Name: Mrs. LLY
Medical Record #: CS6949
Date of Birth: 11/20/1961
Date of Presentation: April 17, 2005, 1:30am
History and Physical Conducted: April 17, 2005 3-5pm
Patient's Room: 9319

INFORMANTS:
1) Patient and husband - good reliability, fair insight
2) Records from Durham Regional Hospital

PATIENT PROFILE:
This is a 43 yo married Malaysian-American female.

CHIEF COMPLAINT:
Abdominal pain, nausea, and vomiting

HISTORY OF PRESENT ILLNESS:
This is a 43 year old Malaysian-American female with a medical history significant for a laparoscopic cholecystectomy in 2002. Since the birth of her second child in 1992, she has had periodic episodes of abdominal pain with or without N/V about every 6-24 months, usually localized to the RUQ. Most of these episodes have resolved spontaneously without medical care, but she has sought medical care on several of these occasions. In August of 2002, she underwent a laparoscopic cholecystectomy. Following this operation, she continued to have periodic abdominal pain, of the same character and at the same frequency as what she had been experiencing before her operation.

She presents with her latest of these episodes. She had been in her usual state of health, until she woke up on Tuesday (4/12) morning with a constant, pinching abdominal pain localized to the right upper and middle upper abdomen. She also felt nauseous, but did not vomit. This pain got worse with intake of food, and seemed to improve after several hours of not eating. By Wednesday morning, her symptoms had mostly resolved, but she continued to feel some mild abdominal discomfort. This mild discomfort continued until Saturday morning, when she began to feel significantly worse. Her abdominal pain increased significantly (10/10) and she began to vomit everything she ate. By Saturday afternoon, she became diaphoretic and her pain continued to increase. She took a Kroger's antacid Saturday evening, but otherwise did not do anything to treat these symptoms herself. She presented to the Duke ED early Sunday morning, where care was initiated and the patient was admitted to the Gen Med 4 service. She denies having chest pain, dyspnea, fevers, chills, back pain, and diarrhea at any point during this episode. Her most recent bowel movement was Saturday evening, and it was dark brown in color.

PAST MEDICAL HISTORY:
1) s/p laparoscopic cholecystectomy, August 2002, DRH, pathology showed chronic cholecystitis

2) Occasional RUQ pain , +/- N/V, every 6-24 months since 1992, as described in HPI
3) Upper endoscopy in October 2002 at DRH for epigastric pain w/ normal LFTs showed mild gastritis, but unable to explain the patient's symptoms
4) Upper endoscopy in Malaysia, date, indication and results not available
5) s/p tubal ligation, 1992
6) biopsy of endocervical polyp in 2003, showed benign columnar endocervical epithelium

CURRENT MEDICATIONS:
Patient does not take any regular medication. She does take multivitamins, vitamin C, and calcium supplements "on and off." She took one tablet of Kroger's antacid Saturday evening, but she does not take antacid regularly.

ALLERGIES/SENSITIVITIES:
NKDA

SOCIAL HISTORY:
The patient was born in Malaysia, and lived there until her early 20s, when she moved to the United States with her husband. She grew up in a semi-rural area she frequently swam in the rivers. Her native language is Hokkien. She is not aware of any gastrointestinal or other diseases that are endemic to the area she is from. She currently lives in Durham with her husband and their two children, all of whom are healthy. They own and manage a Chinese restaurant called "Shanghai." Her aunt (Fong) also lives in Durham. She has no pets, has not eaten any new foods recently, and has no recent sick contacts. Her most recent travel was to Malaysia last summer. She has never used alcohol, tobacco, or any other drugs

HEALTH MAINENANCE:
The patient's PCP is Dr. Mantoch. Further details regarding her health maintenance were not obtained.

FAMILY HISTORY:
The patient's mother has diabetes and hypertension, but is otherwise healthy. Her father is healthy and has no medical problems that she knows of. She does not know of any diseases that run in the family, gastrointestinal or otherwise.

