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Serum Triglyceride Concentrations in Miniature Schnauzers with and without a History of Probable Pancreatitis

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Background: The association between hypertriglyceridemia and pancreatitis remains obscure in dogs. A possible role of hypertriglyceridemia as a cause of pancreatitis in Miniature Schnauzers has been suspected.

Hypothesis/Objectives: To compare serum triglyceride concentrations between Miniature Schnauzers with and without a recent history of pancreatitis.

Animals: Seventeen Miniature Schnauzers with a history of pancreatitis (group 1) and 34 age-matched Miniature Schnauzers without a history of pancreatitis (group 2) were prospectively enrolled.

Methods: Prospective case-control study. Two samples were collected from each of the 17 Miniature Schnauzers with pancreatitis: 1 during pancreatitis and 1 after clinical and biochemical resolution of pancreatitis. Serum triglyceride and cholesterol concentrations were compared between group 1 (after resolution of pancreatitis) and group 2.

Results: Miniature Schnauzers in group 1 were significantly more likely to have hypertriglyceridemia (>108 mg/dL) (71%) after resolution of pancreatitis than Miniature Schnauzers in group 2 (33%; odds ratio = 5.02; 95% confidence interval = 1.4–17.8; $P = .0163$). Serum triglyceride concentrations were significantly higher in dogs of group 1 (median: 605.0 mg/dL) after resolution of pancreatitis than in dogs of group 2 (median: 73.5 mg/dL; $P = .002$).

Conclusions and Clinical Importance: Miniature Schnauzers with a history of pancreatitis were 5 times more likely to have hypertriglyceridemia than controls. Hypertriglyceridemia might be associated with the development of pancreatitis in some dogs of this breed. Additional studies are needed to further clarify the role of hypertriglyceridemia in the development of pancreatitis in Miniature Schnauzers as well as other dog breeds.

Key words: Dog; Hyperlipidemia; Hypertriglyceridemia; Pancreas.

The cause of pancreatitis usually remains unknown in dogs.[1,2] Several risk factors for pancreatitis have been identified or suspected in dogs, including trauma, certain drugs, endocrine diseases, obesity, and dietary indiscretion, but a definitive cause and effect relationship has not been established for most of them.[2–4] Hypertriglyceridemia has long been considered a possible cause of or risk factor for pancreatitis in dogs.[2,3,5–7] Furthermore, it is widely believed that a suspected high prevalence of pancreatitis in Miniature Schnauzers[3,4] might be associated with the documented high prevalence of hypertriglyceridemia in this breed.[5–10]

The association between hypertriglyceridemia and pancreatitis remains obscure in dogs. Hypertriglyceridemia is commonly reported in dogs with pancreatitis, but the etiology of hypertriglyceridemia is not always possible to determine in these cases.[2,3,5,11,12] The presence of hypertriglyceridemia in dogs with pancreatitis might be because of a pre-existing disorder in lipid metabolism, which may or may not be related to the etiology of pancreatitis, or it might be the result of pancreatitis.[7] Although it is widely believed that hypertriglyceridemia can develop as a result of pancreatitis, this has not been convincingly shown in dogs with naturally occurring pancreatitis. Furthermore, with the exception of the results of an older study,[13] hypertriglyceridemia does not seem to be a consequence of experimental pancreatitis in dogs.[5,14,15] In order for hypertriglyceridemia to be considered as a possible etiologic factor for pancreatitis, it should precede the development of pancreatitis. Hypertriglyceridemia that precedes the development of pancreatitis might be primary (for example hypertriglyceridemia of certain breeds such as Miniature Schnauzers) or secondary to other diseases including diabetes mellitus, hyperadrenocorticism, hypothyroidism, or drug administration (glucocorticoids).[7] A possible role of hypertriglyceridemia as a cause of pancreatitis in Miniature Schnauzers or other dog breeds has been suspected based on knowledge extrapolated from human medicine, where severe hypertriglyceridemia is a well recognized cause of pancreatitis,[16–19] in vitro or experimental animal studies,[20,21] and anecdotal clinical impressions.[6]

The possibility of hypertriglyceridemia potentially developing as a result of pancreatitis complicates the determination of the role of hypertriglyceridemia as an etiologic factor of pancreatitis in both humans and dogs.[7,16] One approach that is often used in human studies in order to overcome this obstacle is the measurement of serum triglyceride concentrations after pancreatitis has resolved.[19,22–25] This is based on the logical assumption that a defect in lipid metabolism causing hypertriglyceridemia must be a pre-existing condition in order for a patient to develop pancreatitis as a result of hypertriglyceridemia. If this is the case, hypertriglyceridemia should also persist after resolution of pancreatitis.[19,22–25] In contrast, hypertriglyceridemia secondary to pancreatitis should resolve when pancreatitis is no longer present.

