Journal of Feline Medicine and Surgery (2003) **5**, 271–277
doi:10.1016/S1098-612X(03)00051-2

jfms

Feline pansteatitis revisited: hazards of unbalanced home-made diets

MMRE Niza*, CL Vilela, LMA Ferreira

CIISA-Faculdade de Medicina Veterinária, Pólo Universitário do Alto da Ajuda, Rua Prof. Cid dos Santos, 1300-477 Lisbon, Portugal

Pansteatitis is caused by the consumption of high levels of unsaturated fatty acids and/or the insufficient intake of vitamin E, leading to inflammation of adipose tissue. This disease has been related to fish-based diets. However, non-conventional diets must also be considered. The authors present case records of two cats with pansteatitis, for which diet consisted mostly of pig's brain, comparing them with eight cases of disease in cats eating mainly oily fish.

Cats fed pig's brain did not show clinical signs, while cats eating oily fish presented inappetence, depression, reluctance to move and subcutaneous nodules painful on palpation. Cats eating pig's brain did not show any change in blood parameters, while cats fed oily fish presented leukocytosis and anaemia. Histological examination confirmed pansteatitis in all cats, independently of the diet.

All animals except one of the cats eating oily fish recovered after medical treatment and change of the feeding regime.

Date accepted: 3 June 2003

Introduction

Pansteatitis or yellow fat disease is a nutritional disorder characterised by a marked inflammation of adipose tissue and deposition of a ceroid pigment in fat cells, as a result of vitamin E deficiency. The imbalance can result from an inadequate intake of vitamin E in the diet, from the consumption of high levels of unsaturated fatty acids that deplete the vitamin E, or both (Scott et al 1995). Unsaturated fatty acids present in the lipids of cellular membranes are very vulnerable to oxidative damage. Vitamin E, a known potent antioxidant, interrupts the oxidation of these fats by donating electrons to the free radicals that induce lipid peroxidation. If low amounts of vitamin E are fed, dietary and body fat undergo oxidative degradation, leading to the formation of peroxides and hydroperoxides. The accumulation of reactive peroxides in the cat's adipose tissue results in pansteatitis (Case et al 1995).

All cases described in literature relate pansteatitis with the consumption of fish, mainly oily fish like tuna or sardines (Coffin and Holzworth 1954, Munson et al 1958, Griffiths et al 1960, Summers and Sykes 1982, Hagiwara et al 1986), white fish like coley, herring and cod (Flecknell and Gruffydd-Jones 1978, Tidholm et al 1996) or combinations of sardines, anchovies and mackerel (Koutinas et al 1993).

The main clinical signs of pansteatitis are fever, lethargy, inappetence and pain on palpation of the skin and subcutaneous nodular masses that may show a smooth or a lumpy consistence. In advanced stages of the disease, affected cats lose agility, are unwilling to move and even a gentle touch causes pain (Munson et al 1958). Diagnosis is based on clinical signs and histological examination of biopsies. In gross examination, the adipose tissue shows a dark yellow or orange-brown appearance. Histological examination shows fat necrosis and a septal panniculitis, with a ceroid substance within fat vacuoles and macrophages, probably representing an intermediate product of lipids (White 2000). The diagnosis may be further confirmed by plasma tocopherol levels (<300 µg/100 ml) or erythrocyte membrane stability testing (Kienzle and Hall 1994), although these tests are not frequently performed.

*Corresponding author. Tel: 351-213652800; Fax: 351-213652822. E-mail: necas@fmv.utl.pt

1098-612X/03/050271+07 $30.00/0

The main purpose of this paper is to relate feline pansteatitis with non-conventional diets, discussing the clinical features and aetiology observed in two cats fed mainly pig's brain, in comparison with eight cases of yellow fat disease in cats fed oily fish. The most relevant clinical and laboratory data for an accurate diagnosis are also described.

Materials and methods

This study included 10 cats presented for consultation at the Teaching Hospital of the Veterinary Faculty of Lisbon from 1988 to 1996. The diets of two of these cats consisted mainly of pig's brain, while the remaining eight cats were fed mainly oily fish. The first two cats were both domestic shorthaired females (mother and daughter belonging to the same owner), aged 5 and 3 years; the eight cats fed oily fish were six males and two females, of which one was a Siamese cat, another a Persian cat and all the remaining six were domestic shorthaired cats with ages ranging from 6 months to 1.5 years. The diagnosis was based on clinical signs, macroscopic observation of subcutaneous tissue and haematological findings, and confirmed by biopsy.

