

## CASE REPORT

# Fanconi Syndrome in a Basenji

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### Abstract

Renal tubular dysfunction resembling canine Fanconi syndrome in a Basenji dog is described. The signs and laboratory results were similar to other reports of this disease. Therapy to correct some of the clinical signs and serum chemistry abnormalities was attempted.

**Key words:** Canine, renal tubular dysfunction, Fanconi syndrome, Basenji.

### Résumé

#### Rapport d'un cas du syndrome de Fanconi, chez un Basenji

Cet article décrit un cas de mauvais fonctionnement des tubules rénaux, semblable au syndrome de Fanconi canin, chez un Basenji. Les signes cliniques et les résultats des épreuves de laboratoire ressemblaient à ceux des autres cas de cette maladie, déjà rapportés. L'auteur tenta un traitement destiné à corriger certains des signes cliniques et des écarts biochimiques du sérum.

**Mots clés:** canin, mauvais fonctionnement des tubules rénaux, syndrome de Fanconi, Basenji.

### Introduction

A disease involving multiple defects of renal tubular reabsorption, resembling Fanconi syndrome in man (1), has been described in dogs (2,3,4,5,6,7). This disease is thought to have a familial incidence in the Basenji (2,3). Fanconi syndrome in dogs is characterized by excessive urinary loss of water, glucose, phosphate, sodium, potassium, amino acids, proteins and other solutes (6). Signs do not appear until adulthood and the course of the

disease may be several months or years (2,3). Urinary tract infection is not typical of this syndrome despite persistent glucosuria (7).

### Case History

On February 6, 1984 a six year old spayed female Basenji weighing 11.5 kg was presented with a history of polyuria/polydipsia of two to three weeks duration. This dog's previous medical history included urinary incontinence which was controlled with 1.0 mg stilboestrol per week and hypothyroidism which was controlled with levothyroxine at a dose of 0.3 mg SID. The dog was urinating large amounts more often than usual and was drinking in excess of one liter of water daily.

The primary abnormalities noted on

physical examination were a weight loss of 2 kg since the previous visit in October 1983 and a dry hair coat. A laboratory analysis of blood and urinalysis were completed (Table I). A water deprivation test was also performed. The dog's initial urine specific gravity was 1.006. A urine specific gravity of 1.028 and five percent weight loss both occurred 24 hours into the test.

Based on the history, physical examination and laboratory findings a tentative diagnosis of renal tubular dysfunction, possibly canine Fanconi syndrome, was made. Unfortunately an investigation could not be made into the family history as the dog had been adopted from the local SPCA.

Treatment was begun by supplementing the diet with some of the

TABLE I  
BASENJI WITH RENAL TUBULAR DYSFUNCTION

	Laboratory Results			
	Normal Values <sup>a</sup>	Feb. 6	Mar. 20	May 4
Glucose	3.6 - 7.2 mmol/L	4.3	3.5	5.9
BUN	3.5 - 10.7 mmol/L	5.7	5.0	7.1
Creatinine	88 - 176 µmol/L	88	133	150
Sodium	140 - 155 mmol/L	144	144	138
Potassium	4.0 - 5.7 mmol/L	3.7	4.0	3.6
Calcium	2.12 - 2.86 mmol/L	2.52	2.32	2.49
Phosphorus	0.97 - 2.26 mmol/L	0.94	0.97	1.23
Total protein	54 - 75 g/L	65	64	67
Albumin	23 - 40 g/L	33	33	32
Total bilirubin	0 - 17 µmol/L	3.4	1.7	1.7
SAP	12 - 84 IU/L	67	138	56
SGPT	9 - 63 IU/L	47	25	60
Urine S.G.		1.006	1.011	1.011
Urine pH <sup>b</sup>			6	5
Urine protein <sup>b</sup>		++	++	++
Urine glucose <sup>b</sup>		+++	+++	+++
Urine sediment		NAF	NAF	NAF

<sup>a</sup>Central Lab for Veterinarians, Surrey, B.C.

<sup>b</sup>Multistix, Ames.

Present address: Veterinary Services, Calgary Zoo, P.O. Box 3036, Station B, Calgary, Alberta T2M 4R8.

components which were lost due to impaired tubular reabsorption. This included 25 mg of potassium (as gluconate) per day and a vitamin/mineral supplement containing 77.3 mg phosphorus as calcium phosphate (Visorbites — Norden Laboratories, Lincoln, Nebraska 68501).