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Therefore, the presence of hypertriglyceridemia soon after resolution of pancreatitis provides retrospective evidence of a pre-existing condition, which if severe enough, could be considered as an etiologic factor for pancreatitis.[19,22–25]

Studies investigating disorders of lipid metabolism in patients with a history of naturally occurring pancreatitis have been reported in humans but not in veterinary species. The aim of this study was to compare serum triglyceride concentrations between Miniature Schnauzers with a recent history of pancreatitis and those without such a recent history of pancreatitis.

Materials and Methods

Study Design

This study is a prospective case-control study.

Dogs with Pancreatitis (Group 1)

Over a 2-year period (November 2006–November 2008), a total of 35 Miniature Schnauzers with a diagnosis of pancreatitis were initially considered for prospective enrollment into this study. The diagnosis of pancreatitis was based on the presence of at least 2 clinical signs compatible with pancreatitis (vomiting, anorexia, depression, abdominal pain, diarrhea) and a serum Spec cPL concentration $\geq$400 μg/L (the currently recommended cut-off value for pancreatitis).[a,b,26] The dogs were enrolled on a sequential basis based solely on whether the owner had agreed to submit a follow-up sample after clinical resolution of pancreatitis. The 35 dogs considered for enrollment into the study were from various regions of the United States.

After clinical recovery from pancreatitis, the owners of these dogs were asked to have a follow-up blood sample collected. The person communicating with the owners concerning the request for a follow-up sample was blinded to the serum triglyceride concentration of the dog during the initial evaluation. Thus, no preference was given to dogs with hypertriglyceridemia. The dogs were eventually included in the study if at the time the follow-up sample was collected they had (a) complete absence of any clinical signs, (b) a serum Spec cPL concentration within the reference interval ($<$200 μg/L),[26] (c) a serum free T4 or total T4 concentration within the reference interval, and (d) no history of diseases or use of drugs known to affect lipid metabolism (other than pancreatitis). Dogs fulfilling these enrollment criteria were designated as group 1 (dogs with clinically resolved pancreatitis and a serum Spec cPL concentration within the reference interval). Only the follow-up blood sample, which was obtained after clinical and biochemical resolution of pancreatitis, was used for statistical comparison with the control group. The initial sample (during pancreatitis) was excluded from the analysis because it would not have been possible to determine whether possible hypertriglyceridemia in this initial sample was primary or had developed secondary to pancreatitis. For the same reason, serum Spec cPL concentration was measured in the follow-up sample of each dog, and dogs with serum Spec cPL concentrations outside the reference interval (ie, $>$200 μg/L) were excluded from further analysis. Serum triglyceride and cholesterol concentrations were evaluated in both the initial (during pancreatitis) and the follow-up sample for all dogs in group 1.

Control Dogs (Group 2)

Each dog enrolled into group 1 was age-matched with 2 clinically healthy Miniature Schnauzers with no history of pancreatitis (group 2). The selection of dogs into this group was based solely on their age (which had to match the age of each 1 of the dogs in group 1). The person selecting the dogs was blinded to the serum triglyceride concentration of the dog. Inclusion criteria for dogs in group 2 were identical to those for dogs of group 1, with the exception of the history of pancreatitis. Therefore, dogs in group 2 were included in the study if they had (a) absence of any clinical signs at the time of blood collection, (b) a serum Spec cPL concentration within the reference interval, (c) a serum free T4 or total T4 concentration within then reference interval, and (d) no history of diseases or current drug use known to affect lipid metabolism. Serum triglyceride and cholesterol concentrations were measured in all dogs of group 2.

