

Examples

2 SOAP's – one extensive (by necessity) and one less extensive

Problem Analysis: Ichiro – ATLes Case #3

Problem #1 = Diarrhea – chronic, small bowel

SO: Diarrhea in this animal is chronic and appears to be progressing (getting worse). The high volume & low frequency suggests that the diarrhea is small intestinal in origin, as does the absence of fresh blood, mucus, and tenesmus, which are the cardinal signs of large bowel diarrhea in small animals. The chronic small bowel diarrhea accompanied by weight loss is most suggestive of a small intestinal malabsorption syndrome, possibly with protein loss into the feces.

A: {a} General pathophysiologic mechanisms and {b} Mechanisms most likely in this case:

{a} Non-gastrointestinal diseases such as hypoadrenocorticism, chronic renal failure, and hepatic disease can produce diarrhea. {b} There is no evidence for these on physical examination or laboratory data, although hypoalbuminemia could be a reflection of poor hepatic function, and not all hepatic disease will be accompanied by abnormal liver enzyme activities

Primary gastrointestinal disease: Malabsorption seems most appropriate.

{a} Maldigestion due to exocrine pancreatic insufficiency is one possible explanation. Undigested nutrients cannot be absorbed and provide substrate for intestinal bacteria. The result is osmotic diarrhea. {b} In Ichiro, the results of the TLI do not support this DfDx. It may be useful to repeat the test (see below).

{a} A small intestinal malabsorption syndrome is a good consideration. The decreased ability to absorb nutrients, typically due to an infiltrative mucosal disease, creates an osmotic diarrhea and weight loss. Depending on the cause of the malabsorption there may also be a significant exudative component to the diarrhea as water and other components of the interstitial fluid or plasma are lost into the intestinal lumen. Low serum cobalamin could be seen with severe ileal inflammation causing poor absorption of cobalamin. Likewise upper intestinal malabsorption may result in lower than normal folate levels. {b} Hypoalbuminemia is consistent with these diseases due to increased loss (a protein-losing enteropathy; PLE) and/or malabsorption. More commonly, however, BOTH albumin and globulin (a larger molecule) are lost with PLE.

{a} Small intestinal bacterial overgrowth (SIBO) is most often secondary to another disease process, such as exocrine pancreatic insufficiency, or diseases associated with stasis of intestinal contents (tumors, inflammatory bowel disease, partial obstructions). This overgrowth of normal flora, especially in the proximal small intestine, results in a number of functional changes including alterations in digestion & absorption of fat, diminished activity of digestive enzymes in the brush border, and increased secretion of fluids by enterocytes. The diarrhea that is seen is primarily osmotic. {b} SIBO is supported in this case by the low serum cobalamin and increased folate. However, the lack of response to a broad spectrum antibiotic often used for bacterial overgrowth would make this differential less likely, especially as a primary cause. The SIBO is most likely secondary to an underlying problem, which we need to identify.

{c} DfDx's for this problem

- ❖ Inflammatory bowel disease:
Lymphocytic/plasmacytic enteritis and eosinophilic enteritis are most common: produce diarrhea by osmotic and exudative mechanisms. Hypoalbuminemia is consistent with these diseases due to increased loss and/or malabsorption.
- ❖ Lymphangiectasia:
Hypoalbuminemia is also consistent with this disease due to protein loss. Diarrhea is not always seen with this disease, but when it occurs is primarily osmotic and associated with fat malabsorption in particular.
- ❖ Intestinal neoplasia:
A diffuse intestinal lymphoma, in particular, can result in malabsorption due to infiltration of the mucosa with neoplastic cells. The infiltrate impedes nutrient absorption and may make the mucosa "leaky". A focal neoplasia (e.g. lymphoma or adenocarcinoma) can produce partial obstruction (see SIBO above). Hypoalbuminemia may be seen with neoplastic diseases due to transmucosal loss.
- ❖ Small intestinal bacterial overgrowth (SIBO): See above.
- ❖ Other considerations:

Food allergy or intolerance: would still be a consideration as 2.5 weeks is not sufficient to adequately assess response to dietary change (allergy may require 4-6 weeks); wouldn't commonly expect hypoalbuminemia with these differentials.

GI parasites: These would seem unlikely now given the absence of a response to a broad-spectrum anthelmintic. Whipworms usually cause signs of colitis (no evidence for), and rarely an Addison's-like disease which is not supported in this dog. One consideration is still giardiasis, which can be difficult to diagnose and difficult to treat. However, the treatments instituted already would have been expected to be effective against giardia. Likewise, we don't expect hypoalbuminemia with giardiasis – although the hypoalbuminemia could be resulting from something besides GI disease.