REVIEW OF SYSTEMS:
General – The patient denies fevers, chills, fatigue, and decreased appetite. She had not had trouble sleeping until last night, when she could not sleep at all due to her nausea and abdominal pain. She was able to maintain good PO intake until Sautrday morning when she began to vomit.

Skin – Patient denies rashes and skin lesions anywhere on her body.

HEENT Eyes – Denies blurry vision, double vision, and any changes in visual acuity. She does wear reading glasses.

Ears – Denies changes or problems in her hearing.
Nose/throat/mouth/teeth – Denies congestion, rhinorhea, sore throat, and dental pain. She has had some seasonal allergies over past several weeks.

Respiratory – Denies dyspnea and cough.

Cardiovascular – Denies chest pain, palpitations, pericheral edema.

Breasts – Denies changes, pain, or masses in breasts.

Gastrointestinal – Patient has diarrhea and constipation. She has had significant abdominal pain, nausea, and vomiting as described above. Denies bloody or tarry stool.

Genito-Urinary – Denies dysuria, polyuria, and hematuria. Last menstrual period started 2 days ago. Does not take oral contraceptives or HRT.

Neurologic – Patient reports occasional headache, the most recent of which was yesterday. She denies hx of seizures, paralysis, weakness, and neurological problems in general.

Musculoskeletal – Denies muscle and joint pain.

Endocrine – Denies diabetes.

Hematopoetic – Denies easy bruising and bleeding.

PHYSICAL EXAMINATION:

Vital Signs – temp 36.4°C, BP 112/60, pulse 70, RR 20

General – Pleasant 43 year old female currently in significant distress due to abdominal pain. Patient is quiet and mildly drowsy. She has been vomiting while in ED.

Skin – No lesions or rashes were noted on the face, neck, upper extremities, back, chest, or abdomen.

Lymph nodes – No periauricular, cervical, supraclavicular, axillary lymphadenectomy.

HEENT – No scleral or sublingual icterus. Oropharynx was clear and mucosa was moist. Dentition mostly present.

Neck – Trachea midline. Thyroid gland normal and nontender. No carotid bruits. No JVD.

Chest – Chest symmetric. Lung clear to auscultation bilaterally.

Heart – Normal S1 and S2. A 2/4 diastolic murmur is present, best heard at the left upper sternal border. Murmur does not radiate into neck. PMI normal.

Abdomen – Extreme tenderness in RUQ. Other quadrants only mildly tender. Murphy's sign absent. Nondistended. Normal bowel sounds. Small scar in middle upper abdomen consistent with surgical history. No organomegally noted.

Rectal – Adequate tone, no masses noted, guiac negative.

Extremities Peripheral pulses – 2+ radial, pedis dorsalis, and posterior tibial pulses bilaterally.
Peripheral edema – No edema in lower extremities.

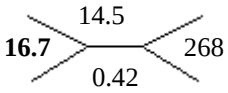
Musculoskeletal – Normal passive and active ROM in upper and lower extremities. No focal joint inflammation or abnormalities were noted.

Nervous System Mental status – She is alert and oriented x 3.
Cranial nerves – 2-12 intact.
Sensory – Light touch intact to face, UEs, and LEs bilaterally.
Motor – 5/5 strength in upper and lower extremities.
Extremities – Patellar reflex 2+ bilaterally.

LABORATORY DATA:

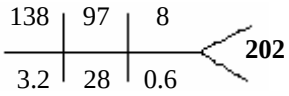
On Presentation to ED (1:44 am):

Hematology



Differential: **92.0% neutrophils** (**15.3 total**), **5.0% lymphocytes** (0.8 total), 2.9% monocytes, 0.0% eosynophils, 0.1% basophils

Chem 7



GI – 0.7 | 58
78 | 37

Other Chemistry - Ca 9.0, Phos 3.4, **total protein 8.5,** albumin 4.4, lipase 31, amylase 31