Blood Collection and Handling

Owners who decided to participate in the study were sent styrofoam boxes containing ice packs and the material necessary for blood collection, and were asked to schedule an appointment with their veterinarian. Veterinarians were instructed to collect 10 mL of blood into a red-top tube (with no additive), wait for clot formation, centrifuge the sample, separate the serum from the clot, transfer the serum to another red-top tube, and send the samples to the Gastrointestinal Laboratory packed on ice by overnight courier. Owners had been instructed not to feed their dogs for at least 12 hours before the scheduled blood collection. Upon receipt, serum samples were immediately aliquoted and stored at −80°C until further use.

Questionnaires and Consent Forms

Owners were asked to complete a questionnaire for each dog. Questions covered date of birth, sex, body weight, current diet(s), current medications, and current and past health history of the dogs. Questionnaires from all dogs were reviewed to determine whether the dogs fit the inclusion criteria for each group. All owners had to sign an informed owner consent form. The study protocol was reviewed and approved by the Clinical Research Review Committee at Texas A&M University.

Assays

Serum triglyceride (reference interval: 26–108 mg/dL) and cholesterol (reference interval: 124–335 mg/dL) concentrations were measured by enzymatic assays.[c] Serum Spec cPL concentrations were measured using an analytically validated immunoassay as described elsewhere.[26] Serum free T4 concentration was measured with a commercial equilibrium dialysis radioimmunoassay.[d] Serum total T4 concentrations were measured by a solid-phase chemiluminescent competitive assay.[e]

Statistical Analyses

A commercial statistical software package was used for all statistical analyses.[f] Data were analyzed for normal distribution by the Shapiro-Wilk test. Normally distributed data were analyzed by *t*-tests or paired *t*-tests where appropriate. Not normally distributed data were analyzed by the Wilcoxon test or Wilcoxon rank sum test (for paired data). Proportions were compared between groups by Fisher's exact test. Significance was set at $P < .05$ for all analyses.

Results

Dogs in Groups 1 and 2

All dogs were prospectively enrolled into this study. Of the 35 dogs initially considered for enrollment in group 1, 17 met the inclusion criteria for this group. Five dogs were excluded because they had clinical signs of anorexia, diarrhea, and/or abdominal pain at the time of the 2nd

blood collection, 4 dogs were excluded because they had been diagnosed with diabetes mellitus (2) or hypothyroidism (2), and 9 dogs were excluded because of a Spec cPL concentration in the follow-up sample above 200 μg/L (3 of these dogs also had clinical signs associated with pancreatitis). Follow-up samples from the 17 Miniature Schnauzers that were included in group 1 were collected within a median interval of 12 weeks (range: 2–41 weeks) after the 1st sample collected at the time of diagnosis of pancreatitis. Group 1 consisted of 9 female (8 spayed) and 8 male (6 castrated) dogs.

Group 2 consisted of 34 Miniature Schnauzers that had fulfilled the inclusion criteria outlined above and had been age-matched with the dogs in group 1. Group 2 consisted of 22 female (8 spayed) and 12 male (3 castrated) dogs.

Comparisons between Groups 1 and 2

All comparisons between groups 1 and 2 were made using the information and test results that corresponded to the follow-up sample (which was collected after clinical and biochemical resolution of pancreatitis) for group 1. The weight was known for 16/17 dogs in group 1 and 30/34 dogs in group 2; there was no significant difference of the mean body weight between group 1 (7.8 kg) and group 2 (7.6; $P = .6702$). As outlined in the study's inclusion criteria, all dogs had a serum free T4 (15 dogs in group 1 and 32 dogs in group 2) or total T4 (2 dogs in each group) within the respective reference interval. Of the dogs in group 1, 8/16 (50.0%) were mainly on a home-made (n = 1) or commercial (n = 7) diets labeled as low fat, including prescription (n = 5) and nonprescription (n = 2) diets. Of the 34 dogs in group 2, 1 was mainly on a home-made low fat diet, none were on prescription low fat diets at the time of blood collection, while 4 dogs (11.8%) were on commercial diets labeled as low fat. Dogs in group 1 were significantly more likely to be on a low fat diet than dogs in group 2 (OR = 5.8, 95% CI = 1.5–22.7, $P = .0143$).

