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Transient acquired Fanconi syndrome with unusual and rare aetiologies: A case study of two dogs

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Abstract: The acquired form of Fanconi syndrome is seldom identified in dogs; those cases that have been reported have been secondary to hepatic copper toxicosis, primary hypoparathyroidism, ingestion of chicken jerky treats, exposure to ethylene glycol, or gentamicin toxicity. However, to the best of our knowledge, there have been no reports of acquired Fanconi syndrome secondary to *Babesia* infection or ingestion of cosmetics in dogs. We here report on two dogs presented with a history of marked polyuria, polydipsia, and lethargy. Laboratory examinations showed glucosuria with normoglycaemia and severe urinary loss of amino acids. One dog was infected with *Babesia gibsoni* and the other dog had a history of cosmetics ingestion. The first dog received treatment for *Babesia* infection and the second dog received aggressive care to correct metabolic acidosis, electrolyte imbalances, and other add-on deficiencies. In both dogs, the Fanconi syndrome was successfully managed following the treatment for the underlying causes. In conclusion, both *Babesia* infection and cosmetics ingestion should be considered as a possible aetiology for transient acquired Fanconi syndrome in canine patients.

Keywords: Babesiosis; cosmetics ingestion; haematological profile; enzymes; urinalysis; ultrasonography

Fanconi syndrome is an abnormality of reabsorption in the proximal renal tubule resulting in the excessive urinary loss of water, glucose, phosphate, sodium, potassium, bicarbonate, uric acid, amino acids, and protein (Yearley et al. 2004). Fanconi syndrome is classified into two types: complete and incomplete. The complete type is characterised by the loss of all solutes including bicarbonate, glucose, amino acids, proteins, and phosphate (Watanabe et al. 2005). Leakage of a subset of these solutes was described as incomplete type Fanconi syndrome (Watanabe et al. 2005). In human medicine, Fanconi syndrome was first described in 1924 by Lignac and associated with genetic conditions including Wilson's disease, hereditary fructose intolerance, galactosemia, cystinosis, mitochondrial cytopathy, tyrosinemia, Dent's disease, Lowe syndrome, and multiple myeloma (Izzedine et al. 2003; Yearley et al. 2004; Hall et al. 2014). Acquired Fanconi syndrome in humans was associated with

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drug toxicity due to anti-cancer agents, anti-viral agents, and antibiotics (Izzedine et al. 2003; Hall et al. 2014). Idiopathic inherited forms of Fanconi syndrome in dogs have been well documented in Basenjis, affecting up to 10% of the Basenjis in the United States; the condition has been recently reported in Irish Wolfhound siblings in 2018 (Yearley et al. 2004; Bommer et al. 2018). The acquired form of the disease is seldom identified in dogs and these cases have mostly been secondary to hepatic copper toxicosis, primary hypoparathyroidism, ingestion of chicken jerky treats, exposure to ethylene glycol, or gentamicin toxicity (Freeman et al. 1994; Appleman et al. 2008; Thompson et al. 2013; Hooijberg et al. 2015; Bommer et al. 2018). While recovery of the proximal tubular defect has not been reported in canine inherited Fanconi syndrome except in one case (Jamieson and Chandler 2001), secondary Fanconi syndrome patients, in contrast, usually recover following symptomatic treatment and treatment of the underlying cause. To the best of author's knowledge, this is first report of canine transient acquired Fanconi syndrome associated with *Babesia* infection and cosmetics ingestion that successfully recovered following the treatment of the underlying cause.

Case description

A 7-year-old, castrated, male Bichon Frise (Dog 1) and a 5-year-old, spayed, female mixed breed (Dog 2) were presented to the Konkuk University Veterinary Medical Teaching Hospital.

