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Opsomer, Han ; Wichert, Brigitta ; Liesegang, Annette

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CASE REPORT

Wildlife

Homemade diet as nutritional support for a dog suffering from chronic pancreatitis and inflammatory bowel disease

Han Opsomer | **Brigitta Wichert** | **Annette Liesegang**

Institute of Animal Nutrition and Dietetics, Vetsuisse Faculty, University of Zurich, Zurich, Switzerland

Correspondence
Annette Liesegang, Institute of Animal Nutrition and Dietetics, Vetsuisse Faculty, University of Zurich, Winterthurerstrasse 270, 8057 Zurich, Switzerland.
Email: aliese@nutrivet.uzh.ch

Abstract

A spayed Boston terrier was referred for nutritional advice after failing to respond to long-term medical and dietary treatment for chronic pancreatitis. Based on clinical appearance, consecutive bloodwork and ultrasound examination, the concomitant occurrence of inflammatory bowel disease was likely. An exclusion diet (novel protein approach) and a stepwise balancing schedule were formulated to address combined nutritional recommendations, as well as the possibility for hypersensitivity to play a role. After initiation of the exclusion diet, the patient relapsed twice. Once due to noncompliance of the owner, once due to reaction to an added supplement. After correction, no major relapses were noted for at least 1 year. To the authors' knowledge, this report is the first to describe the successful supportive care of chronic pancreatitis and concurrent inflammatory bowel disease by prescribing a homemade diet.

KEYWORDS

dogs, inflammatory bowel disease, nutrition, pancreatitis

BACKGROUND

Pancreatitis remains a challenge for the veterinarian. Its nonspecific presentation[1,2] and the lack of sensitive, noninvasive tests (although markedly improved) often prohibit straightforward diagnosis.[3–6] This is even more true for chronic pancreatitis.[7] Its occurrence is therefore likely underestimated.[8–10] In addition, it is suspected that (chronic) pancreatitis is caused by an interplay of multiple risk factors rather than one trigger, which in human medicine have been summarised in the Toxic-metabolic, Idiopathic, Genetic, Autoimmune, Recurrent and severe acute pancreatitis and Obstructive Pancreatitis Risk (TIGAR-O) system.[6] This categorises the predisposing factors as (a) toxic metabolic, (b) idiopathic, (c) genetic, (d) autoimmune, (e) recurrent and severe acute pancreatitis or (f) obstructive. The importance of each category in the pathogenesis of chronic pancreatitis may be species-dependent. In dogs, hereditary, nutritional and metabolic triggers (e.g., hyperlipidaemia, obesity, high fat foods, etc.) are recognised,[3,7,11–15] whereas in cats this is far less obvious.[4,9,16] As an inciting cause is rarely determined, treatment and subsequent management often remain nonspecific,[6,7,17] with nutritional support playing a key role in the successful approach. However, concurrent conditions, like inflammatory bowel disease (IBD), can add to the complexity thereof.[17] For both pancreatitis and IBD, dietary principles base on a highly digestible diet (digestibility of protein ≥87%, fat and digestible carbohydrates ≥90%), which dictates the use of foodstuffs (e.g., rice, muscle meat and extracted oils as carbohydrate, protein and fat source, respectively).[18–20] To avoid exceeding the patient's digestive capacity, frequent small meals (individual tolerance) are warranted.[21,22] Pancreatitis as well as IBD also warrant a moderate to reduced fat content in the diet (dry matter basis [DMB]: ≤10%–15% for pancreatitis, 8%–15% for IBD; metabolisable energy [ME] basis: ≤20%–30% for pancreatitis, ≤15% for IBD in case of lymphangiectasia).[21–26] A moderate protein content of the diet (15%–30% DMB) has been advised in case of pancreatitis, whereas in IBD both an increase and decrease of the protein content can be argued.[22,23,27] The necessity of supplementation with vitamins is dependent on the type of illness. For pancreatitis and IBD, special focus goes to cobalamin and fat-soluble vitamins. For IBD, folate and thiamine warrant attention as well.[28,29] Hypersensitivity should always be suspected in case of IBD. The implementation of an exclusion diet is therefore advised.[21,30] Due to the partly contradicting nutritional recommendations with such comorbidities, an individual approach is mandatory to implement the best-fitting compromise. Often, a homemade diet is the best option to meet the specific nutritional profile of the patient.[30]