Of the 17 Miniature Schnauzers in group 1, 12 (71%) had serum triglyceride concentrations above the reference interval in the follow-up sample, while only 11 (33%) of the 34 Miniature Schnauzers in group 2 had serum triglyceride concentrations above the upper limit of the reference interval. Miniature Schnauzers in group 1 were significantly more likely to have hypertriglyceridemia than Miniature Schnauzers in group 2 (OR = 5.02; 95% CI = 1.4–17.8; $P = .0163$). To take into account the severity of hypertriglyceridemia, the proportion of dogs with moderate to severe hypertriglyceridemia (> 500 mg/dL)[7,18] was compared between groups. Ten out of 17 (59%) dogs in group 1 and 3/34 (9%) in group 2 had serum triglyceride concentrations above 500 mg/dL (OR = 14.8; 95% CI = 3.2–68.1; $P = .0003$). Serum triglyceride concentrations were significantly higher in dogs of group 1 (median: 605.0 mg/dL; range: 41.0–2,134.0 mg/dL) than in dogs of group 2 (median: 73.0 mg/dL; range: 23.0–3,975.0; $P = .002$; [Fig 1]).

Serum cholesterol concentrations were significantly higher in dogs of group 1 (median: 287.6 mg/dL) than in those of group 2 (median: 204.0 mg/dL; $P = .0001$; [Fig 2]). Five dogs in group 1 (29.4%) and 1 dog in group 2 (2.9%; $P = .0136$) had serum cholesterol concentrations above the upper limit of the reference interval, and hypercholesterolemia was associated with hypertriglyceridemia in all cases.

Comparisons within Group 1

Within group 1, serum triglyceride concentrations were not significantly different during (median: 215.5 mg/dL) and after resolution of pancreatitis (median: 580.0 mg/dL; $P = .552$). However, great variation of values within the same dog existed between the 2 time-points for many of the dogs (Fig 3). Interestingly, serum triglyceride concentrations were normal or below the lower limit of the reference interval in 3 of the dogs during pancreatitis (20, 51, and 85 md/dL) and increased (429, 2,134, and 1,851 mg/dL, respectively) after resolution of pancreatitis (Fig 3). These dogs were each on the same diet (n = 2) or on diets with a similar fat content (n = 1) at both time-points.

In the same group, mean serum cholesterol concentrations were not significantly different during (251.7 mg/dL) and after resolution of pancreatitis (272.7 mg/dL; $P = .3523$).

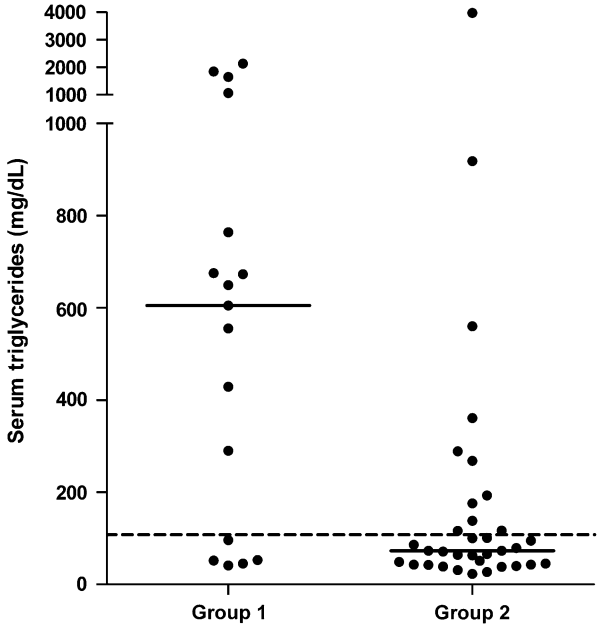

Fig 1. Comparison of serum triglyceride concentrations between dogs of groups 1 and 2. This graph shows the distribution and comparison of serum triglyceride concentrations between dogs of groups 1 and 2. Serum triglyceride concentrations were significantly higher in dogs of group 1 (median: 605.0 mg/dL) than those in dogs of group 2 (median: 73.0 mg/dL; $P = .002$). The dashed line represents the upper limit of the reference interval. Note: the Y-axis in split.

