrome in dogs is not well understood, treatment recommendations have centered on the use of supplements to minimize the systemic effects of chronic, large-scale losses of important solutes. Treatment recommendations in the human and veterinary medical literature have tended to focus on control of chronic acidosis and electrolyte abnormalities,[2,3,9,29] with the potential need for potassium and phosphate supplementation commonly addressed. For people with Fanconi syndrome, vitamin D supplementation is also often recommended because metabolic bone disease is a frequent component of Fanconi syndrome in people.[2,29-31] Use of a normal diet is typically recommended unless renal failure is present.[3,9] The use of bicarbonate or citrate to treat acidosis in patients with Fanconi syndrome is challenging, as very high doses may be required because of ongoing tubular bicarbonate losses.[2,29,32]

To the authors' knowledge, no long-term studies of survival times or quality of life in dogs with Fanconi syndrome have been published. The purposes of the study reported here were to determine survival times and lifespans of dogs with idiopathic Fanconi syndrome, assess whether early diagnosis had an effect on prognosis, and determine quality of life in affected dogs by assessing owner perceptions of their dogs' general condition.

Materials and Methods

Two methods were used to solicit participation in the study. First, notices explaining the study and soliciting owner participation were placed in various print and electronic media; these primarily consisted of publications and electronic chat groups devoted to the Basenji breed. Second, databases maintained by individuals with long-time involvement with the Basenji breed and Basenji health issues were searched to identify owners of affected dogs, and attempts were made to directly contact these owners.

Information on dogs included in the study was obtained through questionnaires distributed to owners and veterinarians of dogs with idiopathic Fanconi syndrome and by examination of medical records when available. All owners who participated filled out a short, written questionnaire and provided contact information for their veterinarians. In addition, some owners supplied medical records for their dogs. Attempts were made to contact veterinarians of all dogs about whom owner-supplied information had been gathered. Veterinarians who could be contacted were asked to provide information about the particular dog that had led to the initial contact and about any additional dogs with idiopathic Fanconi syndrome they had treated, if any.

Dogs for which neither a questionnaire completed by the attending veterinarian nor medical records were received were excluded from the study, as were dogs for which the documented criteria used in making the diagnosis were judged to be inadequate. In particular, dogs that did not have glucosuria with a normal or low blood glucose concentration, glucosuria with aminoaciduria, or glucosuria with persistent metabolic acidosis in the absence of any other probable underlying cause of acidosis were judged to have insufficient documented diagnostic criteria for inclusion. Dogs for which the diagnosis was reported to be unconfirmed and dogs in which abnormalities were reported to have been transient were also excluded.

Statistical analyses—The product-limit survival method was used to evaluate percentages of dogs with idiopathic Fanconi syndrome alive at various times after diagnosis of the disease and determine whether age at the time of disease diagnosis and identification of clinical signs of disease prior to diagnosis were associated with survival time. The log-rank test was used for significance testing. Because age at the time of disease diagnosis varied considerably and was found to be significantly associated with survival time, the life-table method was used to compute median estimated years of life remaining at 4.5 to 9.5 years of age (corresponding to the approx 95% confidence interval of age at the time of disease diagnosis for dogs included in the study). Dogs that were still alive at the end of the study were censored in this analysis, meaning that follow-up time accumulated to the last contact was credited, but a death event was not recorded. All analyses were performed with commercially available software.[a]

Results

As a result of solicitations placed in print and electronic media, 32 owners provided information on 39 dogs reported to have idiopathic Fanconi syndrome. Evaluation of databases maintained by individuals with longtime involvement with the Basenji breed and Basenji health issues yielded names of 80 owners of potentially affected dogs. Attempts were made to contact these 80 owners, 47 of whom provided information on 63 dogs. Veterinarians who were contacted supplied information on an additional 2 dogs. Thus, a total of 104 dogs were considered for inclusion in the study. Of these, 44 were excluded because they did not meet the necessary criteria. The remaining 60 dogs (representing 57 owners) were included in the study.