CASE PRESENTATION

A spayed Boston terrier (6.5 years old at the time of presentation), weighing 7.4 kg (estimated to be ideal), with a

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known heart murmur (2/6, frequently monitored, no medical treatment) was referred to the Institute of Animal Nutrition and Dietetics of the Vetsuisse Faculty, University of Zürich, due to the patient's failure to respond adequately to long-term treatment for pancreatitis with a reduced fat diet (commercial therapeutic dry and wet food), initiated by the Internal Medicine Department of the Vetsuisse Faculty, University of Berne. Before referral, the patient suffered from severe episodes of apathy, anorexia, vomiting and occasional diarrhoea. This resulted three times in hospitalisation at two-monthly intervals whenever the patient deteriorated rapidly. Despite its reduced appetite, the patient always retained an ideal to slightly increased body condition scores (BCS), ranging from 5/9 to 6/9 (estimated 10% overweight). A likely reduced activity (and therefore energy expenditure) during acute phases might explain this stable BCS. Muscle mass was considered normal. Aside from slight abdominal discomfort and occasional mild dehydration, no other abnormalities were found during clinical examinations. Repeated blood examinations showed hypoproteinaemia, hypoalbuminaemia, hypocalcaemia, hypokalaemia and increased amylase, lipase and canine C-reactive protein (CRP) values. The blood examination before referral also showed a marked hyperglycaemia, hypocholesterolaemia and hypoglobulinaemia (Table 1). Unfortunately, it was not clear when this blood sample was taken in relation to the start of the therapy. This is important as the clinical data show a glucose bolus was administered due to hypoglycaemia, although no corresponding value thereof could be traced in the records. Although unlikely, it was unknown whether the animal had eaten shortly before taking this sample, which could have influenced the blood glucose as well. Neither a canine pancreatic lipase immunoreactivity (cPLI) test nor a check-up of the patient's vitamin B status was conducted.

Abdominal ultrasound following the episode before referral excluded obstructive disorders (either partial or total) and indicated acute pancreatic pathology, likely concurrent with chronic pancreatitis. Clear signs of lymphangiectasia were not found. Parasitic infection was excluded (faecal examination, monthly deworming with milbemycin oxime and praziquantel). When hospitalised, the patient was treated with intravenous (IV) fluid therapy, anti-emetics (ondansetron: 1.55 mg IV every 8 hours), antibiotics (metronidazole: 76 mg IV every 24 hours), analgesics (methadone: 0.8 mg IV every 4 hours) and subsequently managed on a low-fat diet (see nutritional approach) as guidelines indicate.[17] Due to the recurring nature of this case, the ultrasound examination, the consecutive bloodwork and the patient's clinical presentation, IBD was suspected to be a complicating factor. Biopsies for confirmation were rejected by the owner. In dogs, IBD is more considered as a differential or a concurrent condition with chronic pancreatitis,[17] in contrast to humans and cats where a correlation between infiltrative gastrointestinal disease and (chronic) pancreatitis has been noted.[31,32]

NUTRITIONAL SUPPORT

The nutritional constituents of the patient's subsequent diets are specified in Table 2 and were calculated with Diet Check Munich 2005 Version 3.0 (RV Software; based on NRC 2006, modified by Dobenecker and Kienzle, Munich, Germany).