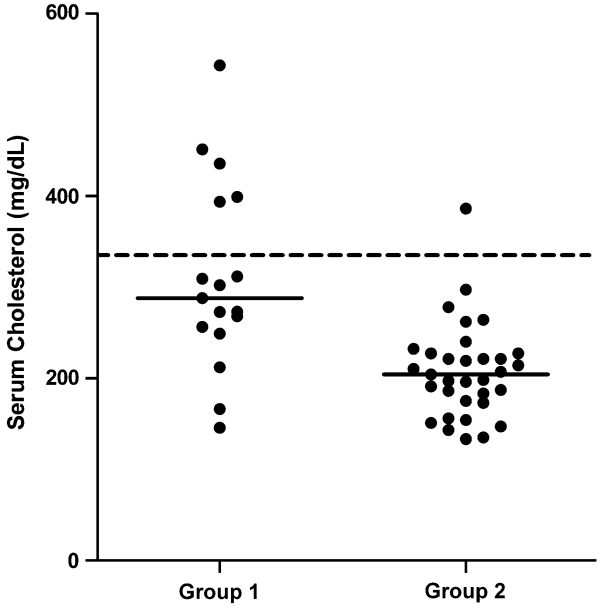

Fig. 2. Comparison of serum cholesterol concentrations between dogs of groups 1 and 2. This graph shows the distribution and comparison of serum cholesterol concentrations between dogs of groups 1 and 2. Serum cholesterol concentrations were significantly higher in dogs of group 1 (median: 287.6 mg/dL) than those in group 2 (median: 20.0 mg/dL; $P = .0001$). Only 5 dogs in group 1 and 1 dog in group 2 had serum cholesterol concentrations above the upper limit of the reference interval. The dashed line represents the upper limit of the reference interval.

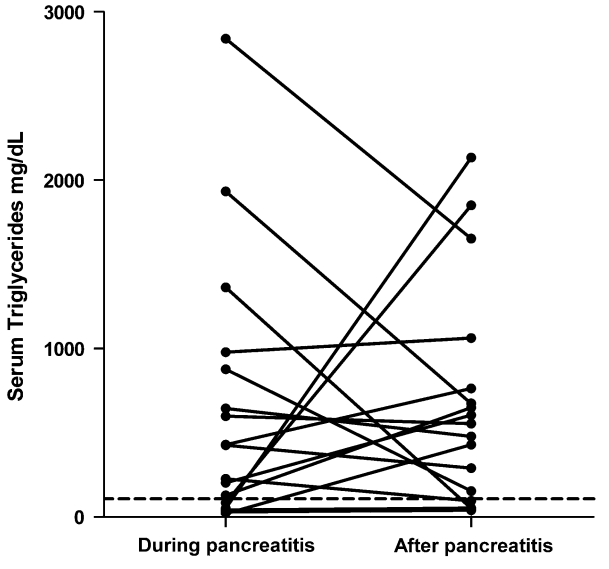

Fig. 3. Comparison of serum triglyceride concentrations during pancreatitis and after resolution of pancreatitis in dogs of group 1. This graph shows the distribution and comparison of serum triglyceride concentrations in dogs of group 1 at the time of diagnosis of pancreatitis and also after clinical and biochemical resolution of pancreatitis. Serum triglyceride concentrations were not significantly different at the time of diagnosis of pancreatitis (median: 215.5 mg/dL) than after resolution of pancreatitis (median: 580.0 mg/dL; $P = .552$). For many of the dogs, great variation of serum triglyceride concentrations existed between the 2 time-points. The dashed line represents the upper limit of the reference interval.

Discussion

In the present study, Miniature Schnauzers with a history of pancreatitis were 5 times more likely to have hypertriglyceridemia and almost 15 times more likely to have moderate to severe hypertriglyceridemia (defined as >500 mg/dL) than age-matched Miniature Schnauzers with no history of pancreatitis. The majority of Miniature Schnauzers with a history of pancreatitis (59%) had moderate to severe hypertriglyceridemia as compared with only 9% in the control group. The presence of hypertriglyceridemia after resolution of pancreatitis is considered to provide retrospective evidence of a pre-existing disorder in lipid metabolism.[19,22,23,25] Knowing whether severe hypertriglyceridemia is a risk factor or even a cause of pancreatitis is of major importance in dogs, because control of hypertriglyceridemia might prevent the development or recurrence of pancreatitis. This has been confirmed in human patients with severe hypertriglyceridemia and pancreatitis, in whom control of hypertriglyceridemia limits the risk of recurrence of pancreatitis.[19,27] People with uncontrolled severe hypertriglyceridemia commonly have recurrent pancreatitis.[18,27]