The 60 dogs included in the study consisted of 58 Basenjis, 1 Cocker Spaniel, and 1 Dachshund. Fifty-six were from the United States, 2 were from Canada, 1 was from Germany, and 1 was from Sweden. Of the 57 owners, 48 (84%) classified themselves as pet owners and 9 (16%) classified themselves as both breeders and pet owners. Of the 48 veterinarians who provided information on the number of other dogs with Fanconi syndrome they had treated, 25 (52%) reported that they had treated no other affected dogs, 15 (31%) reported having treated 1 to 3 other affected dogs, and 8 (17%) reported treating 5 to > 30 other affected dogs.

Twenty-nine (48%) dogs were dead at the time data collection was closed, and 31 (52%) were still living. There were 31 males (52%) and 29 females (48%). Fifty-three of the 58 (91%) dogs for which neutering status was indicated were reported to have been spayed or castrated. The remaining 5 dogs consisted of 3 sexually intact females and 2 sexually intact males. Age at the time of diagnosis of Fanconi syndrome was reported for 58 of the 60 dogs; median age was 5.7 years (range, 3.5 to 10 years), with 51 of the 58 (88%) being between 4 and 7 years old at the time of diagnosis (**Figure 1**).

Fifty-seven of the 60 (95%) dogs were reported to have been managed by use of a single therapeutic regimen known as the Gonto protocol (**Appendix**). Compliance with protocol recommendations was reported to be imperfect for 6 of these 57 dogs. Of the remaining 3 dogs, 1 was treated with potassium supplementation and fluids IV as needed, 1 was treated with bicarbonate and potassium supplementation on

the basis of regular laboratory assessments of blood gas values and serum potassium concentration, and 1 was not treated.

Information regarding whether the diagnosis was made before or after the onset of clinical signs was provided for 59 dogs. In 15 (25%) dogs, the diagnosis was made before the onset of clinical signs, whereas in the remaining 44 (75%), the diagnosis was made after the onset of clinical signs. The most common clinical signs at the time of diagnosis were polyuria and polydipsia.

Subjective owner assessments of their dogs' general condition were available for 29 dogs that were alive at the time of data collection. When owners were asked to rate their dogs' general condition on a scale from 1 to 5, with 5 being excellent and 1 being poor, 15 (52%) rated their dogs' condition as 5, 10 (34%) rated their dogs' condition as 4, 3 (10%) rated their dogs' condition as 3, and 1 (3%) rated their dogs' condition as 2. None of the dogs were reported by their owners to have a poor general condition.

Laboratory results consistently collected for all dogs in the study were limited to abnormalities that allowed the diagnosis of Fanconi syndrome to be made. More extensive records were available for 17 dogs, 16 of which had metabolic acidosis at least once, as determined on the basis of venous blood gas analyses. The 1 dog that consistently had venous blood gas results within reference limits had 2 years of medical records available for examination and was reported to be receiving 2,275 mg of sodium bicarbonate PO every 12 hours throughout this period. Dosage of sodium bicarbonate for the 16 dogs with documented metabolic acidosis ranged from 1,300 mg PO every 12 hours to 3,250 mg PO every 8 hours. For some of the dogs in this group, acidosis was occasionally severe, with blood pH as low as 6.97. In other dogs, blood gas values remained consistently within or very close to reference ranges, and any deviations were mild. Information on bicarbonate dosage was not specifically solicited; however, bicarbonate dosage for dogs for which dosages were reported ranged as high as 15,600 mg/d.

Of the 17 dogs for which medical records providing information beyond that required for the initial diagnosis of Fanconi syndrome were available, 8 developed persistent azotemia. In 1 dog, azotemia was present at the time of diagnosis. In 4 dogs, azotemia gradually worsened over a period of years. In 3 dogs, azotemia rapidly became worse and likely contributed to death or euthanasia of the dog, although 2 of these dogs also had other serious conditions. In 1 dog, insufficient information was available to determine progression of azotemia. Of the 4 dogs with slowly progressive azotemia, 3 were reported to have died or been euthanatized as a consequence of renal disease.

