"Fiocco" e la sua sindrome di Fanconi transitoria secondaria al consumo di stuzzichini per cani a base di carne di pollo essiccata.

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SUMMARY
"Fiocco" and his transient Fanconi syndrome due to jerky treats consumption.
Fiocco, a 9-years-old intact male Rottweiler dog was presented with a 2-week history of polyuria and polydipsia and finicky eating behavior. Physical examination was unremarkable, while urinalysis revealed severe glucosuria without hyperglycemia, proteinuria and granular/epithelial cell casts. Urinary electrophoresis revealed the presence of tubular proteins while urine culture excluded UTI and pyelonephritis. Blood gas analysis detected a mild hyperchloremic metabolic acidosis, with other electrolytes at the lower or upper limit of the reference range. Given the above findings, a condition of renal glucosuria (Fanconi sydrome) due to jerky treats consumption was hypothesized. The jerky treats and all other commercial "extras" were immediately discontinued; dog was hospitalized and an adequate treatment was established. The suspicion was reported to Italian regulatory authorities. Dog recovered uneventfully. The present case report represents the first case of an acquired Fanconi syndrome following the consumption of jerky treats in Italy. For adequately addressing the disease, it is important to make a careful dietary history and perform urinalysis in dogs with nonspecific signs. If an association between the illness and jerky treat consumption is suspected, such treats must be immediately withdrawn. Clinicians should be aware of the toxicosis associated with treats and report cases to regulatory authorities to prevent outbreaks, as we did while managing this patient.

KEY WORDS
Keywords: Fanconi syndrome, jerky treats, chicken stripes, nutrition, dog

SIGNALMENT:

"Fiocco", a 9-years-old intact male Rottweiler dog, weighting 34 kilos (BCS, body condition score 4,5/9 with a normal MMI, muscle mass index).

HISTORY

Fiocco was presented with a 2-weeks history of polyuria and polydipsia. The owner also reported the occurrence of a finicky eating behaviour during the last 3 days: the dog progressively started to refuse the bowl filled with his commercial dry food (Royal Canin Ageing 8+ Maxi), while he was still accepting home-cooked meals (mainly rice, roasted chicken and turkey), biscuits and chicken jerky treats. No vomiting or diarrhoea have been noticed. The dog was used to live indoor, with a free access to a private garden. He was up -to -date with his vaccination, according to WSAVA Guidelines 14 and received regular protection against ectoparasites and Dirofilaria immitis (Frontline Tri-act®, Fipronil+Permetrin, spot-on, once month; Guardian®, Moxidectin 0,17mg/kg SC once year). Fiocco's previous clinical history was unremarkable, apart from an acral-lick dermatitis (ALD) involving the right forearm and occurred since the loss of her sister, Diana, one year before the consultation. ALD was treated by the previous clinician with two courses of a topic ointment (RepyGel®, aldemidrol as main component, applied BID for 5-10 days), an oral steroid (Vetsolone®, Prednisolone 0.5-1.0 mg/kg SID for 5-8 days/course, tapered; last administration 90 days before referral), an antibiotic (ICF VET®, Cephalexin 10mg/kg OS BID for 5-7 days; last administration 90 days before referral) and a behavioural therapy (currently ongoing). Antianxiety drugs, such as Fluoxetin, were prescribed but denied by the owner.

PHYSICAL EXAMINATION

The dog presented with normal mentation, slight dehydration (≈5%) but a normal BCS (body condition score) and MMI (muscle mass index). No abnormalities were detected upon physical examination. The only finding that captured the attention was an alopecic skin lesion on the dorsal surface of the right forearm, between metacarpal and elbow, due to its extensive licking and characterized by fibrosis and scar tissue.

