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# Acute Pancreatitis

DRIP 1: INTRODUCTION

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# Acute Pancreatitis

## *Diagnosis & Management*

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Hi, everybody. Welcome to Acute Pancreatitis. It's one of the most common issues for which we all see our dogs and cats for a variety of clinical signs, typically referable to the gastrointestinal tract. We have quite a bit to discuss.

# Objectives



Review relevant pathophysiology



Highlight risk factors & clinical signs



Discuss diagnostic testing



Discuss therapeutic interventions

So we're going to dive right in, starting first with just the relevant pathophysiology. This is not an ivory tower talk. I know nobody really wants to hear about pathophysiology in depth, but there is some modicum of pathophysiology that we need to understand, to understand the diagnostic tests that we're doing, to understand the therapies that we're prescribing for these kiddos. But as always, we're going to spend the vast majority of our time talking about what these patients look like, how we accurately diagnose them, and what therapies do we administer to get them home to their families.

## Normal Safeguards

Digestive enzymes stored as inert zymogens in acinar cells

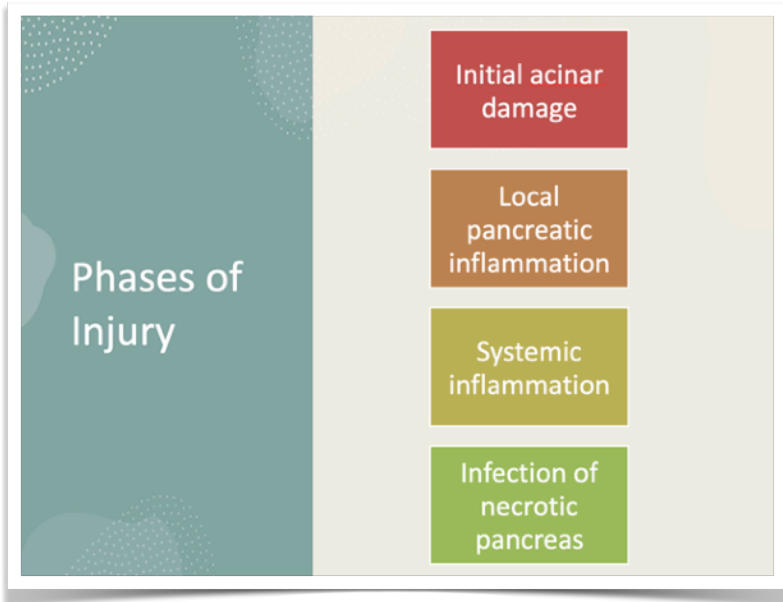
Zymogen granules remain separate from lysosomal granules enclosed in membrane-bound organelles

Pancreatic secretory trypsin inhibitor (PSTI) within acinar cells

Large anti-proteases in circulation

So, when we talk about the pancreas under normal circumstances, the pancreas is a relatively well-protected organ. So we have these potent digestive enzymes that are stored in their inactive form. And they're kept separately from lysosomal granules.

Now things like pancreatic secretory trypsin inhibitor-- that's a big mouthful of words-- that's in the cytoplasm of acinar cells, and it binds to trypsin. So it prevents it from doing its digestive tasks. And we also have large anti-proteases in peripheral circulation that can combat any prematurely released digestive enzymes.



And so we think of the pancreas being damaged in phases.

So the first phase is the initial acinar damage. And that inevitably is going to lead to hopefully local pancreatic inflammation. We just want to keep it isolated to the pancreas, but as we all know. Unfortunately, despite the body's best efforts, or because of concurrent illnesses, that local pancreatic inflammation isn't always contained.

Sometimes these pro-inflammatory cytokines that are produced in that local pancreatic environment gain access to the systemic circulation. And they go off to affect other organ systems, like the kidneys, like the liver, like the lungs in cats, in particular. So as much as we always want that local inflammation to remain very focally in the region of the pancreas, we don't always get our wish.

And the inflammation becomes the systemic. Our patients develop, for example, systemic inflammatory response syndrome. And then, of course, because of splanchnic circulation, a dirty intestinal tract, for lack of a more classy

way of saying it, the pancreas can also become colonized with gastrointestinal bacteria, leading to infection and necrosis.

