

Domain 1b Appendix Example – Stephen Hines

The Characterization / Sorting Exercise

INSTRUCTIONS:

1. If it's not already done for you, cut along the dotted lines to create 3 sets of colored "cards"
 - Clinical diagnoses (or clinical scenarios, depending on exercise)
 - Lesions
 - Mechanisms of disease (pathogenesis or pathophysiology)
2. The information on these cards was used on a previous Sys Path. However **BEFORE** you try to use the cards to create a 3 or 4 way match, please do the following FIRST.
 - Starting with the CLINICAL SCENARIOS:
 - Characterize each case and SORT them in several different ways that make sense to you. In other words, identify problems and key features, and then try to put the scenarios into categories.
HINT: consider using some of the "semantic qualifiers" that we used in our discussion in lab. These might include acute versus chronic, signalment, etc.
 - Within your group, discuss some of the different ways you each came up to sort the cases.
 - Consider writing down Master Problem Lists, key features, and DfDx's – as we have in lecture and lab.
 - Now, do the same with the LESIONS cards:
 - Are there characteristics that allow you to group these in a variety of different ways?
 - There are likely to be a limited numbers of diseases that each lesion fits – maybe even just one. Can you make a diagnosis or construct a DfDx list?
 - Lastly, do the same with the MECHANISMS:
 - Again, there are likely to be a limited numbers of diseases that each mechanism fits – maybe just one. Can you identify the disease or lesion that fits each?
3. **AFTER** you've considered each set of cards, use the cards to create 3 or 4 way matches. Enter the letter or number of each into the provided table.
 - There should be multiple copies so each member of your group might want to first try it on their own – before the group talks about it. Or you can work on it together.
 - Not all the cards will be used in matches, but you should still have some good ideas what is being described.

Diagnoses – cut into cards (card stock)

<p>A. Alimentary lymphoma</p> <p><i>2014 Exam #1 – Problem #4</i></p>	<p>B. <i>Clostridium difficile</i></p> <p><i>2014 Exam #1 – Problem #4</i></p>
<p>C. <i>Clostridium perfringens</i></p> <p><i>2014 Exam #1 – Problem #4</i></p>	<p>D. Coccidiosis</p> <p><i>2014 Exam #1 – Problem #4</i></p>
<p>E. Coronavirus</p> <p><i>2014 Exam #1 – Problem #4</i></p>	<p>F. Cryptosporidiosis</p> <p><i>2014 Exam #1 – Problem #4</i></p>
<p>G. Enterotoxigenic <i>E. coli</i></p> <p><i>2014 Exam #1 – Problem #4</i></p>	<p>H. Exocrine Pancreatic Insufficiency</p> <p><i>2014 Exam #1 – Problem #4</i></p>
<p>I. Giardiasis</p> <p><i>2014 Exam #1 – Problem #4</i></p>	<p>J. Inflammatory Bowel Disease</p> <p><i>2014 Exam #1 – Problem #4</i></p>
<p>K. Lactose intolerance</p> <p><i>2014 Exam #1 – Problem #4</i></p>	<p>L. Lymphangiectasia</p> <p><i>2014 Exam #1 – Problem #4</i></p>
<p>M. Pancreatitis</p> <p><i>2014 Exam #1 – Problem #4</i></p>	<p>N. Paratuberculosis (Johne's)</p> <p><i>2014 Exam #1 – Problem #4</i></p>
<p>O. Parvovirus</p> <p><i>2014 Exam #1 – Problem #4</i></p>	<p>P. Rotavirus</p> <p><i>2014 Exam #1 – Problem #4</i></p>
<p>Q. Salmonellosis</p> <p><i>2014 Exam #1 – Problem #4</i></p>	<p>R. SIBO</p> <p><i>2014 Exam #1 – Problem #4</i></p>

1. Repeated. 1. The lesion consists of villous atrophy and mild to (possibly) moderate inflammation that is neutrophilic in nature.

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3. Grossly, the pancreas is reduced to what appears to be a thin, transparent membrane – perhaps with small remnants of pancreatic tissue.

2014 Exam #1 – Problem #4

5. Repeated. 5. The primary lesion is acute, diffuse, severe, ulcerative colitis.

2014 Exam #1 – Problem #4

7. Necropsy reveals hemorrhagic enterocolitis. On histopathology, there is severe loss of enterocytes and a neutrophilic infiltrate in the mucosa. Many of the remaining enterocytes contain various stages of an intracellular protozoal parasite.

2014 Exam #1 – Problem #4

6. Full thickness biopsies of the intestine show marked dilation of lymphatics in both the mucosa and submucosa. There is little to no increase in cells within the lamina propria or submucosa.

2014 Exam #1 – Problem #4

9. Repeated. 9. The pancreas is diffusely soft, swollen, and edematous - with multiple 2-4 cm areas of hemorrhage scattered throughout.

2014 Exam #1 – Problem #4

4. Repeated. 4. There are typically no gross lesions in the intestines (except perhaps for soft to watery intestinal contents) and no significant microscopic lesions – including no evidence of mucosal damage and little to no inflammation.

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2. Repeated. 2. Villi are diffusely short, blunt, and infiltrated with cells that broadened the lamina propria. The epithelium appears intact. The infiltrate is a mixture of lymphocytes and plasma cells that are confined to the mucosa.

2014 Exam #1 – Problem #4

8. Villi are short and blunt, and crypts are hyperplastic. The epithelium is intact, but in many sections numerous small round protozoa are attached to the luminal surface of enterocytes.

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Mechanisms: pathogenesis or pathophysiology – cut into cards (card stock)

h) An essential part of the pathogenesis is production and secretion of potent cytopathic microbial exotoxins.