Biopsy specimens were fixed in 10% buffered formalin, embedded in paraffin, processed routinely and stained with haematoxylin and eosin.

All cats were treated with α-tocopherol (50 mg/kg once a day, *per os*) for about 30–45 days. Treatment was only discontinued 2 weeks after clinical recovery. Asymptomatic cats were also treated during 45 days and did not receive any further treatment. Cats fed oily fish received also prednisolone, 2 mg/kg daily for 10 days, followed by 2 mg/kg on alternate days for 10 days. One of these cats (case 5) also received enrofloxacin (5 mg/kg daily) and intravenous fluids only for 2 days; however, as there was no noticeable improvement in the clinical state, the owners requested euthanasia. In all cases, diets were changed to commercial cat food.

Results

Distribution of diet, age, sex and breed of clinical cases of pansteatitis in cats submitted to non-conventional diets (n=2) and oily fish-based diets (n=8) are shown in Table 1.

In cases 1 and 2, the two female cats (mother and daughter) submitted to a non-conventional diet based mainly on pig's brain, did not show any abnormal clinical sign leading to pansteatitis suspicion. Yellow fat disease was only suspected when the animals went through a routine surgery (ovariohysterectomy) that revealed a yellow subcutaneous and intra-abdominal fat (Fig 1). Biopsy samples of subcutaneous and intra-abdominal fat were collected for histological examination and pansteatitis was confirmed. Both cats showed a complete recovery from the surgery.

All cats except one (case 5) which diet was based mainly on fish showed similar clinical signs: inappetence, fever, depression, reluctance to move and subcutaneous nodular masses in the abdomen, painful on palpation (Fig 2). Cases 4, 7, 8 and 9 showed generalised hyperaesthesia and they did not allow being touched. Cat 5 was seriously ill and was directed to the hospital for a second opinion. He was prostrated, hypothermic (36.3°C), dehydrated, very thin, in lateral decubitus and it was very painful for him to move. He showed multiple nodules in the abdomen; some of them ruptured with suppurative discharge. The owners said that the animal had been ill for 1 week, and he had worsened in the last 3 days. The cat had been previously medicated with amoxicillin–clavulanic acid (15 mg/kg, q12h, *per os*).

The results of the haematological findings are summarised in Table 2. Cats 1 and 2, fed mainly pig's brain, did not show any abnormality in the haematological parameters. Cats with pansteatitis derived from fish-based diets showed neutrophilic leukocytosis, and cats 3, 4, 9 and 10 also presented normocytic and normochromic anaemia. Cat 5 presented a nonregenerative anaemia. Tests for feline leukaemia virus infection (FeLV) and feline immunodeficiency virus (FIV) were negative for all 10 cats. Radiographic examination was undertaken in cats fed pig's brain (cases 1 and 2) and in four cats fed fish-based diet (cases 4, 5, 9 and 10). No abnormalities were observed in the skeleton of any of these cats.

Histopathological analysis performed on biopsy specimens of subcutaneous tissue collected from all cats confirmed pansteatitis, with part of the normal fat tissue being replaced by interstitial inflammation due to the repair of necrosis. No differences were found between asymptomatic and clinical disease cases. Inflammatory cells were predominantly macrophages, eventually forming multinucleated giant cells. Some of the remaining adipocytes contained ceroid material that had not been removed by solvents, showing up as an amorphous pale yellow material sometimes forming a rim

Table 1. Distribution of diet, age, sex and breed of clinical cases of pansteatitis in cats submitted to non-conventional diets (n=2) and fish-based diets (n=8)