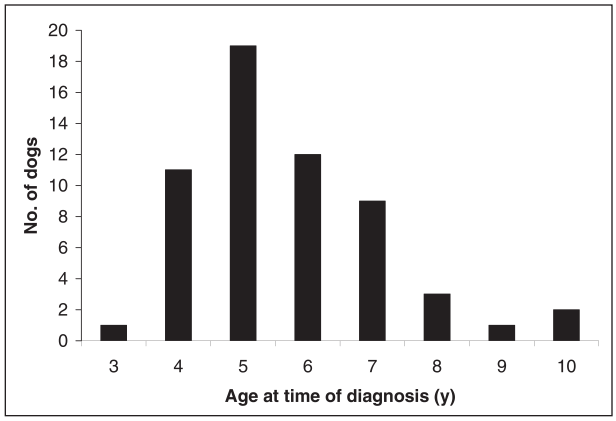

Figure 1—Age at the time of diagnosis for 58 dogs with idiopathic Fanconi syndrome.

The percentage of dogs still alive gradually decreased as time from diagnosis of Fanconi syndrome increased (**Figure 2**). Median survival time after diagnosis of Fanconi syndrome was 5.25 years (range, 7 days to 9.8 years). Age at the time of diagnosis was significantly ($P < 0.01$) associated with survival time. However, median survival time for dogs in which the diagnosis was made before the onset of clinical signs was not significantly ($P = 0.13$) different from median survival time for dogs in which the diagnosis was made after the onset of clinical signs. For dogs between 4.5 and 9.5 years old at the time of diagnosis, median estimated lifespan, calculated by adding age at the time of diagnosis to median calculated survival time after diagnosis, was 11.3 to 12.1 years (**Figure 3**).

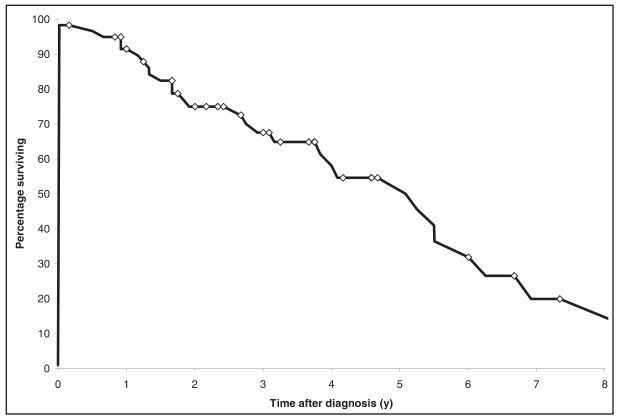

Figure 2—Survival curve (percentage of dogs surviving as a function of time after diagnosis) for dogs with idiopathic Fanconi syndrome. Diamonds represent censored observations.

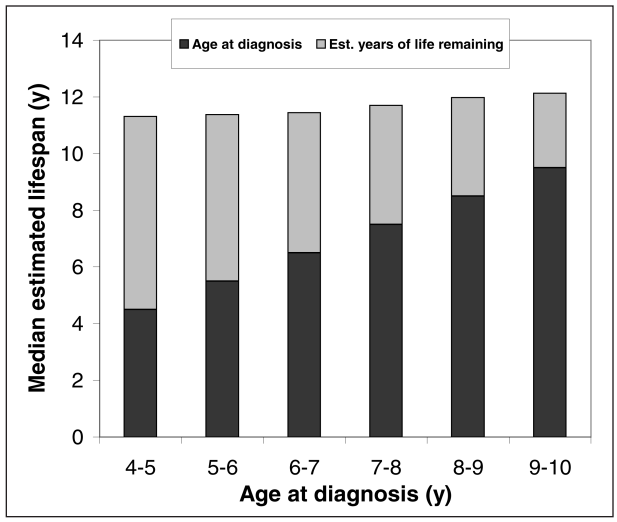

Figure 3—Median lifespan for dogs with idiopathic Fanconi syndrome, estimated as age at the time of diagnosis plus median calculated survival time after diagnosis.