# Risks Factors

Cause is frequently unknown

Breed/Pedigree

- *Dogs*: Schnauzers, Yorkshire terriers, Dachshunds, Poodles, Cocker Spaniels, Fox Terriers, Alaskan Malamutes
- *Cats*: Siamese, Domestic Shorthairs

Biochemical

- *Dogs*: hyperlipidemia, hypercalcemia
- *Cats*: hypercalcemia

So what are some risk factors? Well, in general, we don't know what actually truly triggers the pancreas. We know that some breeds are overrepresented. In the canine world, the classic poster child are your Schnauzers, miniature, standard, but Siamese cats-- in studies you'll see domestic shorthairs. I think that's just because it's the most common breed of cat, and that's why it's overrepresented. But technically, in some of the literature, you'll see domestic shorthairs listed as an overrepresented pedigree.

Hyperlipidemia in dogs, we all know is a risk factor, but so too is hypercalcemia in both dogs and cats.



# Risk Factors

## Miscellaneous

- *Dogs*: previous surgery other than neutering
- *Cats*: bile reflux, duodenal reflux
- *Both*: CBD obstruction, ischemia, pancreatic tumors

## Medications

- *Dogs*: azathioprine, KBr, sulfonamides, N-methyl-glucamine, anticholinesterases, L-asparaginase (?)
- *Cats*: organophosphates

Other risk factors are altered males and females, at least in the canine world are overrepresented compared to intact males and females. Older dogs-- although, in my experience, it doesn't matter what age you are, but in the literature geriatric dogs are overrepresented and certainly concurrent metabolic diseases. In the cat, for example, we often hear of triaditis, that triple cocktail of pancreatic inflammation, a form of inflammatory bowel disease, and cholangitis, cholangiohepatitis.

But in dogs, we see a lot of concurrent diabetes mellitus. And to me that one makes sense. If one part of the pancreas is unhappy, what's to prevent the other part of the pancreas from not becoming unhappy? Chronic kidney disease, various neoplasia, certainly inflammatory bowel disease, just like in their feline compatriots, and believe it or not, autoimmune disorders. I think it's because of the profound systemic inflammation that is happening in the body.

# Risk Factors

## Nutrition

- *Dogs*: ingestion of unusual food items, getting into trash, ingestion of table treats

## Infections

- *Dogs*: babesiosis
- *Cats*: *Eurytrema procyonis*, *Amphimerus pseudofelineus*, *Toxoplasma gondii*, calicivirus, FIP (?), feline parvovirus (?)

Nutrition, we all refer to the day after Thanksgiving, the day after the 4th of July, or even the 4th of July itself, the day after Christmas as pancreatitis days in urgent and emergency care, because dogs tend to have some form of dietary indiscretion. They get into things that they shouldn't have or their owners give them things that they shouldn't have given to them, all those fun table treats, not really an issue in our feline patients. Hyperlipidemia is not seemingly a major issue in our kitty friends.

Certain infections also seem to increase the risk in our canine kiddos. Babesiosis has been associated with pancreatitis. So if you live in a part of the country or part of the world where you deal with Babesia species, be aware that could-- babesiosis could manifest as pancreatitis.

In our kidneys, lots of infections, but the ones that stick out to me are your liver flukes, which are your *Eurytrema* species and your *Amphimerus pseudofelineus*, but toxoplasmosis is definitely on the list. I just diagnosed pancreatitis induced by *Toxoplasma* in a cat earlier this week. So it definitely does happen.

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And then miscellaneous things, so surgery, going into the abdominal cavity. How many of you, when you're going through senior rotation or maybe doing an internship worked with a surgeon and the surgeon said, that's the pancreas. Don't touch it. Don't even look at it, because in my opinion, it is the most fickle organ of the body.

You look at it sideways, it's going to get angry, give you the middle finger, and get inflamed. There's a lot of evidence that reflux from the duodenum or straight up biliary reflux is a contributing factor in cats, because of their unique anatomy. And then, of course, pancreatic tumors, obstruction of the common bile duct, because of either a lith or a neoplasm all are categorized as miscellaneous risk factors.

Drugs are often discussed, but to be honest with you, the evidence for drug-induced pancreatitis is more in the correlative category rather than the causative category for the vast majority of these. For example, the organophosphate's, association with cats was from a case series.

Azathioprine is pretty well-documented, as is potassium bromide. I personally feel that I have seen dogs develop pancreatitis rarely after an L-asparaginase injection, when we used to use that a lot more in chemotherapy for lymphoma, but again taking a step back and trying to be objective, the evidence is wishy-washy at best.