Case no.	Diet	Age	Sex	Breed
Non-conventional diets				
1	Boiled brain (mainly pork) — 90% Commercial cat food — 10%	5 years	Female	Domestic shorthaired (mother)
2	Boiled brain (mainly pork) — 90% Commercial cat food — 10%	3 years	Female	Domestic shorthaired (daughter)
Fish-based diets				
3	Raw fresh sardines — 70% Frozen boiled whiting — 30%	6 months	Male	Domestic shorthaired
4	Frozen or fresh boiled fish of different species — 85% Commercial cat food — 15%	4 months	Female	Siamese
5	Boiled fresh sardines — 30% Boiled frozen whiting — 15% Boiled fresh horse-mackerel — 15% Boiled meat (mainly chicken) — 40%	6 months	Male	Persian
6	Raw fresh sardines — 50% Boiled fresh mackerel — 25% Boiled fresh horse-mackerel — 25%	8 months	Male	Domestic shorthaired
7	Raw fresh sardines — 30% Boiled frozen whiting — 60% Boiled fresh mackerel — 10%	1 year	Male	Domestic shorthaired
8	Raw fresh sardines — 50% Boiled frozen whiting — 50%	8 months	Male	Domestic shorthaired
9	Boiled fresh sardines — 35% Boiled frozen whiting — 35% Boiled meat (turkey, chicken) — 30%	1.5 years	Female	Domestic shorthaired
10	Boiled fresh sardines — 40% Boiled fresh mackerel — 40% Raw meat (beef, chicken) — 20%	9 months	Male	Domestic shorthaired

adherent to the cell membrane (Fig 3). Using special stains this same material was acid fast positive.

All cats except case 5 did not show clinical signs of pansteatitis after medical treatment (α-tocopherol alone for cats 1 and 2, or associated with prednisolone for cats with a fish-based diet) and change of the diet to commercial pet food (Whiskas and/or Iams). Cat 5 also received enrofloxacin, but as there was no improvement after 2 days of treatment euthanasia was request by the owners. Post-mortem examination was not allowed.

Discussion

Pansteatitis has traditionally been associated with diets based on fish. In early reports, pansteatitis was associated with canned fish-based diet (Coffin and Holzworth 1954), mainly canned red tuna (Cordy 1954, Munson et al 1958, Griffiths et al 1960, Summers and Sykes 1982).

However, other diets may also lead to the development of this disease. Watson et al (1973) have reported a case of pansteatitis in a cat fed mainly meat, eating canned fish only once a week. Although the majority of cases of yellow fat disease described in the present study can be attributed to a fish-based diet, two cats submitted to a non-conventional diet developed pansteatitis. Cats 1 and 2 (mother and daughter) were living together, and their diet was based on pig's brain for the last 3 years before being presented to consultation. The daughter started eating that diet immediately after weaning. It is known that pig's brain is very rich in lipids (39.5–42.5%) of which 60% are unsaturated (Ferreira and Graça 1981). Associated with an insufficient intake of vitamin E, this diet may also lead to the formation of reactive peroxides, resulting in the development of pansteatitis. To our knowledge, this is the first time that a non-conventional diet, brain-based, is assigned as a putative cause of pansteatitis.