Information on neurologic status of dogs with Fanconi syndrome was not solicited from owners or veterinarians; however, owners of 11 of the 60 (18%) dogs reported development of seizure activity or other neurologic dysfunction (eg, ataxia, dementia, or central blindness) several years after Fanconi syndrome was diagnosed. One dog died after developing severe neurologic abnormalities, and 7 dogs were reported to have been euthanatized because of the severity of their neurologic disease. These 8 dogs represented 13% of the 60 dogs included in the study and 28% of the 29 dogs that were dead at the time of the study. Two of these dogs were reported to have undergone extensive diagnostic testing, and in both, a diagnosis of granulomatous meningoencephalitis was made. Necropsy of the dog that died after developing severe neurologic abnormalities revealed leukoencephalomalacia and polioencephalomalacia, with findings localized to the neocortical regions of the brain.

Renal failure was the most commonly reported cause of death or euthanasia and was given as the sole or primary cause of death for 12 of the 29 (41%) dogs that were dead at the time of the study. Twelve dogs were reported to have been euthanatized for other reasons, including cardiovascular disease (n = 1), gastrointestinal tract disease (1), suspected hepatic failure (1), splenic tumor (1), trauma (1), granulomatous meningoencephalitis (2), and neurologic disease of uncertain cause (5); 4 of these dogs reportedly had renal failure, and renal failure may have been a contributing factor in the decisions to have the dogs euthanatized. Four additional dogs reportedly were euthanatized, but a specific precipitating cause other than clinical deterioration was not reported. The remaining dog that was dead at the time of the study was the dog that died after developing severe neurologic abnormalities.

Discussion

For dogs in the present study, age at the time of diagnosis of Fanconi syndrome was consistent with previous suggestions that this is a disease of adult onset, with most dogs being middle aged at the time of diagnosis. The fact that 51 of 58 (88%) dogs were between 4 and 7 years old at the time Fanconi syndrome was diagnosed suggests that veterinarians should maintain a high index of suspicion for Fanconi syndrome when examining Basenjis in this age range. None of the dogs in the present study were < 3.5 years old at the time Fanconi syndrome was diagnosed, but Fanconi syndrome has been identified in some Basenjis as young as 2 years old and in dogs of other breeds at 1 year of age or younger.[10,22]

Numbers of male and female dogs in the present study were nearly equal, suggesting that males and females are affected with equal frequency. A previous survey-based study[4] of the prevalence of Fanconi syndrome among Basenjis in the United States found affected females outnumbering males by a ratio of 3:1, but the authors interpreted this as an artifact of the high number of breeders who participated in the study. The fact that 53 of 58 (91%) dogs in the present study had been spayed or castrated was likely reflective of the fact that most pet dogs in the United States are neutered and not a reflection of a decreased risk among sexually intact dogs.

In 15 of 59 (25%) dogs in the present study, the diagnosis of Fanconi syndrome was made before the onset of clinical signs. In most of these instances, initial suspicion of Fanconi syndrome arose as a result of routine monthly screening of urine for glucose by owners using a standard urine dipstick test. Two dogs had glucosuria identified by means of a urinalysis conducted as part of a routine health screening by the dogs' veterinarians. In each case, the finding of glucosuria led to more extensive diagnostic testing and identification of Fanconi syndrome. Thus, although some dogs with Fanconi syndrome may not have glucosuria or may have glucosuria only intermittently, it does appear that in many dogs, the onset of detectable glucosuria precedes the onset of clinical signs, allowing for earlier recognition of incipient disease.[17,22,27] In the present study, median survival time for dogs in which the diagnosis was made before the onset of clinical signs was not significantly different from median survival time for dogs in which the diagnosis was made after the onset of clinical signs. This may have been an effect of the small sample sizes involved or may simply be reflective of the relatively normal lifespans of dogs included in the study.