Dog 1 was diagnosed with *Babesia* infection and treated at a local veterinary hospital, but was referred due to recurrence of infection. History taking and physical examination revealed clinical features of lethargy, polyuria, polydipsia, and hyperthermia (39.7 °C). Haematological profile testing of Dog 1 revealed a decreased haematocrit (32.5%, reference range 37.3–61.7%), severe thrombocytopenia (20 × 10^9/l, reference range 148–484 × 10^9/l), and hypophosphatemia (0.65 mmol/l, reference range 0.8–2.2 mmol/l). It also revealed altered serum enzyme activities, i.e. elevated serum alanine aminotransferase (3.8 µkat/l, reference range 0.17–1.67 µkat/l), and serum gamma glutamyltransferase (0.25 µkat/l, reference range 0–0.18 µkat/l). All other indicators like glucose (5.8 mmol/l, reference range 4.1–7.9 mmol/l), creatinine (97 µmol/l, reference range 44–159 µmol/l), and blood urine nitrogen (5 mmol/l, reference range 2.5–9.6 mmol/l) were within the normal range (Table 1).

Abdominal ultrasonography showed bilaterally increased echogenicity of the renal cortex and medulla, and mild dilation of the left renal pelvis (Figure 1A). The ureters and urinary bladder showed no pathologic changes. Systolic blood pressure was normal at 140 mmHg. Venous blood gas results showed metabolic acidosis with respiratory compensation (pH = 7.38, reference range 7.31–7.42; [HCO_3] = 15.9 mmol/l, ref-

Table 1. Serial haematology and biochemistry examination of Dog 1 (associated *Babesia* infection) following treatment

	Dog 1			
Bloodindicator	week 0	week 5**	week 8	reference
RBC (× 10^{12}/l)	4.18*	6.8	6.5	5.5–8.5
PCV (%)	32.5*	50.4	47.6	37.3–61.7
Hb (g/l)	106*	182	161	120–180
MCV (fl)	77.8*	74.1	73.2	60–74
MCHC (%)	32.6	36.1	33.8	31–36
WBC (× 10^9/l)	7.5	12.21	11.34	5.05–16.76
Platetes (× 10^9/l)	20*	450	359	148–484
Total protein (g/l)	66	67	67	52–82
Albumin (g/l)	33	39	35	22–39
Globulin (g/l)	33	28	32	25–45
Glucose (mmol/l)	5.8	7	6.5	4.1–7.9
Creatinine (µmol/l)	97	70.7	79.6	44–159
Urea (mmol/l)	5	4.6	7.1	2.5–9.6
Inorganic phosphate (mmol/l)	0.65*	1.65	ND	0.8–2.2
Total calcium (mmol/l)	2.4	2.4	ND	1.98–3
ALT (µkat/l)	3.8*	3.85*	1.5	0.17–1.67
GGT (µkat/l)	0.25*	0.23*	0.01	0–0.18
Sodium (mmol/l)	158	152	158	144–160
Chloride (mmol/l)	117	114	117	109–122
Potassium (mmol/l)	3.9	3.9	4.3	3.5–5.8

ALT = alanine aminotransferase; GGT = gamma glutamyl transferase; Hb = hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; ND = not determined; PCV = pack cell volume; RBC = red blood cells; WBC = white blood cells

*Value outside reference range. **_Babesia_ PCR (polymerase chain reaction) test for Dog 1 was negative

