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Acute pancreatitis in the dog- current approach to management

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Acute pancreatitis is a relatively common problem in canine medicine. Many cases respond well to the traditionally cited treatment of fasting, intravenous fluid and electrolyte supplementation. However, there are a significant number of cases that do not readily respond to this treatment and require protracted and expensive hospitalisation, with a high mortality rate. Predicting which animals will require extra treatment is often difficult. Necrosis of the pancreas or peri-pancreatic area cannot be used alone as a poor prognostic indicator in dogs, and even the severity or extent of the necrosis does not necessarily correlate with survival as it is the systemic complications induced by the necrosis that is most likely to cause death or complications[1]. No single routine clinicopathological measurement or clinical finding can be used to predict severity and outcome in dogs either[2,3]. A recent study (in press) suggests severity scoring assessing complications in the cardiac and/or respiratory systems, gut integrity and vascular forces may be more accurate at predicting outcome and length of hospitalisation than measurement of serum C-reactive protein[3]. It may be important to triage the severe cases as soon as possible, to mirror the experience in people, where the best results are obtained if treatment is begun no more than 60 hours after the onset of symptoms[4].

Treatment options

Management of fluid, electrolyte and acid-base abnormalities is the first and most critical step in management of all of pancreatitis cases, regardless of severity.

Nutrition- parenteral or enteral?

There have been recent advances in understanding the role the gut plays in perpetuating inflammatory responses in a number of diseases[5]. Total parenteral nutrition has often been the nutritional support of choice in severely affected dogs, but doesn't appear to significantly reduce mortality or experimentally reduce severity of pancreatic inflammation and is problematic to administer (central line and sepsis control required, expertise in calculating formulation etc)[6,7]. Studies in experimentally induced pancreatitis in dogs have shown early intra-jejunal feeding to be beneficial in improving gut barrier function and it has minimal effect on entero-hormonal release[8,9,10]. Although jejunal feeding tube insertion may be readily undertaken if the animal has abdominal surgery, insertion via endoscopic placement is technically very difficult in dogs. Generally the small diameter of the jejunostomy tubes also makes some food preparations difficult to administer. A more recent study in people showed no difference in clinical outcome when using naso-gastric versus naso-jejunal feeding[11]. There is certainly no doubt that oesophageal feeding tubes (not gastric in order to reduce reflux and subsequent oesophagitis) are easier to place and maintain in dogs than jejunal feeding tubes.

Although evidence is mounting that early enteral nutrition is preferable, it is not so clear what is the best diet to feed. We have assessed pancreatic response (by measuring c-PLI, c-TLI, gastrin and cholecystokinin) in healthy dogs fed a variety of foods[12]. We found no discernible difference in feeding normal commercial dog food compared to low-fat dog food. There was however a slight trend towards less exocrine pancreatic stimulation if dogs were fed a low-fat diet in combination with oral pancreatic enzyme extracts and medium chain triglycerides. The use of 'micro-enteral' or trickle feeding has been shown to be beneficial in cases of canine parvovirus, and so could be recommended for any severe gastrointestinal disease[13].

Anti-emetics, pro-kinetics and gastric acid inhibitors

Control of nausea and vomiting is extremely important if enteral feeding is to be successful. In cases where there is significant ileus or vomiting then pharmacological intervention is certainly indicated. However, there may be some basis to avoiding metoclopramide, a dopaminergic antagonist, as dopamine has been shown to be beneficial in rats with experimentally induced pancreatitis[14]. Use of new generation 5-HT2 or NK-1 receptor antagonists that work on the vomiting and CTZ centres may be preferable.

There is no proven benefit shown in dogs (nor people) with pancreatitis by inhibition of gastric acid secretion *per se*[15]. Widespread ileus and gastric hypomotility are common consequences of pancreatitis and the resultant peritonitis. Persistent ileus may contribute to and exacerbate refractory vomiting. Many of the agents used as anti-emetics also have some pro-kinetic effects, although this benefit may only be minor. Although cisapride has been withdrawn from the market, it is still readily available from compounding pharmacies. It has a serotonergic effect with minimal dopaminergic inhibition. In animals with refractory vomiting we administer the dose rectally at 1/3 of the oral dose

(reported bioavailability 35-40%), although this has not been supported by any pharmacological studies. Due to potential adverse effects (cardiac arrhythmias have been reported in people, along with abdominal cramping) we tend to reserve use of this drug for dogs with profound ileus who otherwise would not tolerate enteral feeding.

Plasma

The use of plasma is widely accepted, but not proven, as being beneficial in cases of severe canine pancreatitis. In cases where there is development of a coagulopathy, plasma administration is imperative (along with Vitamin K supplementation). Once we are able to objectively determine which dogs are severely affected upon presentation it may be that administration of plasma daily (70 ml/kg/day) for the first 2-3 days of hospitalisation may be 'prophylactically' beneficial.

Antibiotics

There is no proven benefit in using antibiotics in dogs with pancreatitis, but they are commonly administered. Dogs very seldom get infected local necroses and it may be that by using early enteral nutrition alone we will reduce the risk of sepsis by reducing bacterial translocation[9,10]. If antibiotics are indicated then selection of a broad spectrum antibiotic effective against gram-positive and gram-negative bacteria given parenterally is recommended.

Treatment of local complications

Development of pancreatic fluid collections or pseudocyst (sterile) can be performed via surgical omentalisation, ultrasound-guided drainage or benign neglect, with close monitoring, In one study surgical treatment of pancreatic masses resulted in a very high mortality rate[15]. In my experience, benign neglect in an otherwise recovering animal is certainly the best path to take, with frequent re-assessment. Similarly, when extra-hepatic bile duct obstruction occurs, ultrasound-guided aspiration of the gall bladder can be performed relatively safely[16]. As the obstruction is often functional ileus rather than physical obstruction this procedure may not be necessary in most cases.

Prednisolone

There are many systemic complications in pancreatitis that may be beneficial to counteract, as cytokine and leucocyte products, elastase, oxygen radicals, elastase and interferon are all important in the role they play in acute pancreatitis[4]. As use of newly developed drugs effective against cytokines is not a realistic option in most canine cases, other options to decrease systemic complications do need to be revisited in veterinary medicine. Older canine experimental studies evaluated the use of methylprednisolone (at a dose rate of 30 mg/kg IV) given prophylactically and 30 minutes after induction of pancreatitis[18,19]. They found that there was an increase in cardiac output but no alteration of pancreatic blood supply. As such, recommendations for the use of corticosteroids has been limited to extreme cases where cardiovascular shock develops. Recent studies in people have shown amelioration of haemodynamic status, reduction in number of organ failures and increased incidence of survival when low to medium dose corticosteroids are administered[20]. Given that this benefit in people is present in the face of increased risk of sepsis and infection, it is highly probable that this benefit may also be seen in our patient population. However, recommendation of the routine use of corticosteroids in canine pancreatitis is certainly premature and not scientifically validated.

Conclusion

It will be difficult to plan clinical studies on canine acute pancreatitis that will be capable of determining the specific benefits of individual treatment strategies. As such, there needs to be a more structured approach addressing the multi-agent approach targeting the multi-organ affects of the systemic inflammatory cascade.

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