P: Initial Plan to address this problem:

- ❖ Repeat folate & cobalamin: especially now, following antibiotic treatment. Is there any indication of change?
- ❖ Repeat TLI: just to be sure we can rule out EPI, which is a treatable disease. A single value could always be in error.
- ❖ Repeat CBC, Panel and UA: These values are now several weeks old and things may have changed. This will also help rule out non-GI causes of diarrhea, such as liver or renal disease.
- ❖ Imaging – recommend abdominal ultrasound as the most sensitive method to evaluate thickness of the intestinal mucosa, look for abdominal masses, and examine mesenteric lymph nodes for increased size. Since IBD and alimentary lymphoma are important DfDx, this may be best non-invasive way to look for compatible lesions. However, both of these diseases can, at least initially, produce very subtle lesions that might not be detected.
- ❖ Future consideration: in order to make a Dx, we may ultimately need to biopsy intestine, either via endoscope or laparotomy.

SOAP = Subjective, Objective, Assessment/analysis, Plan

Problem #2 = Hypoproteinemia - due to hypoalbuminemia

{a} General pathophysiologic mechanisms

Hypoalbuminemia can be caused by 3 general mechanisms:

1. Decreased production - liver disease (there is currently little evidence for liver disease).
2. Increased loss via the:
 - Gastrointestinal tract
 - Kidney (especially glomerular disease; a protein-losing nephropathy)
 - 3rd space – lost into a cavity such as chest or abdomen (e.g. due to vasculitis)
3. Decreased intake (chronic malnutrition)

{b} Mechanisms most likely in this case:

1. There is currently little evidence for liver disease. However, a more sensitive function test like bile acids would be necessary to address more carefully.
2. GI loss is supported by history of diarrhea and weight loss with DfDx's as for problem #1. Protein-losing enteropathies are classically associated with panhypoproteinemia due to loss of both albumin and globulin.
3. Loss into urine: a urinalysis should be performed to exclude the contribution of urinary tract losses to the low albumin in this dog.
4. 3rd spacing: There is no evidence or history of fever, edema or ascites as might be expected with pleuritis or peritonitis; no inflammatory leukogram or thrombocytopenia as might be seen with vasculitis.
5. Decreased absorption of protein could be contributing to the hypoproteinemia. However, even dogs with EPI and severe malassimilation most commonly have normal serum protein due to production by the liver.

{c} DfDx for this problem – See Problem #1 regarding PLE

{d} Initial Plan (P) to address problem:

- ❖ Repeat panel to see if hypoproteinemia is worsening or otherwise changed.
- ❖ Perform a U/A to rule out protein-losing nephropathy (renal disease).
- ❖ Pursue intestinal disease as described under Problem #1.

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Over to see an example of a DfDx analysis ⇨

DfDx Analysis (pathology) - EXAMPLE

Inflammatory Bowel Disease

{a} Lesion: Enteritis, lymphoplasmacytic, chronic, diffuse (or segmental), severe

Gross: The mucosa may be grossly unremarkable. However, in severe cases, the mucosa may be thickened due to infiltration by lymphocytes and plasma cells.

NOTE: When severe, the thickening MAY be detectable by an experienced and skilled ultrasonographer. However, the absence of ultrasound changes does not rule out IBD. The intestine may be diffusely affected, however the lesions are commonly segmental, affecting some sections of the gut and not others.

Microscopic: Diffuse infiltration of the intestinal mucosa by mononuclear cells, lymphocytes and plasma cells. Affected mucoa is commonly also characterized by broad, short villi (villous atrophy), which results in a decreased absorptive surface area.

{b} Pathogenesis: Although broadly considered an idiopathic disease, this lesion likely represents dysregulation of the mucosal immune system. Immune cells are likely responding inappropriately to normal bacterial flora in the intestinal tract or possibly something else in the intestinal lumen. Some cases may represent a hypersensitivity to something in the diet. The result is infiltration of the mucosa with lymphocytes and the local release of inflammatory mediators and cytokines.

{c} Pathophysiology: Infiltration of the mucosa with inflammatory presents a physical barrier to nutrient absorption, producing malabsorptive osmotic diarrhea. The accompanying villous atrophy also decreases the absorptive surface area and results in villi being populated by enterocytes which do not reach full maturity before they are extruded into the intestinal lumen. The infiltrating cells and the inflammatory mediators/cytokines that are produced locally also result in "leaky" mucosa and the potential loss of plasma proteins into the feces. Long term malabsorption can produce severe weight loss and eventually cachexia.

NOTE: If the proximal bowel (stomach and/or duodenum) or large intestine are affected, vomiting and large bowel signs may be prominent.

{d} Diagnosis: the only way to make a definitive diagnosis is demonstration of the lesion by histopathology. Lesions may be obtained by endoscopic mucosal biopsy IF the proximal GI tract or colon are affected. Otherwise, an exploratory laparotomy is required. Other diagnostic approaches include ruling out other possible DfDx's and demonstrating compatible lesions by ultrasonography. Ultrasound is not specific (lymphoma is the primary DfDx) nor particularly sensitive, and requires significant expertise on the part of the ultrasonographer.