Diet A (diet fed before patient developing clinical signs) consisted of two commercial extruded dry foods supplemented daily with an estimated 100 g of varying fruits (e.g., apple, orange, banana, berries). As no data were available on the mineral content of the commercial products, it was not possible to verify if all the patient's requirements were met. Diet B (diet prescribed by the Internal Medicine Department of the Vetsuisse Faculty Berne) consisted of mixture of highly digestible low-fat dry and wet food, supplemented daily with 10 g of cottage cheese. This initial nutritional adjustment, as recommended by the Internal Medicine Department of the Vetsuisse Faculty Berne (Diet B compared to Diet A), markedly reduced the fat content (g) and concentration (% of dry matter) of the diet, while increasing the protein and nitrogen-free extract (NfE) content (mainly digestible carbohydrates like starch and soluble fibre from sugar beet pulp, psyllium, etc.) to keep the daily ration isoenergetic. As this general approach was insufficient to prevent subsequent flare-ups, an alternative nutritional treatment (Diets C_1 and C_2; Tables 2 and 3) was initiated after referral to the Institute of Animal Nutrition and Dietetics. Diets C_1 and C_2 represent the exclusion diet and its adaption to the patient's requirements, respectively (completely homemade, Table 3), advised by the Institute of Animal Nutrition and Dietetics of the Vetsuisse Faculty Zürich.

After 8 weeks, the original exclusion diet was balanced stepwise by adding further feedstuffs and supplements at 2-week intervals to ensure the latest added ingredient was tolerated. The protein content was reduced in addition to a further decrease of the fat content in Diets C_1 and C_2 (compared to Diets A and B). A vitamin B tablet (relatively high in cobalamin) was added. When this supplement seemed to cause symptoms to reoccur (see follow-up), it was replaced by a water-based vitamin B solution. Furthermore, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) were supplemented (195 and 166 mg, respectively) as well. The omega 6 to omega 3 polyunsaturated fatty acid ratio of the complete diet was calculated as 2:1. With the exception of mineral requirements in Diet A (could not be verified), all diets (A, B, C_2) covered the recommendations for a healthy adult dog with an ideal bodyweight of 7.4 kg, as specified by the National Research Council.[19] The patient maintained an ideal body condition (5/9) before developing clinical signs. The energy content of the original diet (Diet A) was therefore

LEARNING POINTS/TAKE-HOME MESSAGES

- Nonresponsive chronic pancreatitis patients to initial treatment, should be examined for aetiological factors or concurrent disease.
- Concurrent gastrointestinal, infiltrative disease should be considered.
- Dietary support remains the basis for successful management of the patient.
- Inflammatory bowel disease patients can respond well to the prescription of an exclusion diet.
- Homemade diets are easy to adapt if necessary.

TABLE 1 Initial blood results in first opinion practice (D-124) and subsequent blood examinations conducted at the Internal Medicine Department of the Vetsuisse Faculty, University of Berne

	Reference	D 132	D 92	D 90	D 21	D 20	D 52	D 54
Glucose (mmol/L)	4.16–6.69		4.78		**16.37**		4.5	
Cholesterol (mmol/L)	3.47–10.03				**1.26**			**2.34**
Total protein (g/L)	55–73			**52**	**34.2**	**50.8**		**47.9**
Albumin (g/L)	30–41		**23.6**	**26.1**	**24.3**	32.5		**28.2**
Globulin (g/L)	19–39				**9.9**			19.7
AP (U/L)	9–132				35			62
ALT (U/L)	26–126				41			50
γGT (U/L)	<7				4			3
Amylase (U/L)	500–1500	**>2500**						
Lipase (DAG) (U/L)	200–1300	**2936**						
DGGR (U/L)	24–108		77		**253**			36
Canine CRP (mg/L)	<10.0	6.1	**12.6**					**67.3**
	<10.7				7.9			
Urea (mmol/L)	3.3–10.8				6.0			
Creatinine (μmol/L)	52–117		70		58			
Calcium (mmol/L)	2.42–2.85				**2.08**			**2.19**
Potassium (mmol/L)	3.5–5.8	4.5						
	3.95–5.40		**3.81**					4.40
	3.7–4.8				**3.03**		3.87	
Chloride (mmol/L)	109–122	116						
	106–118		117					115
	108–122				**107**		**107**	

Note: Measurements using differing reference values are given per level. The day of implementation of the prescribed diet by the Institute of Animal Nutrition and Dietetics of the Vetsuisse Faculty, University of Zurich is taken as Day (D) 0.
The bold value in the table indicates outside reference value.
Abbreviations: DAG, 1-oleoyl-2,3-diacetylglycerol; DGGR, 1,2-o-dilauryl-rac-glycero-3-glutaric acid-(6′-methylresorufin).

considered appropriate and was maintained during further treatment under reservation of necessary amendments depending on the further progress. This corresponds to 98% and 118% of the metabolisable energy requirement according to FEDIAF and NRC calculations, respectively[19,33] (Table 4).