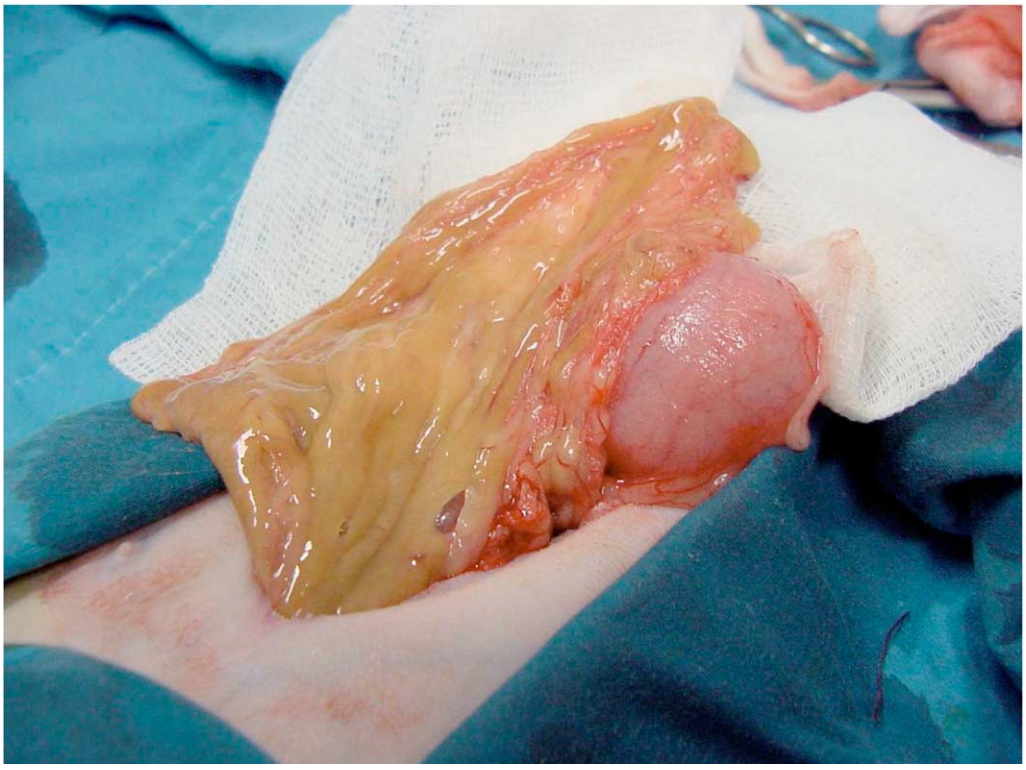

Fig 1. Cat with asymptomatic pansteatitis (case 1). A strong yellow-orange colouration of the mesenteric fat, indicative of yellow fat disease, is patent, but no other lesions were observed.

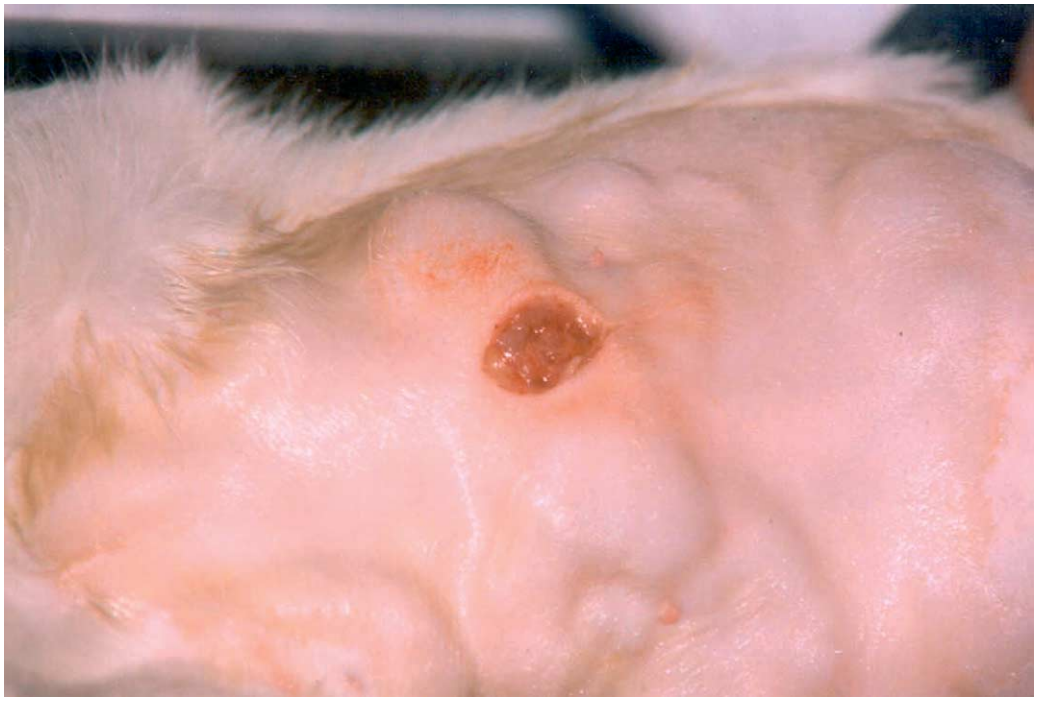

Fig 2. Cat with pansteatitis (case 8), showing subcutaneous nodular masses in the abdomen. Subcutaneous adipose tissue with orange-brown colour, characteristic of feline pansteatitis, is evident.

Table 2. Haematological profile of cats with pansteatitis due to non-conventional diets (n=2) and fish-based diets (n=8)