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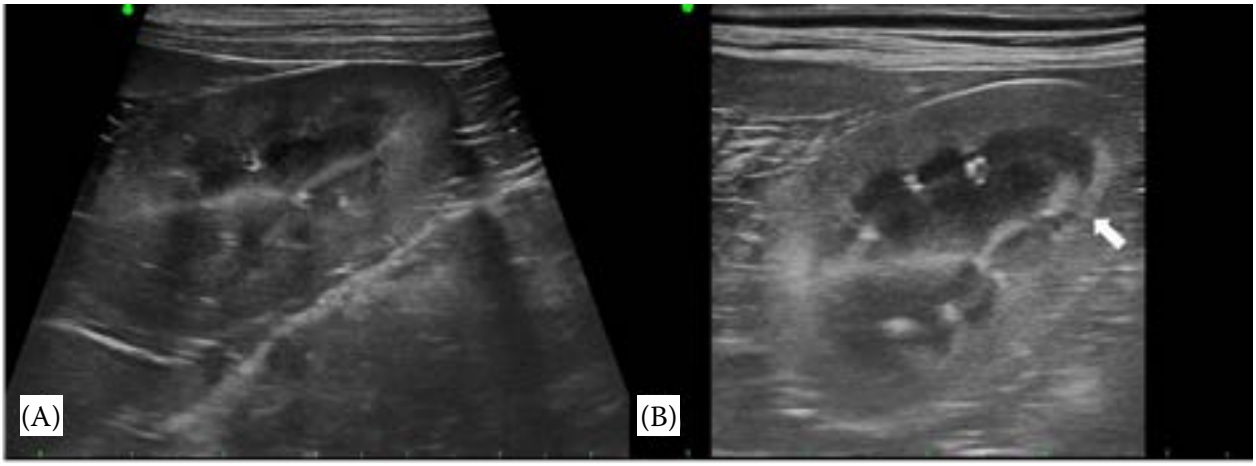

Figure 1. Renal ultrasonographic images. Sagittal view of left kidney of Dog 1 (A) and Dog 2 (B). Both dogs show increased echogenicity of bilateral cortico-medullary junction and dilated renal pelvis of left kidney. Note the hyperechoic medullary rim sign (arrow) in Dog 2's kidney

erence range 20–29 mmol/l; pCO_2 = 30 mmHg, reference range 32–49 mmHg). The urine collected by cystocentesis was dark yellow and clear. Amorphous crystals were detected on microscopic evaluation of the sediment and no pathogens were observed on cytology of the urine. Urinalysis (IDEXXVetLab UA analyser™; IDEXX, Seoul, Republic of Korea) was positive for glucosuria (2+), negative for ketonuria, pH 7.0, and showed a urine specific gravity of 1.074. Urine protein/creatinine ratio was within the normal range (0.15). Urine bacterial culture showed no growth.

Considering the above results, Dog 1 was suspected to have incomplete Fanconi syndrome. Quantitative analysis of urinary amino acids (SML, Seoul, Republic of Korea) revealed severe generalised aminoaciduria (Table 2) confirming the diagnosis of Fanconi syndrome. To rule out other

Table 2. Urinary amino acid analysis in Dog 1 (associated *Babesia* infection) following treatment

	Urinary amino acid concentration (μmol/mmol urinary creatinine)					
Amino acids	1 week	3 weeks	5 weeks	10 weeks	24 weeks	control
Aspartic acid	1	1	1	0	1	1
Threonine	872	601	792	219	121	15
Serine	612	371	432	110	52	43
Glutamic acid	11	16	12	6	4	< 10
Glutamine	1 621	1 617	1 616	309	185	31
Glycine	2 097	957	1 675	507	143	54
Alanine	1 845	442	959	81	66	16
Valine	420	39	104	11	4	< 10
Cystine	23	47	43	10	12	< 10
Methionine	186	18	57	6	2	< 10
Isoleucine	161	13	37	4	1	< 10
Leucine	225	18	68	12	2	< 10
Tyrosine	226	57	107	16	7	< 10
Phenylalanine	112	21	46	9	3	26
Histidine	461	208	229	109	27	< 10
Lysine	81	24	65	24	24	58
Arginine	23	29	28	7	15	< 10

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Table 3. Serial urinalysis of two dogs with Fanconi syndrome

	Dog 1				Dog 2				
	week 0	week 3	week 5*	week 8	week 0	week 2	week 3	week 16	reference
pH	7	5	6	6	5	7	7	8	6.0–7.5
Glucose	2+	3+	2+	negative	4+	3+	1+	negative	negative
Ketone	negative	1+	negative	negative	2+	negative	negative	negative	negative
UPC	0.15	0.46	0.27	< 0	2.64	0.08	< 0	< 0	< 0