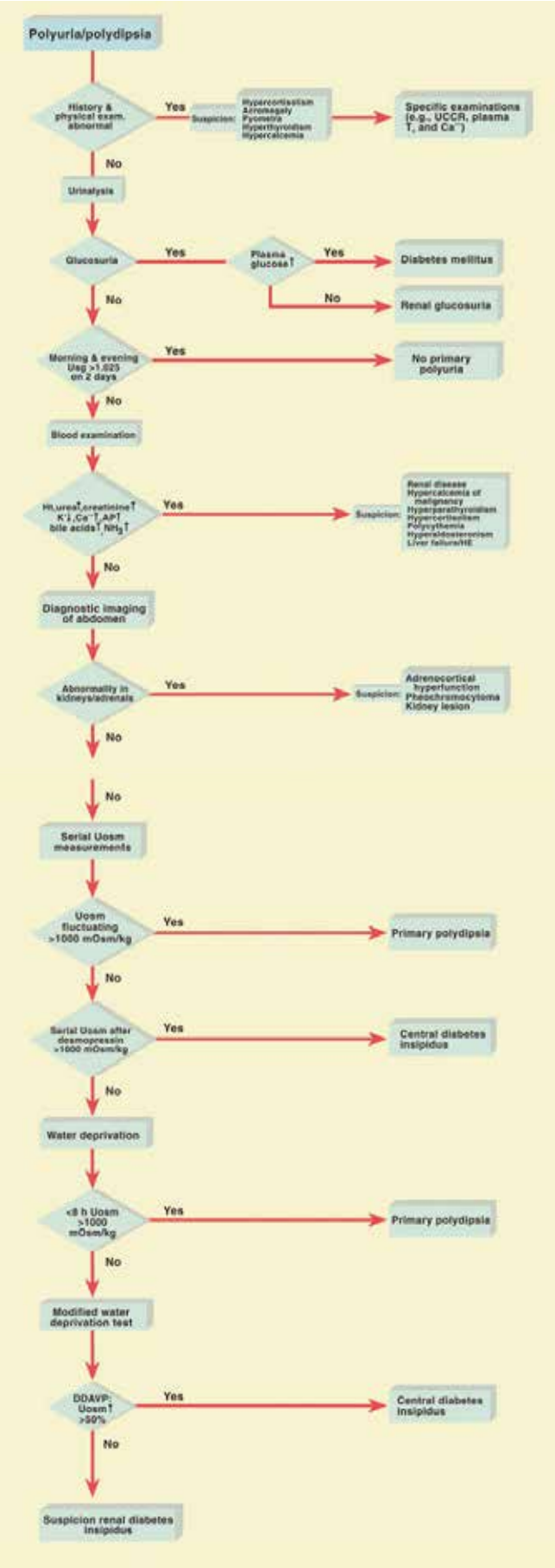
ASSESSMENT I

Polyuria and polydipsia were considered as main findings, assuming dehydration to be related. Differentials for finicky eating behavior/decrease appetite were examined too. The alopecic skin lesion noticed on the right forearm was judged as a direct consequence of ALD, already diagnosed. Other possible causes of obsessive licking (pain, pruritus due to pyodermatitis, paresthesia due to neurological conditions, orthopedic diseases, etc) were considered less likely, considering history and clinical presentation.
Differential diagnosis for polyuria and polydipsia were screened

by considering Rijnberk and Kooistra algorithm (See Graph. 1). (9) Diabetes mellitus, renal glycosuria and its causes, primary polyuria, renal and liver diseases, hypercalcemia due to malignancies, hypercortisolism, polycythemia, primary polydipsia, central and renal diabetes insipidus where then listed as more likely causes.

Dehydrated dogs, indeed, are used to eat less. However, finicky eating behavior and decreased appetite are often related in cats and dogs. Food preferences and minor behavioral issues are responsible for many instances of finicky eating behavior. However, a very large number of diseases can suppress appetite in pets. Common non-medical causes are listed below, with more common causes coming first and less common coming later. Food preference [1] is a leading cause of reduced enthusiasm for food. Like people, pets may be disinclined to consume a food that they do not find palatable. Some animals will refrain from eating as a form of attention-seeking behavior [2]. This is common when pets discover that their owners will give them attention or attempt to hand feed them when they do not eat their food. Stress, fear, or anxiety [3] may cause decreased appetite. Occasionally, owners may have inappropriate expectations [4] regarding their pet's appetite. In these cases, the pet eats normally, but the owner perceives that its appetite is inadequate. Pets that have already been fed by another family member [5], or that have surreptitiously consumed food in the owner's absence may appear to have a low appetite for several hours. Referring to medical causes of decreased appetite, most of them overlap with differentials for polyuria and polydipsia. They include metabolic diseases [6] such as hypoadrenocorticism (Addison's disease) or diabetes mellitus/diabetic ketoacidosis, renal glucosuria and its causes [7], kidney disease [8], liver failure [9] and malignancies [10]. It has been reported that also cardiac diseases [11] can cause anorexia. Decreased appetite may be also a sign of pain [12] or could represent a side effect of many medications [13]. Moreover, any disease or syndrome that lead to gastrointestinal upset [14] could potentially induce anorexia or disorexia; they include dietary indiscretion, intestinal parasites or infections, inflammatory bowel disease, gastric and intestinal neoplasia, pancreatitis, etc. At last but not at least, dental/throat or esophageal diseases [15] may cause a preference for soft food or a decreased enthusiasm for food in general.