Haematological profile	Range (mean)			Reference intervals
	Non-conventional brain-based diet (n=2)	Fish-based diet (n=8)	Total (n=10)	
Leukocytes (10^3/µl)	7.19, 18.5 (12.85)	20.9–31.23 (25.06)	7.19–31.23 (22.80)	5.5–19.5
Erythrocytes (10^6/µl)	7.11, 7.99 (7.55)	2.38–8.98 (5.87)	2.38–8.98 (6.04)	5–10
Platelets (10^3/µl)	201, 420 (310.5)	150–468 (247.86)	150–468 (255.2)	150–400
Haemoglobin concentration (g/dl)	11.8, 12.8 (12.3)	4.7–15.2 (8.85)	4.7–15.2 (9.32)	8–15
Packed cell volume (%)	36, 39.4 (37.7)	14.1–42.9 (27.57)	14.1–42.9 (28.79)	30–45
Mean cell volume (fl)	49.3, 50.6 (49.95)	42–59.1 (47.94)	42–59.1 (48.28)	39–55
Mean corpuscular haemoglobin concentration (g/dl)	32.5, 38.8 (35.65)	27–35.5 (31.61)	27–38.8 (32.66)	30–36
Segmented neutrophils	4961, 13 505 (9233)	17 886–27 170 (21 261)	4961–27 170 (18 885)	2500–12 500
Band neutrophils	0, 71 (36)	0–679 (248)	0–679 (181)	0–300
Lymphocytes	1726, 3700 (273)	993–4312 (2611)	993–4312 (2801)	1500–7000
Monocytes	288, 370 (329)	0–1078 (418)	0–1078 (466)	0–850
Eosinophils	144, 925 (535)	0–1298 (519)	0–1298 (470)	0–1500
Basophils	0	0	0	Rare

Cats fed a pig's brain-based diet did not present any clinical signs of pansteatitis. The disease was suspected during a routine surgery (ovariohysterectomy), when the incision of the skin showed yellow subcutaneous tissue and intra-abdominal fat, suggestive of the yellow fat disease. Diagnosis was confirmed by histopathological examination. Tidholm et al (1996) have referred to the possibility of absence of clinical signs and accidental diagnosis of pansteatitis in a cat. These situations can represent an initial phase of pansteatitis, as suggested by Tidholm et al (1996), or can be attributed to variation in individual susceptibility to the disease, as referred to by Flecknell and Gruffydd-Jones (1978). In a study conducted by Cordy (1954), only two of the nine cats with experimentally induced pansteatitis showed clinical signs. However, four animals showed histological lesions compatible with the disease (three severe and one moderate). In the present study, it remains to be established if the pansteatitis without clinical signs, developed by cats 1 and 2, fed a diet based on pig's brain, was due to the stage of development of the disease, to different susceptibility of these cats to pansteatitis, or to the diet itself.

The age of the two cats fed a non-conventional diet (3 and 5 years) was higher than the age of the cats fed a fish-based diet reported in our study (ranging from 4 months to 1.5 years), and described by Koutinas et al (1993). Young animals are more prone to the disease but it can develop in older cats, as mentioned by Griffiths et al (1960).

The clinical signs observed in the other cats, fed a fish-based diet (except cat 5) were similar to the ones described by others (Koutinas et al 1993, Tidholm et al 1996). Cat 5 was present to consultation in very poor physical condition, very thin, with hypothermia, dehydration and showing ruptured abdominal nodules with a purulent exudate. The two cats fed a brain-based diet did not show any abnormal haematological parameter. On the contrary, the eight cats fed a fish-based diet had neutrophilic leukocytosis and four of them also showed normocytic and normochromic anaemia, common clinical findings in feline pansteatitis (Koutinas et al 1993). This leukocytosis is caused by necrosis of the adipose tissue (Hagiwara et al 1986).

The proposed therapy for pansteatitis consists in the administration of α-tocopherol, corticosteroids and dietary changes (Case et al 1995, White 2000). There is a wide range of recommended doses of vitamin E. Scott et al (1995) propose 13.5 IU/kg/day, while White (2000) prescribes 25–75 IU every 12 h. In the literature review, the starting point of corticosteroid therapy is variable. Some authors begun corticosteroid therapy concomitantly with vitamin E (Flecknell and Gruffydd-Jones 1978, Koutinas et al 1993), while others used corticosteroids only in severe cases (Griffiths et al 1960) or later in the course of the