**Babesia* PCR (polymerase chain reaction) test for Dog 1 was negative
UPC = urine protein to creatinine ratio

Table 4. Serial haematology and biochemistry examination of Dog 2 (associated cosmetics ingestion) following treatment

	Dog 2			
Blood indicator	week 0	week 1	week 16	reference
RBC (× 10^{12}/l)	7.82	6.51	7.42	5.5–8.5
PCV (%)	51.4	42.7	48.1	37.3–61.7
Hb (g/l)	172	135	169	120–180
MCV (fl)	65.7	65.1	64.8	60–74
MCHC (%)	33.5	33.7	35.1	31–36
WBC (× 10^9/l)	19.9*	27.63*	9.56	5.05–16.76
Platetes (× 10^9/l)	386	316	422	148–484
Total protein (g/l)	63	64	68	52–82
Albumin (g/l)	33	29	33	22–39
Globulin (g/l)	30	35	35	25–45
Glucose (mmol/l)	4.3	5.6	5.8	4.1–7.9
Creatinine (μmol/l)	61.9	44.2	71	44–159
Urea (mmol/l)	2.15*	1.07*	4.6	2.5–9.6
Inorganic phosphate (mmol/l)	0.65*	0.94	1.2	0.8–2.2
Total calcium (mmol/l)	2.3	ND	2.7	1.98–3
ALT (μkat/l)	2*	2.238*	0.77	0.17–1.67
GGT (μkat/l)	0	ND	0.01	0–0.18
Sodium (mmol/l)	159	148	152	144–160
Chloride (mmol/l)	117	115	112	109–122
Potassium (mmol/l)	2.7*	3.7	4.1	3.5–5.8

*Value outside reference range
ALT = alanine aminotransferase; GGT = gamma glutamyl transferase; Hb = haemoglobin; MCHC = mean corpuscular haemoglobin concentration; MCV = mean corpuscular volume; ND = not determined; PCV = pack cell volume; RBC = red blood cells; WBC = white blood cells

differential diagnoses, serological tests for leptospirosis were performed and were negative. History taking revealed that the dog has been fed only commercial canine dry food and has never taken any other food or treats like Chinese chicken jerky. The dog was initially treated with atovaquone (Malaron®; GlaxoSmithKline Inc., Ontario, Canada), 13.3 mg, *per os* (*p.o.*), q8 h, to treat the *Babesia* infection, and azithromycin, 10 mg, *p.o.*, q24 h, to control inflammation. After five weeks of treatment, once polymerase chain reaction (PCR) test revealed that the dog was negative for *Babesia*, atovaquone and azithromycin were tapered off and completely stopped on day 100 and day 91, respectively. Glucosuria resolved on the 20th day after the last negative result of PCR test (Table 3); aminoaciduria also gradually decreased (Table 2).

Dog 2 was referred with complaints of polyuria, polydipsia, lethargy, decreased appetite, and vomiting for two weeks. Interestingly, the patient had a history of having eaten several cosmetics during the absence of the owner (possibly as a result of separation anxiety). Laboratory tests revealed leukocytosis (19.9 × 10^9/l, reference range 5.05–16.76 × 10^9/l), hypophosphatemia (0.65 mmol/l), decreased blood urea nitrogen (2.15 mmol/l), hypokalemia (2.7 mmol/l, reference range 3.5–5.8 mmol/l), and normal range of glucose (4.3 mmol/l). Serial blood examination was performed in company with the treatment (Table 4).

Abdominal ultrasonography showed bilateral renal changes, mild left renal pelvis dilation, and medullary rim sign (Figure 1B). Venous blood gas analysis showed metabolic acidosis (pH = 7.28 and [HCO_3] = 15.8 mmol/l). Urine collected by cystocentesis was bright yellow and clear. Some granular crystals were detected on microscopic evaluation of the sediment and many epitheli-

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al cells were observed on cytology of the urine. Urinalysis revealed glycosuria (3+), ketonuria (2+), pH 5.0, and urine specific gravity of 1.020. Severe proteinuria was detected (urinary protein/creatinine ratio = 2.64) and urine metabolic screen test showed severe amino acid loss into the urine (SML, Seoul, Republic of Korea) (Table 5). Bacterial culture of the urine revealed no growth. To rule out other differential diagnoses, serological tests for leptospirosis and serum parathyroid hormone (PTH) concentration test were performed and revealed to be negative and normal, respectively.