Most dogs in the present study were managed by use of a single therapeutic regimen known as the Gonto protocol. This regimen attempts to use supplementation of vitamins, minerals, amino acids, and bicarbonate to replace renal solute losses and prevent or mitigate the development of chronic metabolic acidosis. The Gonto protocol is unusual in that it espouses a particularly aggressive approach to bicarbonate treatment, as well as routine supplementation of amino acids, vitamins, and minerals including vitamin D. No comparisons of the Gonto protocol with other treatment modalities have been conducted, and such a comparison was not possible in the present study, given the small number of dogs managed with other methods. A comparison of this sort would be necessary to fully assess any contribution made by this treatment protocol to survival times of dogs with Fanconi syndrome.

A review of laboratory findings for those dogs in the present study for which medical records were provided suggested that chronic metabolic acidosis and azotemia were common. However, no conclusions can ultimately be drawn from these data because information was not consistently collected for all dogs.

The median survival time for dogs in the present study suggests that the long-term prognosis for dogs with idiopathic Fanconi syndrome may be better than would be anticipated from a review of the veterinary literature. In a previous study,[22] for instance, 50% of affected dogs were dead within 90 days after the diagnosis was made. Similarly, 12 of 21 (57%) dogs

described in previous reports[5,6,8,10,17,20-22,27] survived < 5 months after the diagnosis was made. In contrast, several of the dogs in these reports survived for prolonged periods after the diagnosis was made, although long-term follow-up information was not available for most dogs. One possible reason for the long (5.25 years) median survival time in the present study could be that increased awareness of the disease during the past 25 years has resulted in a tendency for earlier diagnosis. In addition, the use of supplement-based medical treatment may have resulted in improved outcomes, either by physiologic effects of therapy or by encouraging greater owner commitment to long-term management of their dogs. It should be noted that owner tolerance of polyuria and polydipsia is likely to play an important role in the life expectancy of dogs with Fanconi syndrome, in that supplementation does not improve these clinical problems and may exacerbate them. It is possible that owners of the dogs in the present study may have had a higher degree of tolerance for polyuria and polydipsia than would have been true of a random sample of dog owners in general.

Several characteristics of the owners of dogs included in the present study may have played a role in the favorable long-term outcome. By responding to published solicitations for participation, owners who enrolled their dogs in the study demonstrated a high degree of self-selection and willingness for active involvement, which may have been a reflection of the degree of involvement they had with their dogs' medical care. In addition, the fact that owners of 51 of the 60 dogs were reportedly managing their dogs in full compliance with a sometimes labor-intensive treatment protocol further suggests that these owners were not likely to be representative of a random cross section of the dog-owning public. The high level of commitment of these owners to their dogs' medical care may have been associated with a greater likelihood to endure or work through difficulties that might not have been tolerated by owners with a lower degree of commitment. As a group, it is likely that owners of dogs in this study were more committed to and involved in their dogs' management and care than would be true of a random sample of dog owners. Because of this, it is possible that the lifespan results for dogs in the present study may represent something of a best-case scenario.

In the present study, dogs in which idiopathic Fanconi syndrome was diagnosed between the ages of 4.5 and 9.5 years had a median estimated lifespan ranging from 11.3 to 12.1 years. No references in the scientific literature could be found regarding median life expectancy of Basenjis, and references consulted vary considerably as to life expectancy of breeds of similar size.[33-37] Popular press estimates for Basenji longevity range from 10 to 15 years.[38-40] Given this, the estimated median lifespan for dogs with Fanconi syndrome of 11.3 to 12.1 years in the present study may represent a slight reduction in life expectancy, compared with life expectancy for unaffected dogs, but any reduction would appear to be modest.

Subjective owner assessments of their dogs' general condition in the present study were generally good to excellent, suggesting a high degree of owner satisfaction with their dogs' overall quality of life. Most owners who volunteered additional information on the subject indicated that aside from the often marked polyuria and polydipsia, their dogs were doing extremely well.