Putting together all possible differentials for Fiocco's clinical signs and symptoms (polyuria and polydipsia, decreased appetite) the following causes were elected as being more likely: diabetes mellitus, renal glucosuria (Fanconi syndrome) and its causes, kidney disease, liver failure, hypercalcemia due to malignancies and non-medical causes. Referring to this last aspect, causes [4] and [5] before mentioned were immediately ruled out: owner perception of Fiocco's appetite was judged to be adequate and he was the only person feeding the dog, except for handlers responsible for behavioral therapy and occasionally administering commercial and jerky treats during sessions. Causes [1], [2] and [3] could not be completely excluded do to the history of a concomitant behavioral disorder (ALD); however, we stressed the possibility of a concomitant medical problem. Furthermore, Fiocco was not receiving any medication and no signs of direct or referred pain were reported by the owner or noticed during physical examination. Then causes [12] and [13] were excluded, as well as cause [11], because no other abnormalities aroused during cardiac auscultation, and cause [15], because mouth inspection turned out to be normal and no other signs of esophageal disease (regurgitation, dysphagia, halitosis, ptialism, etc…) were noticed.



Graph 1. Algorithm used to itemize Fiocco's differentials for polyuria and polydipsia. Modified by Rijnberk and Kooistra, 2010. (9)

BLOOD PARAMETERS	Day 0	Day 1	Day 3	Day 6	Day 18	Day 40	Day 90	Day 180	After 1 year	Reference
WBC (103/uL)	9.39		9.20	9.10	10.12	11.2	12	14	8.2	7.5-16.2
RBC (106/uL)	8.00		7.69	7.5	7.46	7.8	7.2	6.8	6.98	5.97-8.11
Hct (%)	45.5		45	44.6	44.4	45	46	44.2	42	40.7-55.3
Hgb (g/dL)	13.9		13.8	13.8	14.7	15	15.9	16.7	14.1	13.1-18.1
Plt (103/uL)	130		154	168	170	220	187	289	245	62-320
TP (g/dL)	6.8		6.5	6.4	6.4	6.3	6.5	6.2	6.8	6-7
Alb (g/dL)	3.7		3.6	3.5	3.5	3.4	3.3	3.5	3.7	3.1-3.7
BUN (mg/dL)	82	62	50	45	28	32	36	29	44	26-37
Crea (mg/dL)	2.6	2.4	2.0	1.5	1.4	1.3	1.2	1.2	1.9	0.4-1.4
Glucose (mg/dL)	100	110	96	89	97				97	98-115
Fructosamine										
Umol/L	230									225-365
ALT (IU/L)	30		36	40	33	56	67	77	78	25-88
ALP (IU/L)	34		28	35	37	33	30	18	23	10-39
TBIL (mg/dL)	0.17		0.18	0.18	0.19	0.17	0.18	0.16	0.15	0.15-0.21
Ca (mg/dL)	10.6		10.4	10.5	10.3	10.2	10.4	10.8	10.4	10.2-10.9
Phos (mmol/L)	1.5	1.4	1.6	1.6	1.3	1.4	1.7	1.6	1.8	0.91-1.90
K (mmol/L)	4.3		4.5	4.8	5.0	5.1	4.8	4.9	5.0	4.2-5.3
Na (mmol/L)	148		150	151	149	151	153	151	150	142-154
Cl (mmol/L)	146		138	132	129	130	133	131	130	106-135
pH	7.31		7.36	7.42	7.41					7.35-7.45
HCO3 (mmol/L)	19.1		21	22	22					20-24
pvCO2 (mmHg)	35.7		43	44	45					40-50
BE (base excess) (mmol/L)	-5.7		-3.7	-2.0	-2.5					-4/+4

Table 1. Relevant blood parameters at admission (Day 0), during hospitalization (Day 1-6) and follow-up consultations (Day 18, 40, 90, 180 and 1 year)

Steroids were not administered chronically and at the end of each course its discontinuation was adequately performed. Moreover, clinical presentation was not fitting with the hypothesis of a hypothalamic-pituitary-adrenal axis unbalance. Iatrogenic hypercortisolims was then considered unlikely.