It was advised to feed the daily diet well mixed, as multiple small meals (at least three) per day. No other ingredients than those prescribed in the recommendation at each specific time point should be fed. In both the initial recommendations by the Internal Medicine Department (Vetsuisse Faculty Berne), as well as the one by the Institute of Animal Nutrition and Dietetics (Vetsuisse Faculty Zürich), the diet was formulated to be highly digestible and fat reduced in compliance with general recommendations for chronic pancreatitis. This was done, as it would relieve the patient's digestive tract and would mitigate the pancreatic stimulation. The focus on multiple small meals per day addresses this as well.[23,24,28] The main difference was the inclusion of IBD as a comorbidity in the approach by the Institute of Animal Nutrition and Dietetics. As hypersensitivity had to be expected to play a role in its pathogenesis, an exclusion diet (novel protein approach, based on the patient's nutritional history) was deemed most appropriate. Strict compliance to the plan described above is mandatory to avoid a reaction to the suspected triggering antigens and therefore prolonging recovery.[21,22,30] As IBD reduces the patient's digestive capacity as well, the implementation of a cooked highly digestible diet, fed as multiple small meals per day, complies with its recommended treatment as well. Even though for IBD, a moderate fat intake usually suffices (if no malabsorption due to, e.g., lymphangiectasia is noted), the reduced fat intake as advised for pancreatitis can be well accommodated to IBD recommendations. The fat source (hemp oil) was however well considered for its high concentration of essential fatty acids, as well as its optimal omega 6 to omega 3 fatty acid ratio. The latter ratio was further optimised by the direct addition of EPA and DHA through cod liver oil and an additional supplement. Both a low omega 6 to omega 3 ratio, as well as the direct addition of EPA and DHA have anti-inflammatory effects. Although clear recommendations on the effective quantities of EPA and DHA in diets for dogs are lacking (range 50–300 mg/kg bodyweight/day[22]), an omega 6 to omega 3 ratio of <5 can be used as a guideline.[21]

The number of necessary portions per day is dependent on the required energy intake of the patient as well as its individual tolerance (as larger portions can induce nausea, maldigestion and in this case overstimulation of the pancreas[23,24,28]). Even though a gradual reduction of feeding frequency for long-term management is advised, a minimum of three meals per day was recommended.[21,22] The lacking examination of the patient's vitamin B status and the unlikeliness of water-soluble vitamins to cause side effects when supplemented, warranted the addition of three main vitamins: cobalamin (vitamin B_{12}), folate (vitamin B_9) and thiamine (vitamin B_1). Cobalamin is mentioned first as both pancreatitis as well as IBD can impair its absorption by either a decreased pancreatic production of intrinsic factor (IF) and

TABLE 2 Comparison of subsequent diets, calculated with Diet Check Munich 2005 and compared to its given recommended allowance

	RA	Diet A (<D 92)	Diet B (D 92)	Diet C_1 (D 0)	Diet C_2 (D 126)
Energy (MJ ME)	2.1	2.1	2.1	2.1	2.1
Crude protein (g)	25	36	41	31	30
Nitrogen-free extract (g)	/	45	75	97	91
Fat (g)	/	20	10	4	9
Fat (% dry matter)	/	16.9	7.2	2.7	6.1
Calcium (mg)	584	1394	1298	55	628
Phosphorus (mg)	438	1011	731	614	586
Magnesium (mg)	88	[a]	125	217	203
Potassium (mg)	584	[a]	765	666	641
Sodium (mg)	117	[a]	355	125	125
Iron (mg)	4.4	[a]	21.5	11.1	13.0
Copper (mg)	0.9	[a]	2.7	0.8	2.4
Zinc (mg)	8.8	[a]	19.4	4.2	12.3
Manganese (mg)	0.7	[a]	4.1	2.8	3.2
Chloride (mg)	175	[a]	1104	254	252
Iodine (μg)	128	[a]	154	0	160
Vitamin A (IU)	737	1670	15,101	0	770
Vitamin D (IU)	79	166	113	0	193
Vitamin E (mg)	4	96	80	1	6
Vitamin B_1 (mg)	0.33	0.55	5.07	0.42	0.91
Vitamin B_{12} (μg)	5	12	16	3	5/17[b]