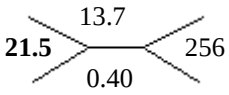
Urinalysis – turbid yellow, specific gravity 1.023, **1+ protein**, **2+ glucose**, **3+ ketone**, **1+ blood**, **20 rbcs**, **8 wbcs**, 0.2 urbilinogen, 4 squamous epithelial cells, 3 hyaline casts, 0-5 bacteria, negative for nitrite, negative for leukocyte esterase, negative for bilirubin

Coagulation Studies – INR 1.1, PTT 27.0

Accuchecks – 178 at 1:49am; 116 at 4:45 pm

8 Hours Later (9:45am):

Hematology



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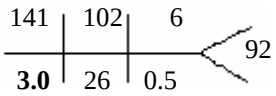

DIAGNOSTIC TESTS:

Chest X-ray, PA and lateral views - The lungs, heart, mediastinum, hila and pleura are normal. Small calcification just below the right hemidiaphragm.

RUQ Ultrasound - stone in CBD (1.8x1.1x1.7 cm), stone in left hepatic duct (1.5x1.5x1.0 cm), CBD dilation of 1.7 cm. Dilation of unspecified size upstream of left hepatic stone.

ASSESSMENT/PLAN:

1) Acute Cholangitis

This patient has choledocolithiasis, with at least two stones present in the biliary tree – one in the common bile duct and one in the left hepatic duct. She also has several signs of acute infection of the biliary tree, including an elevated white blood cell count with a left shift, fever, and RUQ pain and tenderness.

The notably absent signs of acute cholangitis are jaundice, bilirubinemia, and elevated alkaline phosphatase. The absence of these signs does not rule out acute cholangitis, and neither does the fact that only two out of three of the signs in Charcot's Triad are present. In fact, Charcot's Triad is only present in 50 to 75 percent of patients with acute cholangitis (Am J Surg 1975; 130:143). The absence of jaundice, bilirubinemia, and evlevated alkaline phosphatase suggests that either (1) the common bile duct is not completely obstructed, but is just causing sufficient obstruction to allow for stasis and an ascending bacterial infection, or (2) the stone in the common bile duct is not causing stasis at all, and the patient's symptoms are caused entirely by the stone in the left hepatic duct. If only the left hepatic duct is blocked, these signs of stasis would be absent because drainage of bile down the right hepatic duct into the common hepatic duct and eventually into the duodenum would be unobstructed, and bilirubin could be cleared from the blood entirely by this route. It is also known that this patient's biliary obstruction does not involve her pancreas, since she has normal serum lipase and amylase levels.

The diagnosis of acute cholangitis is also strengthened by the fact that acute cholecystitis, which would otherwise be a leading differential diagnosis in this clinical picture, is impossible since this patient's gallbladder is surgically absent. This diagnosis

of acute cholangitis will be confirmed by endoscopic retrograde cholangiopancreatography (ERCP).

Initial management of this patient is to treat her ascending infectious cholangitis with Unasyn (3 g IV q6h), and to fluid resuscitate her. Unasyn is an appropriate choice of antibiotics because it covers the leading seven species associated with nonmalignant cholangitis - Escherichia coli 43%, Klebsiella species 31%, Enterococcus 36%, Streptococcus species 24%, Bacteroides species 17%, and Enterobacter species 17%, and Citrobacter species 17%) (Surg Gynecol Obstet 171:275-282, 1990). Unasyn does not cover the eighth leading cause of nonmalignant cholangitis, which is Pseudomonas species (12%). If the patient had appeared to be septic at presentation (i.e. if she was hypotensive) or if her condition worsens once antibiotic therapy is initiated, providing coverage of Pseudomonas with an agent such as Zosyn (or Cipro and Flagyl) would be considered.