Information on neurologic status was not solicited from owners or veterinarians in the present study; however, seizures and other neurologic dysfunction were reported for 11 dogs, all of which were Basenjis. It is worth noting that some aspects of supplementation in and of themselves have been anecdotally associated with occasional mild neurologic dysfunction. For instance, dogs receiving high-dosage alkali supplementation twice daily were occasionally reported to show transient mild ataxia or dementia shortly after dosing; these signs resolved when the daily dose was further divided so that a smaller amount was given at each time. In many of the dogs with neurologic abnormalities, however, seizure activity or neurologic dysfunction was severe and progressive and therefore unlikely to be associated with bicarbonate administration. Neurologic disease was reported to be the cause of euthanasia for 7 dogs, and an eighth dog with neurologic abnormalities died. Granulomatous meningoencephalitis was diagnosed in 2 dogs, and a third had multifocal leukoencephalomalacia and polioencephalomalacia, but for the remaining dogs, the cause of the neurologic abnormalities was not determined. Whether granulomatous meningoencephalitis or CNS malacia is more common in Basenjis with Fanconi syndrome than in unaffected Basenjis is not clear and further study is needed. Most Basenjis in the United States are derived from a small number of dogs originally imported from Africa, and the possibility of heritable neurologic disease unrelated to Fanconi syndrome must be considered. Alternatively, the possibility that the reported neurologic disease might somehow be connected to Fanconi syndrome or be an unexpected complication related to the supplementation treatment should also be considered. Dogs with neurologic disease in the present sample were all reported to have been managed with the Gonto protocol; however, a dog with similar neurologic abnormalities that was not treated with the Gonto protocol has also been described.[17] Finally, given the small sample size in the present study, the possibility that the high percentage of dogs with neurologic abnormalities was merely a coincidence cannot be ruled out.

Although most dogs with Fanconi syndrome do not have azotemia at the time of diagnosis, development of renal failure is a clinically important concern. Renal failure is a common complication in affected humans, and development of chronic renal failure or fulminant acute renal failure has also been commonly described in affected dogs.[8,20,22,27,31,41,42] Although not all dogs with Fanconi syndrome develop renal failure, renal failure is a common finding and was reported in many dogs in the present study. In addition, renal failure was reported to be the sole or predominant cause of death or euthanasia in 12 of 29 (41%) dogs in the present study and may have been a contributing factor in an additional 4. Chronic proteinuria may be a contributing factor in the development of renal failure in dogs with Fanconi syndrome.[42] Thus, focused therapeutic attempts to try to reduce proteinuria in affected dogs may be worthwhile. On the

other hand, 17 of 29 dogs in the present study died or were euthanatized for reasons unrelated to renal disease, and some of the longest-lived dogs in the study did not ever develop renal failure.

In conclusion, expected median lifespan for dogs with idiopathic Fanconi syndrome in the present study was not substantially reduced, compared with the expected lifespan for unaffected dogs. In addition, affected dogs generally had a good to excellent quality of life, as subjectively assessed by their owners. While the extent to which these findings were a function of dietary or other supportive treatments is not clear, they stand in contrast with the poor long-term prognosis suggested by a review of the veterinary literature and indicate that a good to excellent long-term outcome may be possible for many affected dogs.

[a]SAS Institute Inc, Cary, NC.
[b]Pet Tab Plus, Pfizer Animal Health, Exton, Pa.
[c]Pet Cal, Pfizer Animal Health, Exton, Pa.
[d]Centrum, Wyeth, Madison, NJ.
[e]Amino Fuel, Twinlab, Hauppauge, NY.
[f]Tumil-K, King Animal Health, Bristol, Tenn.
[g]Urocit-K, Mission Pharmacal, San Antonio, Tex.

Appendix

Summary of the Gonto protocol for dogs with idiopathic Fanconi syndrome.[43]