Note: Values given on a daily basis. The day of implementation is noted between brackets for each diet, with the initiation of the prescribed diet by the Institute of Animal Nutrition and Dietetics of the Vetsuisse Faculty, University of Zurich taken as Day (D) 0.
Abbreviations: Diet A, diet before clinical disease; Diet B, diet initially prescribed by the Internal Medicine Department of the Vetsuisse Faculty, University of Berne; Diets C_1 and C_2, exclusion and completely balanced homemade diet, respectively, prescribed by the Institute of Animal Nutrition and Dietetics of the Vetsuisse Faculty, University of Zürich; RA, recommended allowance.
[a]Could not be verified due to lack of data.
[b]After change of cobalamin supplement.

TABLE 3 Quantity of ingredients in Diet C_1 (exclusion diet) and Diet C_2 (balanced homemade diet) as recommended by the Institute of Animal Nutrition and Dietetics of the Vetsuisse Faculty, University of Zurich

	Diet C_1	Diet C_2
Horsemeat (ca. 3% fat, raw weight) (g)	90	90
Bulgur (raw weight) (g)	140	130
Hemp oil (g)	/	3
Cod liver oil (g)	/	0.7
Calcium carbonate (g)	/	1.6
Trace element supplement (g)	/	0.8
Salt (NaCl) (g)	0.2	0.2
Vitamin B supplement (g)	/	2
EPA and DHA supplement (g)	/	1.1

Abbreviations: EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid.

TABLE 4 Estimated and calculated energy requirements

	FEDIAF (2020)	NRC (2006)
Formula	460 kJ ME/kg $BW^{0.75}$	398 kJ ME/kg $BW^{0.75}$
Calculated RA (kJ)	2064	1785
Estimated requirement (kJ)	2100	2100
Ratio calculated to estimated RA (%)	98.29	117.65

Note: FEDIAF: formula for moderate activity (1–3 h/day); NRC: formula for inactive pet dogs. The ideal bodyweight of the patient (7.4 kg) was used to calculate the estimated requirements with the given formulas. The estimated requirement was based on the energy content of the initial diet (Table 2).
Abbreviations: BW, bodyweight; ME, metabolisable energy.

the impaired absorption of the IF–cobalamin complex in the ileum, respectively.[21–24,28,34–36] Although pancreatitis can lead to high folate production,[36] damage of the jejunal mucosa from IBD can impair its absorption.[22,37] Similarly, thiamine can be deficient due to chronic enteropathies and their negative effect on the absorptive capacity in the small intestine.[22,38] In this case especially, thiamine deserves special attention as an increased digestible carbohydrate content of the diet also increases the thiamine requirement.[38] In case of fat maldigestion, fat-soluble vitamins should be provided well above the maintenance requirements.[21–24,28,39] As this appeared to be no issue in this patient, the quantities provided by cod liver oil were deemed sufficient.