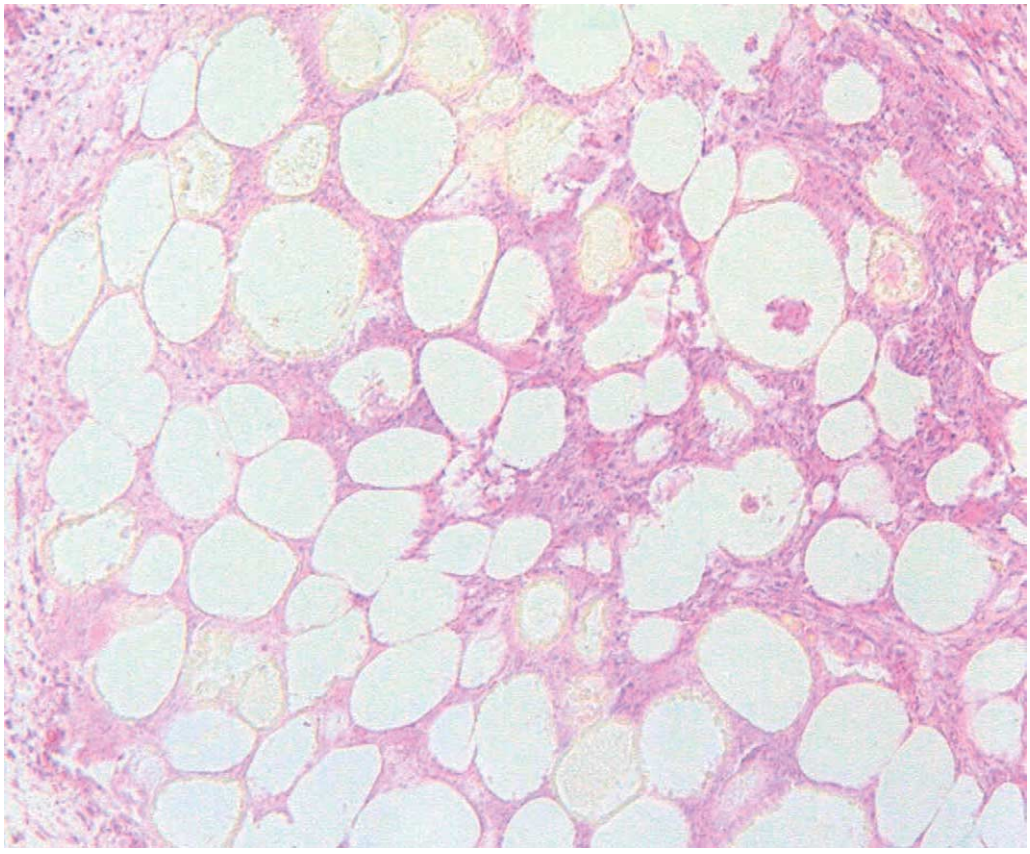

Fig 3. Interstitial inflammation of the fat tissue in a case of feline pansteatitis. Ceroid can be identified within some adipocytes as an amorphous pale yellow material sometimes forming a rim adherent to the cell membrane (H and E ×40).

disease, due to delayed diagnosis of pansteatitis (Hagiwara et al 1986).

The therapy prescribed for all the cats in our study was α-tocopherol, at the dosage of 50 mg/kg once a day, *per os*, for about 30–45 days; the treatment was only discontinued 2 weeks after clinical recovery. Cats 1 and 2, fed the non-conventional diet, did not receive any further medication. Cats fed a fish-based diet, present to consultation with clinical signs of pansteatitis, also received prednisolone, 2 mg/kg daily for 10 days, followed by 2 mg/kg on alternate days for 10 days. In all cases, diets were changed to commercial cat foods (Whiskas and/or Iams). All the animals in the present study that had clinical signs of panstetatis completely recovered from the disease, with the exception of case 5, which was severely compromised. Others (Flecknell and Gruffydd-Jones 1978, Koutinas et al 1993) have also reported a positive therapeutic response. However, some cats may not respond satisfactorily to the treatment or may have a fatal relapse after a brief period of clinical improvement (Watson et al 1973, Hagiwara et al 1986, Tidholm et al 1996). The therapy failure in these cases may be due to late diagnosis or to a special sensitivity of some animals to pansteatitis.

Regarding the two cases of asymptomatic pansteatitis, it was not possible to ascertain the efficiency of the therapeutic measures. The only possibility of confirming the therapeutic success would be through fat biopsy. However, the owners were not receptive to this suggestion, and no further attempts were made, as an invasive procedure was not required. Both cats lived without any clinical signs of pansteatitis until the age of 13.5 and 14 years, when they died in consequence of chronic renal failure.

Although pansteatitis is not a frequent disease, diagnosis must be established as soon as possible, as the success of therapy is diminished by the delay (Koutinas et al 1993, Tidholm et al 1996). Similar to the majority of the nutritional diseases, pansteatitis can be easily avoided. Nowadays, due to an increase of living standards and to a better awareness of the owners, most of the cats are fed commercial diets instead of home-prepared fishmeals. In the past, some cat

foods were prepared with fishmeal and fish oil without added antioxidants, leading to the development of pansteatitis. Presently pet foods contain antioxidants, namely α-tocopherol and α-tocopherol acetate (Gross et al 2000).