1. Ensure that fresh water is freely available at all times.
2. Feed any high-quality, dry dog food; at least once a week, feed a high-protein, canned, mammal-based (eg, beef or lamb) dog food (for dogs with renal failure, feed a low-protein dry or canned diet).
3. Administer a vitamin-mineral supplement[b] for dogs.
   a. For dogs without clinical signs, administer 1/2 tablet, PO, every 12 hours.
   b. For dogs with clinical signs, administer 1 tablet, PO, every 12 hours (a higher dosage may be needed in dogs with hypokalemia or hypocalcemia).
4. Administer a calcium, vitamin D, and phosphorus supplement[c] for dogs (do not use in dogs with renal failure).
   a. For dogs without clinical signs, administer 1/2 tablet, PO, every 12 hours.
   b. For dogs with clinical signs, administer 1 tablet, PO, every 12 hours (a higher dosage is recommended for dogs with persistent loss of muscle mass and any signs of myalgia after correction of blood gas and serum biochemical abnormalities).
5. In dogs with polyuria and polydipsia, administer a multivitamin-mineral supplement[d] for humans.
   a. Administer 1 tablet, PO, every 7 days.
   b. For dogs with renal failure, administer 1 tablet, PO, every 48 hours.
6. Administer an amino acid supplement.[e]
   a. For dogs without clinical signs, administer 1 tablet (or equivalent amount of powder), PO, every 7 days.
   b. For dogs with severe muscle wasting, poor hair coat, or skin problems, increase dosage to as high as 1 tablet, PO, every 48 hours.
   c. For dogs with renal failure, do not exceed 1/2 tablet, PO, every 24 hours.
7. Administer intact sodium bicarbonate tablets on the basis of venous blood gas findings (venous blood pH and Pv_{CO_2}).
   a. The initial dosage should be determined on the basis of venous blood pH and Pv_{CO_2} (Table 1).
   b. The daily dosage should be divided and administered, PO, every 12 hours.
   c. Dosage modifications should be based on the results of follow-up venous blood gas analyses.
8. Administer potassium supplements[f,g] on the basis of serum potassium concentration in dogs with persistent hypokalemia (monitor serum potassium concentration weekly until concentration stabilizes at target concentration).
   a. For dogs with serum potassium concentration between 1.5 and 2.0 mEq/L, administer 15 mEq, PO, every 12 hours.
   b. For dogs with serum potassium concentration between 2.1 and 2.75 mEq/L, administer 10 mEq, PO, every 12 hours.
   c. For dogs with serum potassium concentration between 2.76 and 3.75 mEq/L, administer 5 mEq, PO, every 12 hours.

Initial treatment guidelines are for dogs weighing between 10 and 12.5 kg (22 and 27 lb). Dosages should be adjusted for dogs substantially larger or smaller than this. Follow-up serum biochemical testing and venous blood gas analyses should be performed 8 to 10 weeks after initiation of treatment, 6 months later, and annually thereafter if the dog's condition remains stable. In dogs with renal failure, follow-up serum biochemical testing, venous blood gas analyses, and physical examinations should be performed more frequently.

Table 1—Abbreviated table for calculating recommended initial daily dose of sodium bicarbonate in dogs with idiopathic Fanconi syndrome.

	Venous blood pH											
$Pvco_2$ (mm Hg)	7.40	7.35	7.30	7.25	7.20	7.10	7.00	6.90	6.80	6.70	6.60	6.50
20	9,072	9,072	10,368	10,368	11,664	14,256	15,552	16,848	18,144	18,144	19,440	20,736
30	7,776	7,776	9,072	9,072	10,368	12,960	14,256	15,552	16,848	16,848	18,144	19,440
32	6,480	6,480	7,776	7,776	9,072	11,664	12,960	14,256	14,256	15,552	16,848	18,144
34	6,480	6,480	6,480	6,480	7,776	9,072	10,368	11,664	12,960	14,256	15,552	16,848
36	5,184	5,184	6,480	6,480	7,776	9,072	10,368	11,664	11,664	12,960	14,256	15,552
38	5,184	5,184	5,184	5,184	6,480	7,776	9,072	10,368	10,368	11,664	12,960	14,256
40	3,888	3,888	3,888	3,888	5,184	6,480	7,776	9,072	9,072	10,368	11,664	12,960
42	2,592	2,592	2,592	2,592	3,888	5,184	6,480	7,776	7,776	9,072	10,368	11,664
44	1,296	1,296	1,296	1,296	2,592	3,888	5,184	6,480	6,480	7,776	9,072	10,368

Recommended daily dose of sodium bicarbonate is given in milligrams; the daily dose should be divided and given PO every 12 hours.

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