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b) Repeated: (b) The primary mechanism of diarrhea is osmotic due specifically to malabsorption of dietary fat.

2014 Exam #1 – Problem #4

c) A primary mechanism of disease likely involves diminished activity of membrane-bound digestive enzymes. This reflects the effect of the organism on the intestinal microvilli. Malabsorption due to overall diminished intestinal absorptive surface area also plays a central role in the pathophysiology.

2014 Exam #1 – Problem #4

d) Repeated: (d) An essential part of the pathogenesis is production and secretion of a potent microbial exotoxin. The effects include unchecked secretion of chloride in the crypts and decreased uptake of NaCl by affected villi.

2014 Exam #1 – Problem #4

g) It's controversial how important this is as a primary cause of diarrhea. However, there may be several mechanisms of diarrhea in play, notably maldigestion due to sub-microscopic disruption of the intestinal brush border and microbial inactivation of bile acids in the intestinal lumen.

2014 Exam #1 – Problem #4

f) Replication of this cytopathic pathogen is limited to the intestinal tract. Damage is usually mild because only mature enterocytes are affected. There may also be transient maldigestion of carbohydrates (like lactose) due to impaired disaccharidase activity in the brush border.

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a) Repeated: (a) In dogs, clinical signs are typically preceded by a silent (asymptomatic) phase in which there is selective immune-mediated destruction of enzyme-producing cells.

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e) Repeated: (e) The lesion is produced by a pathogen that replicates in epithelial cells and destroys the cell as it exits. The various stages of the life cycle can be visualized using light microscopy.

2014 Exam #1 – Problem #4

i) Breakdown of immunologic tolerance to luminal antigens (bacterial and/or dietary components) is thought to be critical, perhaps resulting from alterations in the intestinal microflora or some disruption of the mucosal barrier.

2014 Exam #1 – Problem #4

j) The primary mechanism of diarrhea (pathophysiology) is loss of epithelium and exudation. The damage is extensive due to the pathogen's requirement for rapidly dividing cells.

2014 Exam #1 – Problem #4

PROBLEM #4: NO ANSWER IN ANY CATEGORY CAN BE USED MORE THAN ONE TIME !

TIP: FIRST, examine each case individually – making a Problem List and identifying KEY FEATURES. **THEN**, begin by first completing the matches you know. This decreases the number of remaining choices for matches you are less sure about.

Clinical Scenario	Dx	Lesion	Mechanism
<p>A. A 2-year-old MC Yorkshire Terrier is presented for progressive weight loss and diarrhea. The owners report a 3 month history of intermittent soft stools that is now consistent. The dog’s appetite was good but now increasingly intermittent. Feces are yellow to light brown, sometimes greasy, and always voluminous. There is no reported blood, mucus, or straining. A CBC and biochemistry panel show lymphopenia, moderate hypoalbuminemia, and hypocholesterolemia. Fecal floats were negative and there was no response to a hypoallergenic diet, probiotic, or a 2 week course of antibiotics. TLI, folate, and cobalamin were all within normal limits.</p>			
<p>B. <i>Repeated: B. A week after dealing with an outbreak of calf diarrhea, your partner develops what she describes as “impressive” high volume, watery diarrhea and “stomach cramps”. The signs have already lasted another week, and she’s had trouble staying hydrated. The calves had similar clinical signs. There was no blood or significant straining. Most were at least 10 days of age and none were older than about 4-5 weeks. The calves were treated symptomatically and management changes were recommended. Most calves recovered after 7-14 days and now seem fine. She is hoping for a similar outcome, but has already lost 5 pounds.</i></p>			
<p>C. A 7-year-old MC Greyhound has had intermittent diarrhea and vomiting for the past year. His appetite remains mostly good but there is now significant weight loss so that his body condition score is now 2/9. The vomitus may or may not contain food, but there is no blood. Feces have gone from soft to watery. The signs seem to wax and wane but the owners report 2-4 episodes per day during bad periods. Repeated fecal floats for parasites have been negative and there was no response to antibiotics. There has been some improvement since starting a highly digestible, single protein diet. The signs improved considerably in response to high dose corticosteroids, but the owners were unhappy with the side effects. The only abnormalities on CBC, biochemistry panel, and U/A have been hypoalbuminemia and hypoglobulinemia, both of which have worsened over the past 4 months. TLI, folate, and cobalamin are normal.</p>			
<p>D. <i>Repeated: D. After just 2 weeks, a 1.5-year-old FS German Shepherd with chronic small bowel diarrhea and weight loss of approximately 6 weeks duration is responding very nicely to a course of oral antibiotics. Blood work sent to the TAMU GI lab revealed normal TLI but increased folate and decreased cobalamin. Extensive abdominal imaging, which included ultrasound, was unremarkable. A CBC, biochemical panel and U/A were similarly normal.</i></p>			
<p>E. You are called to look at a problem with scours in a small herd of beef calves. The owner reports watery diarrhea in calves up to about 2 weeks of age. Signs in calves have started at anywhere from 2 to 11 days of age. Once diarrhea begins, it can last 6-10 days. There’s been no blood or straining, but the diarrhea is watery. All but one calf recovered but most required extensive therapy using a commercial electrolyte solution and esophageal feeder. 12 of the first 15 calves have been affected. 2 of the 12 had mild fevers and all were significantly dehydrated. The owner is hoping for some help as she’s got 20 more cows due to calf soon.</p>			
<p>F. <i>Repeated: F. Two days after what seemed a successful minor orthopedic surgery, a 3 year old Thoroughbred stallion breaks with profuse, watery diarrhea and fever.</i></p>			

PHOTOS from 2013 labs depicting active student engagement in exercises.