MANAGEMENT I

The dog was hospitalized and a complete urinalysis with urine protein/creatinine ratio (UP/UC) was first performed, according to Rijnberk and Kooistra algorithm. (9) A venous blood sample for hematology and biochemical analysis was also collected. While waiting for their results, radiographic evaluation of the thorax was also obtained to exclude malignancies.

ASSESSMENT I

Diffuse mineralization of costocondral joints and increased bronchointestitial markings were noticed on thoracic radiographs, but judged to be age-related. No signs of malignancies were noticed. Urinalysis showed a specific gravity of 1040, a PH of 6.0, severe glycosuria (4+, determined by dipstick) and proteinuria (2+, determined by dipstick). Urine protein/creatinine ratio (UP/UC) was 0.56. Examination of urine sediment identified some granular and epithelial cell casts. Pyuria or bacteriuria were not detected. Blood tests revealed normal complete blood cell count (CBC) and a mild increase in blood urea nitrogen (BUN) and creatinine. Blood glucose was within normal range (see Annex, Table 1, Day 0).

MANAGEMENT II

Given glucosuria in the absence of hyperglycemia, proteinuria, granular and epithelial cell casts, a condition of renal glucosuria (Fanconi sydrome) was hypothesized and its possible causes were then carefully explored. All other differentials, medical and non-medical, were excluded according to results of hematology, urinalysis and serum biochemistry.
Medications associated with acquired Fanconi syndrome, such as cyclosporin or NSAIDs, had not been administered prior onset of clinical signs. A possible exposure to heavy metals or other potential toxic agents able to induce a tubular damage were excluded. Furthermore, reports of Fanconi syndrome in dog secondary to cephalexin or prednisolone administration were not found in veterinary literature. Metabolic disorders such as hypoparathyroidism or hepatotoxicities were excluded by the absence of abnormalities on liver enzymes and calcium levels. Infectious diseases such as Leptospirosis and Rickettsiosis were then considered. In human medicine, Fanconi syndrome may result from tubular damage secondary to urinary excretion of abnormal proteins. The proteins may be of prerenal origin, as in multiple myeloma; of glomerular origin or of tubular origin, as in all tubulopathies. The latter are the result of impaired

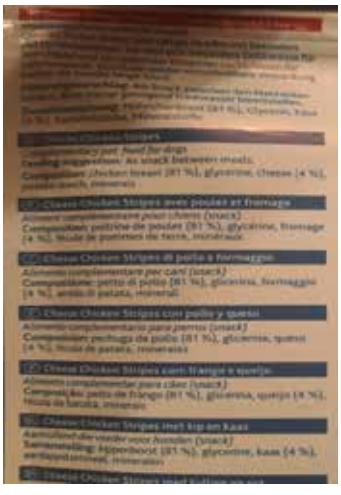
URINALYSIS	Day 0	Day 3	Day 6	Day 18	Day 40	After 1 year	Reference
SG	1040	1035	1035	1035	1030	1026	1023-1047
PH (Unit)	6.0	6.5	6.5	6.5	6.5	6.5	5.5-8.5
Glucose (mg/dL)	4+	2+	1+	negative	negative	negative	negative
Proteins (mg/dL)	2+	2+	traces	negative	negative	negative	Negative
Ketones (mg/dL)	negative	negative	negative	negative	negative	negative	negative
RBC (cells x HPF 40x)	absent	absent	absent	absent	absent	absent	absent
Leucocytes (cells x HPF 40x)	absent	absent	absent	absent	absent	absent	absent
Casts	++	+	absent	absent	absent	absent	absent
UP/UC (mg/dL)	0.56	0.5	0.4	0.36	0.34	0.49	0.1-0.5
Urinary electro-phoresis (SDS-AGE)		Proteins from tubular origin					