The cases of pansteatitis in cats fed fish-based diets, mainly oily fish, are becoming scarcer. However, non-traditional diets like the one reported here may also lead to the development of yellow fat disease. Due to the known risks of transmissible spongiform encephalopathies (TSEs), it is not likely that many owners feed their pets with brain. However, other non-conventional diets, especially the ones rich in unsaturated fatty acids, must be carefully considered as they may represent a potential risk to the development of pansteatitis.

Acknowledgements

The authors would like to thank Prof. C. Peleteiro for her contribution with histologic studies and Profs A. Ferreira and K. Papassouliotis for critical reading of the manuscript.

References

Case LP, Carey DP, Hirakawa DA (1995) Vitamin deficiencies and excesses. In: *Canine and Feline Nutrition. A Resource for Companion Animal Professionals*. St. Louis: Mosby-Year Book, pp. 303–308.

Coffin DL, Holzworth J (1954) 'Yellow fat' in two laboratory cats: acid-fast pigmentation associated with a fish-base ration. *Cornell Veterinarian* **44**, 63–71.

Cordy DR (1954) Experimental production of steatitis (yellow fat disease) in kittens fed a commercial canned cat food and prevention of the condition by vitamin E. *Cornell Veterinarian* **44**, 310–318.

Ferreira GFA, Graça MES (1981) Tabela de Composição dos Alimentos Portugueses. Ed Direcção Geral de Saúde. Instituto Superior de Higiene Dr. Ricardo Jorge. Lisboa/Porto, pp. 225–226.

Flecknell PA, Gruffydd-Jones TJ (1978) Pansteatitis in the cat. *Veterinary Record* **18**, 149.

Griffiths RC, Thornton GW, Wilson JE (1960) Pansteatitis ('yellow fat') in cats. *Journal of the American Veterinary Medical Association* **137**, 126–128.

Gross KL, Wedekind KJ, Cowell CS, Schoenherr WD, Jewell DE, Zicker SC, Debraekeleer J, Frey RA (2000) Nutrients. In: Hand MS, Thatcher CD, Remillard RL, Roudebush P (eds), *Small Animal Clinical Nutrition* (4th edn). St. Louis: Mark Morris Institute, Kansas, pp. 21–107.

Hagiwara MK, Guerra JL, Maeoka MRM (1986) Pansteatitis (yellow fat disease) in a cat. *Feline Practice* **16**, 25–27.

Kienzle E, Hall DK (1994) Inappropriate feeding: the importance of a balanced diet. In: Wills JM, Simpson KW (eds), *The Waltham Book of Clinical Nutrition of the Dog and Cat*. Oxford: Pergamon, pp. 1–14.

Koutinas AF, Miller WH, Kritsepi M, Lekkas S (1993) Pansteatitis (steatitis, 'yellow fat disease') in the cat: a review article and report of four spontaneous cases. *Veterinary Dermatology* **3**, 101–106.

Munson TO, Holzworth J, Small E, Witzel S, Jones TC, Luginbuhl H (1958) Steatitis ('yellow fat') in cats fed canned red tuna. *Journal of the American Veterinary Medical Association* **133**, 563–568.

Scott DW, Miller WH Jr, Griffin CE (1995) Nutritional skin diseases. In: *Muller and Kirk's Small Animal Dermatology* (5th edn). Philadelphia: WB Saunders, pp. 890–901.

Summers BA, Sykes G (1982) Pansteatitis mimicking infectious peritonitis in a cat. *Journal of the American Veterinary Medical Association* **180**, 546–549.

Tidholm A, Karlsson I, Wallius B (1996) Feline pansteatitis: a report of five cases. *Acta Veterinaria Scandinavica* **37**, 213–217.

Watson ADJ, Porges WL, Huxtable CR, Ilkiw WJ (1973) Pansteatitis in a cat. *Australian Veterinary Journal* **49**, 388–392.

White SD (2000) The skin as a sensor of internal medical disorders. In: Ettinger SJ, Feldman EC (eds), *Textbook of Veterinary Internal Medicine* (5th edn). Philadelphia: WB Saunders, pp. 26–29.