According to these laboratory abnormalities, Dog 2 was diagnosed with complete Fanconi syndrome. Treatment was focused on normalisation of leukocytosis, metabolic acidosis, hypophosphatemia, hypokalemia, and improvement of clinical signs during hospitalisation. To minimize inflammation and clinical manifestations, the dog was treated with antibiotics (cephalexin, 30 mg/kg, *p.o.*, q12 h and enrofloxacin, 5 mg/kg, *p.o.*, q12 h), antiemetics (maropitant citrate, 1 mg/kg, *s.c.*), and a gastrointestinal protectant (famotidine, 0.5 mg/kg, *p.o.*, q12 h). To correct the hypokalemia and metabolic acidosis, the dog was administered with sodium bicarbonate (0.5 mEq/kg, intravenous, *i.v.*), potassium citrate (0.5 mEq/kg, *p.o.*, q12 h), phosphorus (0.03 mmol/kg body weight/h, *i.v.*, constant rate infusion), and fluid therapy (Hartmann solution, 5 ml/kg body weight/h, *i.v.*). The treatment was modified based on follow-up serum chemistry and blood gas analysis. Hypokalemia and hypophosphatemia normalised 7 days after initiating treatment (Table 4); glucosuria and proteinuria also gradually decreased with treatment (Table 3). Finally, the clinical symptoms improved, and both patients remained well after discharge until now (i.e. 8 months and 2 years for Dog 1 and Dog 2, respectively).

Table 5. Urinary amino acid analysis in Dog 2 (associated cosmetics ingestion) following treatment

	Urinary amino acid concentration (μmol/mmol urinary creatinine)			
Amino acids	1 week	3 weeks	18 weeks	control
Aspartic acid	3	5	0	1
Threonine	1 169	1 759	235	15
Serine	1 461	1 349	54	43
Glutamic acid	70	35	6	< 10
Glutamine	4 354	7 428	427	31
Glycine	6 565	6 618	57	54
Alanine	1 900	3 850	66	16
Valine	1 498	510	3	< 10
Cystine	145	104	20	< 10
Methionine	389	133	4	< 10
Isoleucine	758	167	2	< 10
Leucine	1 076	307	1	< 10
Tyrosine	385	378	6	< 10
Phenylalanine	514	257	2	26
Histidine	1 018	808	14	< 10
Lysine	1 001	510	15	58
Arginine	465	295	18	< 10

DISCUSSION AND CONCLUSIONS

Fanconi syndrome is a dysfunction of the proximal renal tubule, most commonly due to a genetic aetiology (homozygous mutation in FAN1 gene); among canines, it commonly affects Basenji dogs and usually presents at the age of 3–8 years (Hooijberg et al. 2015; Bommer et al. 2018). Recently, idiopathic hereditary Fanconi syndrome has been reported in a juvenile Irish Wolfhound (Bommer et al. 2018). After being diagnosed with idiopathic hereditary Fanconi syndrome, the average survival time for a dog is 5.25 years (Yearley et al. 2004). On the other hand, acquired Fanconi syndrome is mostly transient and resolves once the primary cause is removed and supportive care is given to correct metabolic acidosis, electrolyte imbalances, and other add-on deficiencies (Thompson et al. 2013; Hooijberg et al. 2015).