Table 2. Urinalysis at admission (Day 0), during hospitalization (Day 1-6) and follow-up consultations at Day 18, 40 and after 1 year

reabsorption of small proteins, such as enzymes, peptide hormones, and light chain immunoglobulins. Their molecular weight varies from 5-50 kD. UTI and pyelonephritis needed to be excluded too, even if urine sediment was negative for bacteria, neutrophils and leucocytes.
For aforementioned reasons, a detailed review of Fiocco's dietary history was performed. In-house tests for Leptospira spp (IDEXX Snap Lepto Test), Ehrlichia spp and Anaplasma spp (IDEXX Snap 4DX Plus test) were executed. A urine sample was submitted to "Laboratorio Veterinario San Marco" (Padova, PD, IT) in order to perform a urinary electrophoresis, able to differentiate proteins of tubular, glomerular or mixed origin and a urine culture. Urinary amino acid profile to demonstrate additional amino acid reabsorption defect was not performed, due to limited availability of this assay. A venous blood gas analysis was also advised, looking for metabolic acidosis.

ASSESSMENT II

Dietary history revealed that Fiocco was administered, since 2 months, with two types of chicken and jerky treats during behavioral sessions (see Annex, Figure 1 a,b,c and Figure 2 a,b). Snap tests performed to exclude Leptospirosis and Rickettsiosis turned out to be negative. Urinary electrophoresis revealed the presence of tubular proteins while urine culture excluded UTI and pyelonephritis. Blood gas analysis detected a mild hyperchloremic metabolic acidosis, with other electrolytes at the lower or upper limit of the reference range (see Annex, Table 1, Day 1).



DIAGNOSIS AND TREATMENT

An acquired Fanconi syndrome secondary to chicken jerky treats consumption was hypothesized. The jerky treats and all other commercial "extras" were immediately discontinued. The suspicion was reported to Italian regulatory authorities. Fiocco was treated with acetated Ringer's solution IV with 30 mEq/L of KCL in order to correct electrolyte unbalance. Potassium sodium hydrogen citrate (Uralyte-U®, PO, BID), famotidine (0.5 mg/kg IV, SID) and maropitant (2 mg/kg SC, SID) were also administered. Other oral alkali components were judged unnecessary on the basis of blood gas analysis. A high-quality protein diet in order to offset the hypothesized urinary amino acids losses was also prescribed.
On day 3 of hospitalization, urinalysis was repeated revealing that glucosuria was still present, but mildly improved (See Annex, Table 1, Day 3). BUN, creatinine, potassium and chloride levels were measured again. Metabolic acidosis was resolved.
On day 6, dog presented clinically normal with a normal appetite. The urinalysis still showed a mild glucosuria with mild azotemia (See Annex, Table 2, Day 6). The dog was then discharged and daily subcutaneous fluid therapy was continued at home by the referring veterinarian.

FOLLOW-UP

Eighteen days after the initial presentation, Fiocco was still clinically healthy. Glucosuria and azotemia completely solved. Referring to the alopecic lesion on right forearm, secondary to ALD, the extensive fibrosis was treated with Airplasma® ablation (onemytis.it). A BiteNot collar (bitenot.com) and a no-licking bandage were placed to protect the surgical site. A topical ointment (RepyGel®, adelmidrol as main component) was administered twice-daily, until healing. Behavioral treatment was prosecuted with the indication of not giving commercial treats during sessions. Only rice crackers or home-cooked biscuits were allowed. Renal function was routinely checked after 40, 90 and 180 days without significative changes. One year after referral, polyuria and polydipsia were noted again and a chronic kidney disease was diagnosed (CKD IRIS Stage 2, borderline proteinuric, no hyperten-

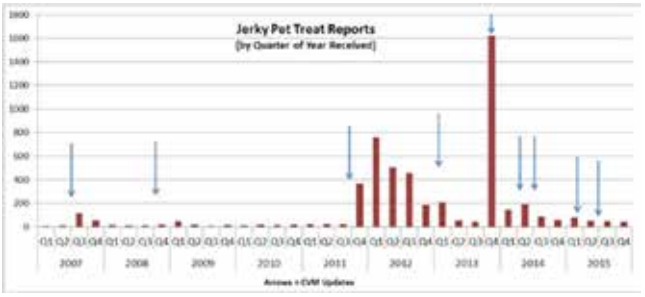
Figure 1 a,b,c. Cheese chicken stripes administered to Fiocco since 2 months before referral and their ingredients.