It remains to be determined whether pre-existing hypertriglyceridemia in this group of Miniature Schnauzers predisposed them to develop pancreatitis. A clear causal association between hypertriglyceridemia and pancreatitis in Miniature Schnauzers is very challenging to illustrate in clinical studies. Ideally, samples before, during, and after resolution of pancreatitis should be collected in order to clearly determine the association between hypertriglyceridemia and pancreatitis. Although this is possible to do in experimental models of pancreatitis, it is impossible in cases of naturally occurring pancreatitis as it cannot be predicted which dogs will develop pancreatitis. Therefore, evidence of pre-existing hypertriglyceridemia in this study was based on the presence of hypertriglyceridemia after recovery from pancreatitis. Most follow-up samples were collected soon after pancreatitis was diagnosed (within 1–4 months) and thus it is very likely that hypertriglyceridemia was present before the development of pancreatitis. This has been shown in a large number of human studies, in which disorders of lipid metabolism that persist after recovery from pancreatitis were considered to be pre-existing.[19,22,23,25] Because of the fact that follow-up samples in group 1 were collected after resolution of pancreatitis (based on both clinical and biochemical evidence), it is unlikely that hypertriglyceridemia in these samples was the result of pancreatitis. There is a theoretical possibility that pancreatitis could have permanently changed lipid metabolism in those dogs but, to the authors' knowledge, this has not been reported or even hypothesized in dogs or any other species.

It needs to be pointed out that, in this study, Miniature Schnauzers with a history of pancreatitis were significantly more likely to be on diets labeled as low fat than dogs of the same breed without such a history. This is very important because the reported serum triglyceride concentrations after recovery from pancreatitis likely

underestimate the true prevalence and severity of serum triglyceride concentrations before the development of pancreatitis. Despite that, Miniature Schnauzers with a history of pancreatitis were significantly more likely to have hypertriglyceridemia than age-matched Miniature Schnauzers with no history of pancreatitis. It is very important to note, however, that the diets were considered to be low fat only based on their label and not their actual fat content. Although this is a rather inaccurate way to determine the fat content of a diet, such an approach was chosen because several dogs were on home-made diets, while other dogs were on more than 1 diet at the same time, and it would be impossible to determine that fat content of the diet for each dog.

Many dogs in the control group had hypertriglyceridemia and yet no known history of pancreatitis. This is also commonly reported in humans, and the reason for this is unknown.[16,17] There are several possible explanations for this observation. First, as mentioned above, a causal association between hypertriglyceridemia and pancreatitis in dogs remains to be determined. If a causal association exists, it is possible that there are differences in lipoprotein composition or metabolism between individuals, which may or may not predispose to pancreatitis. In addition, individual cases of mild pancreatitis might have escaped diagnosis, or some of these patients might develop pancreatitis in the future. It should also be pointed out that in the case of the present study, most hypertriglyceridemic dogs (91%) in the control group had mild (< 500 mg/dL) hypertriglyceridemia, which might be less likely to be associated with pancreatitis. Several studies in humans have evaluated various methods to predict which hypertriglyceridemic individuals will develop pancreatitis but results of these studies are inconclusive.[22,23,25] Such studies have not been reported, but based on the results reported here, are clearly warranted in dogs, in order to provide a better understanding of the possible lipid disorders associated with pancreatitis.