Parenteral administration of vitamins or the use of water-soluble formulations (for fat-soluble vitamins) as in humans[40] and pigs[41] should be considered if monitoring indicates oral supplementation with conventional sources to be insufficient. Despite marked similarities in their dietary principals, prescribing the right diet can be tricky due to different dietary claims for pancreatitis and IBD. For the former, a moderate quantity of protein is advised to reduce stimulation (15%–30% DMB).[23,24,28] For the latter, generally ≥25% protein on a dry matter basis is advised to counter increased losses, which

were also obvious in this case (Table 1).[21,22] As both chronic enteritis and pancreatitis resulting exocrine pancreatic insufficiency can lead to dysbiosis, another point of careful consideration is the addition of fibre to the diet.[42–44] On the one hand, soluble and insoluble fibres can be used as prebiotic to counter dysbiosis and as motility-regulating binding agent, respectively. On the other hand, an increased fibre content renders a lower digestibility of the diet.[21,22,43,45] Furthermore, the supplementation of pancreatic enzymes can improve signs of malabsorption and dysbiosis as well.[46] Ultimately, a reduction of the protein content and no additional fibre or enzymes in the diet were chosen in the approach (see discussion). The compromises in possible approaches, as well as to enable a quick adaptation of the diet if necessary, favour the calculation of a homemade diet. The recommended supplementations of potassium (increased loss due to diarrhoea and emesis), zinc (benefit mainly based on human studies), iron (in case of marked anaemia due to blood loss) and magnesium (hypomagnesaemia due to fluid therapy and anorexia) were deemed not applicable to this case.[21,22]

OUTCOME AND FOLLOW-UP

Approximately 7 weeks after initiation and before further balancing of the exclusion diet, the patient was once again hospitalised after vomiting for 3 consecutive days. An ultrasound indicated an acute phase of the chronic pancreatitis with an additional cholecystitis/cholangiohepatitis. The patient improved within 24 hours on the same treatment as during previous hospitalisation. Further discussion clarified that the owner fed small quantities of one of the commercial products in Diet B as a treat during the weeks before the episode. Should IBD be indeed involved, intolerance or hypersensitivity to ingredients in this product could have caused this relapse. It was decided to continue the initial treatment plan after the owner agreed to keep to the prescribed diet more strictly. During submission, several cutaneous masses were noted as well. Surgical excision and subsequent histological examination defined these as cutaneous mastocytosis and low-grade mast cell tumours to which Boston terriers seem to be predisposed.[47,48] As gastrointestinal location of mast cell tumours can cause a similar clinical appearance as was noted upon submission,[48] tissue samples of the liver, spleen and lymph nodes were assessed for metastasis. None of the biopsies showed spreading of the tumour.

Further adaptation of the diet was well tolerated, and no hospitalisation was needed for at least 3 months after the relapse. Communication with the owner however revealed that after addition of the last supplement (a tablet to add B vitamins to the diet[21]), the patient had vomited several times; however, was still comfortable and retained its appetite. Following this episode, a water-based vitamin B_{12} solution was recommended as an alternative to the tablet. No further issues were reported thereafter to the Institute of Animal Nutrition and Dietetics.

DISCUSSION

A drawback to this case is that no biopsies were ever taken to confirm the diagnosis of chronic (irreversible histological changes like fibrosis present) opposed to recurrent acute pancreatitis (no irreversible changes present)[6,7] nor to determine cellular infiltrates and the type thereof in the gastrointestinal tract (IBD). In addition, it was not always assessed if blood samples were taken during an acute phase of pancreatitis. Nevertheless, a relatively reliable diagnosis could be made based on the clinical presentation, the patient's age and the appearance on ultrasound.[5,7] The initial dietary adaptation by the Internal Medicine Department of the Vetsuisse Faculty, University of Berne focused on a reduction of fat while increasing the protein and carbohydrate content of the diet. In tailoring the nutritional recommendation by the Institute of Animal Nutrition and Dietetics, the patient's history as well as the dietary approach for both chronic pancreatitis and IBD were considered. As with IBD hypersensitivity should be suspected, an exclusion diet was formulated (initial 8 weeks) and subsequently balanced in 2-week intervals.[21,22,30]