Numerous causes have been reported for transient acquired Fanconi syndrome including hypoparathyroidism, Chinese chicken jerky, gentamicin, copper storage hepatopathy, and ethylene glycol toxicosis (Freeman et al. 1994; Jamieson and Chandler 2001; Appleman et al. 2008; Hooijberg et al. 2015). For Dog 2, we ruled out hypoparathyroidism by serum PTH concentration test and there was no history of chicken jerky intake. Dog 1 also did not have a history of chicken jerky intake. Although the exact test for hypoparathyroidism was not performed for Dog 1, the disease was excluded as serum calcium was within normal range. As the incomplete Fanconi syndrome in Dog 1 was secondary to a *Babesia* infection, the clinical features of the syndrome abated following intensive treatment and cure of the infection. Dog 2 acquired complete Fanconi syndrome follow-

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ing cosmetics ingestion; the patient had metabolic acidosis, electrolyte imbalance, proteinuria, and especially severe hypophosphatemia which could cause osteomalacia (Hall et al. 2014). Therefore, the dog received aggressive care to correct the electrolyte imbalance; the syndrome was consequently cured after one month of supportive treatment.

Normoglycaemia with glucosuria in addition to severe aminoaciduria with electrolyte imbalances are typical diagnostic indicators of Fanconi syndrome (Hooijberg et al. 2015). Clinical manifestations of Fanconi syndrome include nonspecific signs such as polyuria, polydipsia, weight loss, weakness, and poor quality of hair coat (Yearley et al. 2004; Thompson et al. 2013). Severe phosphate loss is the chief clinical feature of Fanconi syndrome, as it gives rise to osteomalacia, which presents with bone fractures and muscle weakness (Hall et al. 2014). Ultrasound examination reveals non-specific renal injuries in Fanconi syndrome (Hall et al. 2014). In Dog 1, ultrasound features of mild chronic kidney disease were detected including increased cortico-medullary junction echogenicity, there were no other significant findings. In addition to signs of chronic kidney disease, bilateral renal medullary rim sign was observed during Dog 2's ultrasound examination. This may result from renal toxic change due to ingestion of cosmetic chemicals.

In a case of acquired Fanconi syndrome secondary to intake of Chinese chicken jerky treats, histopathological examination of renal tissue showed dilated tubules, interstitial fibroplasia, necrotic changes in proximal tubules along with regeneration (Thompson et al. 2013). In cases of human Fanconi syndrome secondary to drug toxicity, electron microscopy commonly reveals heterotypical and bulging mitochondria in the renal proximal tubular cells as a characteristic feature (Hall et al. 2014). Unfortunately, at the request of client, no microscopic examination could be performed for our two dogs.

The exact mechanism of Fanconi syndrome has not been clearly identified. Many hypotheses have suggested that a fault in the reabsorptive function of the renal proximal tubular cell (such as sodium-potassium ATPase transport or receptor-mediated endocytosis) is the key mechanism in drug-induced Fanconi syndrome (Hall et al. 2014). Additionally, experimental studies show that mitochondrial toxins inhibit solute transport of renal proximal tubular cell (Hall et al. 2014). In Dog 2, the cosmetic chemicals may have acted as a mitochondrial toxin, disrupting the functioning of proximal tubular cells. In canine vector-borne diseases, indirect immune-mediated responses to these infections play a major role in symptoms of immune dysregulation (Day 2011). Formation of autoantibodies such as anti-RBC antibodies and immune complex accumulation are an important part of canine vector-borne diseases (Day 2011). Above all, immune complex deposition is one of the mechanisms that cause indirect immune-mediated kidney impairment by disrupting immune homeostasis (Tecklenborg et al. 2018). Immune complexes injure the renal cells, activate the complement cascade, and ultimately form the membrane attack complexes that damage cells (Tecklenborg et al. 2018). These processes may have occurred in Dog 1's renal proximal tubular cell. To identify the exact cause, histopathological examination should be done.

In conclusion, in these cases, there was no relevant previously known history to induce Fanconi syndrome and no history of exposure to other toxic substances except *Babesia* and cosmetics intake. Furthermore, Fanconi syndrome secondary to babesiosis or cosmetics intake has not been reported in the past. Therefore, *Babesia* infection and cosmetics ingestion should be considered as a possible aetiology for transient acquired Fanconi syndrome in dogs.

Conflict of interest

The authors declare no conflict of interest.

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