LETTER TO THE EDITOR

Journal of Veterinary Internal Medicine AC



Letter regarding "Association between abdominal ultrasound findings, the specific canine pancreatic lipase assay, clinical severity indices, and clinical diagnosis in dogs with pancreatitis"

Dear Editor.

I would like to comment on the excellent recent article by Cridge et al. (see "Association between abdominal ultrasound findings, the specific canine pancreatic lipase assay, clinical severity indices, and clinical diagnosis in dogs with pancreatitis" J Vet Intern Med. 2020 Jan 17. doi: 10.1111/jvim.15693). The authors are to be congratulated on tackling this important issue.

It is hardly surprising that a sonographic assessment of the likelihood of pancreatitis based on examination of the pancreas itself and the surrounding mesentery fails to correlate with lipase levels since, as the authors themselves acknowledge, features assessed in the scoring system employed (pancreatic enlargement, peripancreatic free fluid, changes in echogenicity of the pancreas itself, and adjacent mesentery) are common to all potential causes of pancreatic edema and pancreatic edema is not specific to pancreatitis.

Obviously, the authors are well aware of this, However, I would like to counter the implication, which might be drawn from the results, that ultrasound is not particularly useful in the diagnosis of acute pancreatitis. Greater specificity can be achieved by factoring in other information gathered during the sonographic examination. In particular, although there is a degree of subjectivity, it is important to assess whether hyperechoic changes in the mesentery are restricted to the peripancreatic area or, instead, are generalized or focused on other abdominal viscera in the cranial abdomen such as the biliary tract, gastrointestinal tract, portal vein, or liver. Localized changes in the pancreas or peripancreatic fat are more likely to be due to a primary pancreatitis and less likely to be due to systemic factors such as hypoalbuminemia.

It is important to rule out alternative causes of pancreatic edema. Anaphylaxis may be an important, underdiagnosed, cause of acute portal hypertension-mediated pancreatic edema. Obviously, we have

no definitive test for anaphylaxis: a diagnosis currently rests on a subjective assessment of criteria such as acute hypotension disproportionate to external fluid losses, elevated serum alanine aminotransferase activity, and portal vein wall edema. Similarly, inadvertent, iatrogenic overhydration (often in a setting of acute abdominal signs and especially in hyponatremic hypoadrenocorticism) can cause pancreatic edema due to posthepatic portal hypertension. Subjective assessment of potential hypotension or overhydration can be made during the sonographic examination by gauging filling of the abdominal vena cava and cardiac chambers.

Sonographic assessment of the probability of primary acute pancreatitis depends on global thoracic, cardiac, and abdominal sonographic examination with particular attention to circulatory status and potential alternative causes of pancreatic edema.

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