Figure 2 a,b. Dry chicken jerky administered to Fiocco since 2 months before referral and their ingredients.

sion).(6) The dog was prescribed with a commercial renal diet (Hill's k/d) and supplemented with omega-3 fatty acids and antioxidants (DuoOmega®, Candioli+ DIRENE®, DRN). Fiocco is now a lively 11-years old geriatric dog. CKD is well managed but, unfortunately, he is still struggling with his ADL, even if improvements have been obtained with behavioral treatment (See Annex, Figure 3). At this precise moment, he is staring at me, begging for treats…
Fiocco, indeed, became my own dog and his owner became my husband. Jerky treats have been banned from our house and a rigid policy of extras' management has been adopted.

DISCUSSION

Fanconi syndrome is a complex renal tubulopathy with marked proximal tubular dysfunction and loss of various combinations of amino acids, phosphate, glucose, bicarbonate, calcium, potassium and other ions as well as potentially excessive urinary loss of water. (2) The defective bicarbonate reabsorption leads to proximal renal tubular acidosis evidenced by acidic urine in the face of hyperchloremic metabolic acidosis. (2) The dog in this report presented with hyperchloremic metabolic acidosis, normoglycemic glycosuria, tubular proteinuria. Urinary amino acid profile to demonstrate additional amino acid reabsorption defect was not performed in this case due to limited availability of this assay at the time.
Common clinical signs in affected dogs include anorexia, polyuria and polydipsia, lethargy, diarrhea, vomiting, weight loss and generalized weakness. Seizures have also been reported in approximately 15% of cases. (7,10,12) A tentative diagnosis is suggested by hematological changes including azotemia, high liver enzyme activities and hyperchloremic metabolic acidosis. Urinalysis characteristically shows aminoaciduria and the presence of glucosuria. Fiocco's clinical presentation was in line with what previously described in literature, even if he was paucisymptomatic and only mild abnormalities were detected on physical examination. This was likely due to the owner's request of a clinical consultation at an early stage of the disease or to still minor damages to proximal renal tubule. The dog also exhibited renal glucosuria without hyperglycemia. This represent a very consistent finding in Fanconi syndrome patients. Electrolytes and other ions, apart from Cl- levels, turned out to be all within reference ranges, even if at higher or lower levels. Hypokalemia, however, was expected to be a consistent finding, according to published case reports. (5,7) We hypothesized that hypokalemia would be observed in a later course of the dysfunction (i.e. if Fiocco presented 3-4 weeks after the onset of clinical signs) because it may reflect the longstanding nature of the disease with depletion of intracellular stores. (7) Mild azotemia was recorded too, without impairment of liver function. Acquired causes of Fanconi syndrome are multifold. (16) In 2006, it was recognized for the first time in dogs consuming chicken jerky treats made in China. (4) New reports of the disease are still ongoing at the U.S. Food and Drug administration (FDA). (13) As of December 31, 2015, FDA has received approximately 5,200 complaints of illnesses associated with consumption of chicken, duck, or sweet potato jerky treats, many of which involve products imported from China, which produces much of the jerky pet treats on the market (See Graph 2). The reports involve more than 6,200 dogs, 26 cats, three people, and include more than 1,140 canine deaths. The complaints FDA has received include reported adverse events involving different sizes, ages, and breeds of dogs. About 60 % of the reports are for gastrointestinal illness (with or without elevated liver enzymes) and about 30 % relate to kidney or urinary signs. The remaining 10% of cases involve a variety of other signs, including convulsions, tremors, hives, and skin irritation. We know that the illnesses and deaths reported are most often, but not always, linked to jerky pet treats sourced from China. Pet owners should be aware, however, that manufacturers are not required to list the country of origin for each ingredient used in their products. Although it is impossible to conclude definitively in every case whether the events reported were caused by eating jerky pet treats, FDA continues to believe that there is an association between some of the reports and consumption of jerky pet treats. (13) Outbreaks have been also reported in Australia and Europe, and at the best of my knowledge this could be the first report in Italy. (1,3,12) Fanconi's syndrome can also occur as a secondary phenomenon as a result of various renal insults from ingested toxins, infections or idiosyncratic drug reactions that collectively damage the proximal renal tubule and interrupt normal functioning. Idiopathic Fanconi's has been reported as well in the Norwegian Elkhound, Labrador Retriever, Shetland Sheepdog and Miniature Schnauzer. Secondary Fanconi's syndrome has been associated with a number of primary disease states, including: congenital renal dysplasia, exposure to heavy metals (lead, mercury, cadmium and uranium), iatrogenic - antibiotics (gentamicin, cephalosporins, tetracyclines, cisplatin, streptozotocin), renal neoplasia - multiple myeloma and monoclonal gammopathies, copper storage hepatopathy, hypoparathyroidism. A differential list of other causes would include Rickettsia spp, and Leptospira spp infection. (8,10,16) In the present case report a detailed history, mainly focused on dietary management and the exclusion of all other possible causes allowed to address chicken jerky treats consumed by the dog as the perpetrators of transient acquired Fanconi syndrome. A possible exposure to heavy metals or other potential toxic agents able to induce a tubular damage were excluded. Furthermore, reports of Fanconi syndrome in dog secondary to cephalexin or prednisolone administration were not found in veterinary literature. In humans, according to FDA reports, Fanconi syndrome is not reported as an adverse reaction in patients receiving cephalexin, while percentage of patients