An increase of fat, protein and digestible carbohydrate content of the diet can be debated, as both pancreatitis and IBD are known to decrease the patient's digestive capacity. The chosen approach by the Institute of Animal Nutrition and Dietetics decreased the protein and fat content of the diet, while simultaneously increasing its digestible carbohydrates. This was based on the rationale that

i. both fat and protein can be considered stimuli for pancreatic excretion in contrast to carbohydrates[23,24,28];
ii. both reduced digestibility as well as quantity of protein exceeding the digestive capacity of the small intestine can negatively impact the microbiome[49,50];
iii. as dietary carbohydrates, protein and fat provide energy, one cannot be reduced without increasing another to keep the diet isocaloric;
iv. there were no signs of maldigestion, which is the main reason to reduce digestible carbohydrates[21,22];
v. a lower total quantity of highly digestible protein with a high biologic value (favourable amino acid profile, e.g., meat) can cover essential amino acid requirements;
vi. absorption of protein can improve when IBD is properly addressed.[22]

It should be kept in mind that chronic enteritis and chronic pancreatitis resulting in exocrine pancreatic insufficiency are known to occur with dysbiosis.[42–44] Dysbiosis was however never determined (e.g., no dysbiosis index, no blood folate measurement).[45] Close monitoring of this patient's condition is therefore warranted. If progression is noted, the use of pro-, pre- and/or synbiotics and supplemental taurine (due to potentially increased oxidation thereof in the gut) may be indicated.[21,43,45,51] In addition, supplementation of pancreatic enzymes can significantly improve signs of malabsorption and dysbiosis, should they occur.[46] As these enzymes however form another source of antigens, careful consideration of their administration is warranted when hypersensitivity is suspected.

Furthermore, in case the patient's heart condition worsens, the diet would need appropriate adaptation as well. As ischaemia is a known risk factor for pancreatitis, poor control of the former could render poor control of the latter.[6]

With regard to the possible occurrence of hypersensitivity and due to the presence of protein (most common allergens in dogs with food allergies) in impurified carbohydrate

sources, it must be mentioned that the use of a novel carbohydrate source is recommended as well. The recommendation of a wheat product like bulgur was therefore not optimal. This even more so as the protein fraction of wheat also contains gluten. Due to the relatively low occurrence of gluten intolerance in dogs,[30,52,53] it was nevertheless considered acceptable. Judging from the outcome in this case, food allergy or intolerance are still rather probable. Without provocation tests, it is however impossible to confirm/distinguish the two.[30]

With regard to the patient's follow-up, there were two occasions where the patient once again developed symptoms: one after 7 weeks, one after 20 weeks. As described above, the recurrence of symptoms after 7 weeks was likely caused by noncompliance of the owner. This illustrates why strictly upholding the set plan in these cases is necessary.[30] The relapse after 20 weeks was more complicated as the owner was now strictly following the set guidelines. Intolerance to components in the vitamin tablet is suspected, as the relapse coincided with the administration thereof and no symptoms were seen after changing to a vitamin B solution.[30] It should be mentioned that considering the noted hypocalcaemia, the addition of calcium carbonate from the onset (Diet C_1) could have been considered. As minerals can cause intolerance as well (although low potential)[30] and an improved retention of calcium is anticipated when IBD is properly addressed, a decision against immediate inclusion was taken.

This case report is rather applicable to veterinary medicine, where a final diagnosis often relies on assumptions due to the inability to investigate further. This often renders the necessity to a combined approach. As in this case of recurring pancreatitis, it must thus be considered if there is another pathology present that should be addressed (e.g., IBD). The combined approach as described above makes it however difficult to distinguish which adaptation or combination thereof was ultimately necessary to render a positive result in this patient.

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CONFLICT OF INTEREST

The authors declare they have no conflicts of interest.

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The authors received no specific funding for this work.

ETHICS STATEMENT

This retrospective report is presented with consent of the owner. When it was not hospitalised, the patient remained under the owner's care. Medical treatment was initiated and evaluated at the Internal Medicine Department of the Vetsuisse Faculty, University of Berne. The nutritional evaluation and treatments were initiated as supportive care and according to best practice under the supervision of board-certified members of the ECVCN. The authors confirm that the ethical policies of the journal, as noted on the journal's author guidelines page, have been adhered to. No ethical approval was required as this is an article with no original research data.

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