During the initial month of therapy the owners noted continued polyuria and polydipsia. The dog's appetite had decreased. After four weeks the owners noted increased volumes of urine. The dog was admitted for examination and further laboratory tests. The results are included in Table I (March 20, 1984). The patient had lost 1.5 kg during the previous five weeks but her attitude was still good. Abnormal laboratory findings included low urine specific gravity, glucosuria and proteinuria. Serum potassium and phosphorus were at the low range of normal.

In order to try to prevent continued weight loss the dog was placed on a diet of high caloric density (Eukanuba — Iams Co., Lewisburg, Ohio, 45338). Ten days later the dog was eating well, gaining weight and appeared to be urinating less.

On May 4, 1984 a third series of laboratory tests were run (Table I). Clinically the dog appeared to be improving and there was a noticeable weight gain. The laboratory findings at this time included hypokalemia and hyponatremia as well as dilute urine, glucosuria and proteinuria.

On June 11, 1984 there was no appreciable change in the dog's condition. The dog still had polyuria/polydipsia but otherwise was well.

### Discussion

The primary abnormalities noted on examination of this patient included a history of polyuria/polydipsia, weight loss and a poor hair coat. The differential diagnoses to be considered included liver disease, renal disease, diabetes mellitus, nephrogenic diabetes insipidus, hyperadrenocorticism and psychogenic polydipsia. The primary laboratory data abnormalities included hypophosphatemia, hypokalemia, glucosuria, mild proteinuria without blood and reduced urine specific gravity. These abnormalities are consistent with a diagnosis of a defect in renal tubular reabsorption,

specifically the canine Fanconi syndrome. The BUN and creatinine values were within normal limits so the renal abnormalities were confined to the reabsorption defects in the proximal tubule of the nephron. The proximal tubule is the site of active reabsorption of glucose, vitamins, sodium, potassium, calcium, phosphorus and bicarbonate (8). Low serum values of calcium were not noted and this may be due to the fact that this substance is actively absorbed in the thick ascending limb and distal tubule of the nephron as well as the proximal tubule. Glucose is also reabsorbed in the distal tubule in small amounts and this would suggest a distal tubule reabsorption defect specific for glucose as well as proximal tubule defects (3,8).

The water deprivation test helped to rule out nephrogenic diabetes insipidus as a cause of reduced urine specific gravity. The consistently low urine specific gravity was probably related to the consistent hypokalemia, as low serum potassium values have been associated with a loss of urine concentrating ability (9). Also the increased clearance and decreased reabsorption of solutes would lead to an osmotic diuresis and a lower urine specific gravity (8).

Metabolic acidosis can be a characteristic of canine Fanconi syndrome (6,7). This may be due to impaired bicarbonate reabsorption or decreased hydrogen ion secretion leading to renal tubular acidosis (3,6). The urine pH in this case was within normal units of pH 5.5-6.0<sup>6</sup> until the animal was placed on a high protein (30% by volume) diet. At this time the pH decreased to 5. This is consistent with the dietary changes and probably does not relate to any degree of metabolic acidosis. It is not recommended to use urinary pH as an indicator of patient systemic acid-base status due to many variables including diet and presence of infection which can affect the pH (6).

Very little has been written regarding therapy for the Fanconi syndrome in dogs. In humans most treatment is directed at osteoporosis which develops as the disease progresses. This has not been recognized as a problem in dogs (3,6). A total cure is not feasible but in this case replacement therapy was attempted to see if serum chemistry and urinalysis parameters could

be returned to normal levels and whether the dog would improve clinically.

Initial therapy was directed to the two major serum abnormalities of hypokalemia and hypophosphatemia. Recommended nutrient requirements for dogs include 198 mg phosphorus/kg body weight and 132 mg potassium/kg body weight (10). During the first month of treatment this dog was supplemented with 2.5% of phosphorus requirements and 7.0% of potassium requirements in addition to the regular dog food ration. These amounts were chosen primarily for convenience in administration of these supplements, i.e. one tablet of each supplement per day. There was mild response to this therapy with respect to serum phosphorus values.

A high caloric diet successfully reversed the weight loss which occurred and also increased serum glucose and phosphorus values to normal levels. There was, however, no remarkable effect on serum potassium. This is consistent with the findings of Easley in which potassium replacement had no effect on serum values (5).