Graph 2. After FDA issued CVM Updates about its Jerky Pet Treats investigation (indicated by the arrows in the graph above), the agency received an increase in reports from the public. The most pronounced increase was in late 2013, when FDA issued our most comprehensive update, which included a "Dear Veterinarian" letter requesting specific clinical data and providing a fact sheet for pet owners. Reported cases appear to be tapering off and have not exceeded 100 per quarter for the past 1.5 years

receiving prednisolone in which Fanconi syndrome is a reported side effect is 0.0044%. (13) We do also considered the possibility of a pre-existing, undiagnosed chronic renal failure worsened by the administration of cephalexin, but clinical history, dosages and times of administration (only 2 courses of 5 and 7 days respectively, 90 days before referral) and follow-up after jerky treats withdrawal strongly supported the suspicious that these treats (and not the antibiotic) could be linked to the development of the disease.
A definitive diagnosis is one of exclusion, requiring a negative serology for Leptospirosis and Rickettsia spp., serum fructosamine ≤250 µmol/L (to exclude subclinical diabetes) and, ideally, a renal biopsy which confirms proximal renal tubular nephropathy. The prognosis in most cases is generally good if the trigger can be removed. Management depends on severity of clinical picture, with some patients (as Fiocco) hospitalized only for few days and others which needed intensive support and emergency cares. The traditional treatment for Fanconi's syndrome was administration of potassium citrate, but oral alkali supplementation with sodium bicarbonate also appear efficacious at maintaining longevity in severe cases. The increased amount of solutes in the urine causes an osmotic diuresis and an inability to concentrate urine. Therefore, it is important that affected dogs always have access to water. In a retrospective study of dogs with acquired Fanconi syndrome, 6 of 102 dogs died or were euthanized as a result of their illness, while survivors required ongoing treatment up to six months for resolution of clinical signs. (12) In many dogs with persistent, chronic Fanconi syndrome progressive renal dysfunction is the main concern and the most common cause of death or euthanasia. At the time of writing, despite extensive investigations by FDA and other associations, the specific causative substance contained in jerky treats and causing the tubular dysfunction has not been determined, and unfortunately it could not be investigated in this report too. In humans, degraded tetracyclines have been implicated as a cause of acquired Fanconi syndrome. (8) Recently, six illegal antibiotics have been detected in chicken jerky treats for dogs. (11) However, there is no evidence indicating the direct relation between these molecules and the disease.
In conclusion, this case report could represent the first case of Fanconi syndrome following the consumption of jerky treats in Italy. For adequately addressing the disease, it is important to make a careful dietary history and perform urinalysis in dogs with nonspecific signs. If an association between the illness and jerky treat consumption is suspected, such treats should be immediately withdrawn. Clinicians should be aware of the toxicosis associated with treats and report cases to regulatory authorities to prevent outbreaks, as we did while managing this patient.

Figure 3. Fiocco. Picture captured at the time of writing

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