Great variation in serum triglyceride concentrations existed within some individual dogs in group 1 between the 2 time-points (during and after resolution of pancreatitis). Although these results must be interpreted with caution because dietary information was not available for all dogs during pancreatitis and because the diet was different between the 2 time-points for some of the dogs, an interesting observation can be made. Serum triglyceride concentrations were normal or below the lower limit of the reference interval in 3 of the dogs during pancreatitis (20, 51, and 85 md/dL) and increased or even dramatically increased (429, 2,134, and 1,851 mg/dL, respectively) after resolution of pancreatitis (Fig 3). These dogs were each on the same diet (n = 2) or on diets with a similar fat content (n = 1) both time-points. Although the reason for this finding is unknown, it might be that extensive anorexia (all 3 dogs were reported to be anorexic) and/or fasting during pancreatitis led to normalization of serum triglyceride concentrations in these dogs during this time-period. Similar findings have been reported in humans with pancreatitis, where even profound increases in serum triglyceride concentrations (> 1,750 mg/dL) returned to normal within 72 hour of initial presentation.[19,24,25] On the other hand, 1 dog that was on the same diet at both time-points had severely increased serum triglyceride concentrations during pancreatitis (1,363 mg/dL) and normal serum triglyceride concentrations after resolution of pancreatitis (52 mg/dL). Regardless of the possible explanation, these findings suggest that serum triglyceride concentrations during the course of pancreatitis may not accurately reflect the true triglyceride concentrations.[16,17,19] Therefore, it might be recommended that serum triglyceride concentrations be measured after resolution of pancreatitis to make sure that the measurements accurately reflect the condition of each dog.

Although serum cholesterol concentrations were significantly higher in Miniature Schnauzers with a history of pancreatitis compared with controls, the medians of both groups were within the reference interval. In addition, only 5 dogs in group 1 had hypercholesterolemia, which was always mild and seen only in association with hypertriglyceridemia. This is in agreement with the results of a previous study in healthy Miniature Schnauzers.[10] To the authors' knowledge, there is no evidence to suggest that hypercholesterolemia might play a role in the development of pancreatitis in dogs or any other species, and this is supported by the findings of the present study.

Because of the fact that the dogs in this study had normal serum free or total T4 concentrations, no clinical signs, and no history of a disease or drug administration known to affect lipid metabolism, it is logical to assume that hypertriglyceridemia in these dogs was likely idiopathic.[7,10] Although an association between endocrinopathies commonly accompanied by secondary hypertriglyceridemia and pancreatitis has been reported previously, the retrospective design of these studies does not allow determination of whether this was because of hypertriglyceridemia or not.[2,12] Further studies are needed to determine what proportion of dogs with pancreatitis has persisting hypertriglyceridemia secondary to other diseases.

One limitation of the present study is that even with a normal Spec cPL concentration and the absence of clinical signs of pancreatitis, mild residual inflammation of the pancreas cannot be definitively excluded. The only way to exclude this possibility would be through histopathologic examination of multiple pancreatic biopsies, a very invasive procedure that cannot be ethically justified in patients that have recovered from pancreatitis. In addition, even if some mild residual inflammation of the pancreas was present in some dogs, it is not known whether this can affect lipid metabolism. Theoretically, longer intervals between the time of the diagnosis of pancreatitis and the follow-up sample could have been used, but then it would have been more likely that, with increasing age, hypertriglyceridemia would develop, because previously reported data suggest that hypertriglyceridemia in the Miniature Schnauzer should be regarded as an age-related condition.[10]

In conclusion, Miniature Schnauzers with a history of pancreatitis were 5 times more likely to have hypertriglyceridemia than age-matched control dogs of the same breed. In addition, Miniature Schnauzers with

a history of pancreatitis were almost 15 times more likely to have moderate to severe hypertriglyceridemia (> 500 mg/dL) than controls. It is logical to assume that hypertriglyceridemia was a pre-existing condition in most Miniature Schnauzers with a history of pancreatitis in this study. This study shows that, in contrast to control dogs, the majority of Miniature Schnauzers with a history of pancreatitis have hypertriglyceridemia. Additional studies are needed to further clarify the role of hypertriglyceridemia in the development of pancreatitis in Miniature Schnauzers as well as other dog breeds.

Footnotes

[a] Spec cPL, IDEXX Laboratories Inc, Westbrook, ME
[b] McCord K, Davis J, Leyva F, et al. A multi-institutional study evaluating diagnostic utility of Spec cPL in the diagnosis of acute pancreatitis in dogs. J Vet Int Med 2009;23:734 (abstract)
[c] Roche/Hitachi MODULAR ANALYTICS D 2400 module, Roche Diagnostics, Indianapolis, IN
[d] Free T4 (by ED), Antech Diagnostics, Irvine, CA
[e] Immulite 2000 Canine Total T4, Siemens Healthcare Diagnostics, Deerfield, IL
[f] Prism5, GraphPad, San Diego, CA

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