

**Clinical Correlation: Abdomen**  
**Case Presentations**  
**September 2004**

- I. A 55 y.o female with a 10 year history of poorly controlled diabetes mellitus presents with sudden onset of RUQ pain. She states the pain 6 out of 10 in severity, and is sharp to burning in nature. It is constant and radiates around to her back. She reports no nausea or vomiting, or change in her bowel habits. She denies any change with food intake.

What is your differential for this woman's RUQ pain?

What signs would you look for on physical exam?

- II. A 45 y.o. male with a significant ETOH abuse history comes into the E.D. with sudden onset of midepigastric pain. He states he had hurt his back recently lifting some papers but this pain is different. It is 10/10 and burning in nature. It "bores" through to his back. Initially, it was only present when he began to eat but now it is constant. He feels slightly nauseated but has not vomited. He did note that recently his stools had turned to a "tarry" color and consistency.

What is this patient's most likely diagnosis? Be specific.

How would you initially treat him?

Shortly after admitting him he was noted to vomit bright red blood. His blood pressure began to drop and his pulse rate increased. What do you hypothesize may have occurred?

- III. A 42 y.o. woman who is six weeks post partum presents with recurrent RUQ pain. She states that she has had "attacks" like this in the past but it is now becoming more severe. She describes her pain as 4-7/10, "colicky" and worsened after some meals. The pain radiates to her right scapula. She has nausea but has not vomited. Her stools have not changed color but had been black shortly after the delivery of her child.

What is the likely cause of this woman's pain and what kind of study might aid you in confirming your diagnosis?

What might her meals contain that seem to exacerbate her symptoms? Why?

The patient undergoes a procedure to remedy her disease process. However, she returns 6 weeks later with severe midepigastric pain, fevers, and persistent nausea and vomiting. What happened?

- IV. A 45 y.o.m. presents to your office for the first time complaining of a "swollen belly." He has noted it increasing in size over the last four months. He denies any alcohol usage but states he did "experiment with drugs" back in the late '70's. On exam, he appears cachectic and his sclera are mildly icteric. You note he has gynecomastia and dilated blood vessels on his abdomen. His abdomen is protuberant and when you percuss it you get dull sounds in the flanks bilaterally.

What is going on in his abdomen and why?

What is/are the mechanisms for the development of his abnormal physical findings? Can you think of any other findings on your exam that might occur as a result of his disease?

- V. A 66 y.o. general surgeon comes into the E.D. from a local restaurant with the "worst pain of my life." He states he developed severe, constant, sharp pain about 20 minutes after eating his meal. He is writhing on the gurney from his pain. However, when you perform your physical exam his abdomen is soft, with slightly diminished bowel sounds but non-tender throughout and without any periumbilical pulsatile masses.

What do you think is going on with this gentleman and what would you want to know about his past medical history?

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**Answers and Study Suggestions**  
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- I. Differential diagnosis for RUQ pain: gall bladder disease, retrocecal appendicitis, hepatitis, peptic ulcer disease, RLL pneumonia, ascending cholangitis, neuropathy (truncal, HZV).

Your exam should obviously focus on the abdomen. Note any skin changes, asymmetry, tenderness to percussion and/or palpation. Look for any chronic changes associated with liver disease (see Case IV).

This woman had a truncal (thoracoabdominal) neuropathy which can be seen in patients with poorly controlled diabetes. The pain is felt to be ischemic in nature, involving the ventral rami of the thoracic spinal nerves (intercostals). They innervate the abdominal wall musculature and the sensory distribution to the skin in this area.

- II. Peptic ulcer disease, specifically a duodenal ulcer.

The etiology of his ulcer is likely multifactorial: ETOH, ? NSAID's (for his back pain), ? H. pylori. Therefore, removing any inciting agents would be first line treatment along with acid suppression.

Most duodenal ulcers lie within the first portion, within 5 cm of the pylorus. Although most are anterior, posterior ulcers may erode through the wall of the duodenum and into the gastroduodenal artery (branch of common hepatic artery from the celiac trunk). This patient is exsanguinating! He will need surgery.

**Review the (4) portions of the duodenum and their anatomic associations.**

III. She likely has gall bladder disease (Four F's....or 5 if from central Pa.). A right upper quadrant ultrasound is the best to diagnose this condition. If the obstruction is in the cystic duct the gall bladder will be dilated and tender with palpation (+ ultrasound Murphy's sign).

Fatty meals and fried foods are often the difficult foods. The gall bladder stores and concentrates bile which aids in the digestion of fats.

**Trace the passage of bile from its formation in the hepatocytes until it enters the second part of the duodenum.**

Occasionally, after a cholecystectomy – particularly if an intraoperative cholangiogram is not performed – there may be retained stones that can obstruct anywhere from the common bile duct to the major duodenal papilla. This woman is showing signs of pancreatitis secondary to obstruction of the pancreatic duct.

**Terms:** cholelithiasis (gall stones), choledocholithiasis (stones within the bile ducts beyond the cystic duct), cholecystitis (inflammation/infection of the gall bladder), cholangitis (inflammation/infection in the bile ducts)

IV. This patient has ascites. It is likely secondary to chronic hepatitis (C more than B) that has resulted in cirrhosis of the liver. Cirrhosis is the end stage of hepatocyte destruction. Initially, they are infected which if not resolved will cause chronic inflammation. Ultimately, fibrosis and scar will form which results in intrahepatic obstruction. The back pressure is applied to the portal system causing *portal hypertension*.

Portal hypertension will cause dilation of accessory veins in order to bypass the liver and return blood to the IVC (portosystemic shunt). The veins that may become prominent are: inferior hemorrhoidal veins (hemorrhoids), umbilical veins (caput medusa), esophageal plexus (bleeding varices), gastric veins (portal gastropathy) and splenic veins (splenomegaly).

The liver also has other functions. Testosterone production (extraction of androsteinedione) – deficiency leading to testicular

atrophy and gynecomastia, albumin synthesis (edema results from decreased oncotic pressure in vascular system), coagulation factors (easy bruising), etc.

**Review the portal and hepatic circulation of the liver.**

- V. “Pain out of proportion to the exam.” This usually indicates ischemia. This is a “good story” for mesenteric ischemia. This involves the *celiac and superior mesenteric arteries*. An embolus (cardiac arrhythmias, valvular heart disease) or thrombosis (severe peripheral vascular disease/atherosclerosis) put patients at a higher risk. It will be exacerbated following a meal because of shunting to the intestinal circulation for digestion.

One should also consider an abdominal aortic aneurysm in the differential. Both must be acted on quickly since they carry a high mortality rate.

**Review the blood supply to the small and large bowel and the areas of anastomoses. What are Griffith’s and Sudeck’s point and why are they clinically significant?**