It is known that hormones such as thyroxine play a role in influencing renal function and this dog was already being treated for hormone deficiencies at the time of diagnosis (11). However, since the dog was clinically regulated with respect to these other disorders it is doubtful that these hormones played a large role in the pathogenesis of the disease in this case.

From the results in this case it would seem to be possible to reverse some of the serum chemistry abnormalities and clinical signs related to canine Fanconi syndrome, namely hypophosphatemia and weight loss. Therapy had no effect on serum potassium levels, glucosuria, proteinuria, reduced specific gravity or the clinical signs of polyuria and polydipsia. It remains to be seen if long term therapy will continue to be effective in maintaining body weight and serum phosphorus values.

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## BOOK REVIEW

*Models of Anaerobic Infection*. Edited by M.J. Hill. Published by Martinus Nijhoff, Dordrecht, The Netherlands. 1984. 287 pages.

The book is a compilation of papers and abstracts of posters presented at the third Anaerobic Discussion Group (ADG) meeting held at Churchill College, University of Cambridge, England, followed by abstracts of the first meeting of the Society for Intestinal Microbial Ecology and Disease (SIMED) held at Boston, Massachusetts. Both meetings were held in 1983. The publication was designed to reflect the philosophy of ADG which is to bring together workers of various disciplines such as medical microbiologists, veterinarians, toxicologists and dental bacteriologists for wide-ranging discussions in informal surroundings. The reader will therefore find in this book a potpourri of topics on anaerobic infections which should appeal to the diverse membership of ADG.

The content is divided into three parts, a preface, a list of contributors, and an introduction to the ADG. The first part includes 19 full-length papers presented at the ADG meeting. The first seven papers describe studies employing animal models ranging from rats for evaluating antimicrobial prophylaxis and therapy in peritonitis to gnotobiotic pigs for demonstrating immune responses in swine dysentery. Four papers discuss aspects of bacterial virulence and adherence and an equal number deal with *in vitro* studies of host defense mechanisms such as polymorph function and the opsonic

activity of sera. Three papers discuss the benefits of applying continuous culture and fermentation systems to ecological themes, particularly with respect to the gut flora. One paper describes mathematical models as a means of explaining microbial growth in natural environment and in wound infections. A paper presenting results based on histological examinations and electron microscopy of ovine footrot provokes the reader to reassess the etiology of the infection. The study reports two previously unreported Gram negative bacteria that are always associated with *Fusobacterium necrophorum* in active lesions. These two organisms can form a peculiar inter-bacterial relationship where a central filamentous rod (mother) is covered with a coat of smaller rods (baby). This phenomenon is reminiscent of the "corn cob" formation observed recently between fusobacteria and streptococci.

The second part includes 21 abstracts of posters from the ADG meeting. Topics range from the use of cannulated pigs as a model system for evaluating steroid degradation in the gastrointestinal tract to the isolation of new anaerobes (e.g. *Anaerobiospirillum* spp.) from animals and man. The third part includes 18 abstracts of papers from the SIMED meeting. Most of these papers are of clinical orientation; a novel study used fiberoptic colonoscopy as an innovative probe to study colonic mucosal flora *in vivo*. A paper of veterinary interest deals with bacterial interference by native gut flora in limiting infections by enteric pathogens in chickens.

The book is well conceived but the content falls short of expectations in terms of new perspectives in clinical microbiology. Some of the full-length papers presented experimental data based on animal models that had been in use for sometime while others reviewed the results of studies already published elsewhere. For instance, in the paper on the adhesion of clostridia, at least a couple of the photographs shown (without proper credits or permission?) can be found in another book published four years before. As expected for this kind of publication, the format and style are quite varied. The book appears to be a direct reproduction of the original manuscripts resulting in minimal editing and photographs lacking in contrast. The use of symbols or arrows to indicate specific lesions or structures could have made certain photographs more comprehensible (e.g. pseudomembranous colitis, pp. 24 and 25). Also, the inclusion of general discussions or specific comments after each paper or series of papers dealing with related topics would have increased the impact of the contributions to current knowledge. Despite these deficiencies, the book is notable for the variety of experimental approaches employed by investigators of diverse backgrounds to elucidate the function of anaerobic bacteria in health and disease.

This book would be most useful as a reference material for laboratory workers and clinicians deeply involved in studies of anaerobic infections.

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