More definitive treatment of the patient's biliary obstruction will be sought at the time of ERCP. At this time, she will likely undergo a sphincterotomy, extraction of the CBD stone, and stent placement. It is unlikely that the stone in the left bile duct will be able to be extracted at the time of ERCP. If this stone does not pass spontaneously and the patient's symptoms persist, it is likely that shock wave lithotripsy or surgical removal or the stone in the left hepatic duct will become necessary. The patient is being kept NPO and will not be kept off of heparin or NSAIDS to prevent a GI bleed from being initiated by the ERCP.

In addition to treating her acute cholecystitis, this patient gives a history that suggests that she has subclinical, but still significantly symptomatic cases of acute cholangitis about once a year. Even though these cases usually resolve spontaneously (presumably by spontaneous passage of the gall stones) it is important to find the underlying cause that predisposes her to frequent choledocolithiasis. Choledocolithiasis generally does no occur in patients without gallbladders, so potential causes of stasis that could allow for a stone to nucleate and grow within the biliary tree will be evaluated.

Such potential causes of stasis include cholengiocarcinoma, Mirizzi's syndrome, Caroli's disease, biliary strictures, and oriental cholangiohepatitis. The patent's fasting serum cholesterol level will also be taken, because extreme hyperlipidemia can also predispose to frequent choledocolithiasis, so this must be ruled out as a cause of this patient's frequent cholangitis.

Given this patient's history of growing up in Southeast Asia (Malaysia), oriental cholangiohepatitis is a very likely cause of her frequent choledocolithiasis. After removal of the stone in the patient's CBD, a retrograde colangiogram should be taken. If this cholangiogram shows a strictured and/or fistulized CBD, the diagnosis of oriental cholangiahepatitis would be almost certain. Further supporting this diagnosis is the location of the stone in the patients left hepatic duct, as is frequently seen in patients with oriental cholangiohepatitis.

If it is found that this patient does have oriental cholangiohepatitis, as is expected, the patient may desire an attempt at more definitive management by resection of the affected portion of the biliary tree. If surgical intervention is undertaken, the patient should also undergo a preoperative CT scan to evaluate for the possible benefit of left hepatic lobectomy. If she does not wish to undergo such an operation, regular surveillance with periodic removal of any stone is another successful management

strategy. Finally, if analysis of the recovered stones show them to be composed of a large component of cholesterol (which is unlikely since cholesterol stones are typically radiolucent, and the at least one of this patient's stones appears to be radioopaque, since it was seen on her chest radiograph) she was benefit from chronic ursodeoxycholic acid (UCDA) which is thought to have some efficacy in preventing the formation of cholesterol stones. This is also unlikely because most stones seen in patients with oriental cholangiohepatitis are composed of calcium bilirubinate.

2) Pain Management

This patient was experiencing 10/10 pain at the time of her presentation, and it is important to treat this pain so that she is as comfortable as possible. Morphine and toradol gave her little relief in ED. Further, morphine is thought to cause contraction of the sphincter of Odis, which could worsen this patient's symptoms and make ERCP more difficult. Her pain will therefore be treated with Dilaudid, 1mg IV q4h prn.

3) Hypokalemia

This patient has a mild hypokalemia, which is most likely caused by the transcellular exchange of K^+ for H^+ to compensate for the non-anion gap metabolic alkalosis caused by vomiting. We will give patient NS with 20 meq KCl to replace this K^+.

4) Hyperglycemia

The patient was initially hyperglycemia on presentation, and has no history of diabetes. Her hypoglycemia is most likely a result of the general inflammatory state associated with her ascending cholangitis. It is also possible that she has previously undiagnosed diabetes. Her blood sugar became significant better controlled (at 116 and 92) after being given IVFs and Unasyn. This supports the theory that this patient's hyperglycemia was being caused by her infection. Acute management of her hyperglycemia will be to monitor it with q6h Accuchecks and to treat it with sliding scale insulin as needed. Control of blood sugar is especially important in the setting of an infection since hyperglycemia can promote infection and worsen sepsis when it exists. We will also measure a hemoglobin A1C to rule out diabetes in this patient.

5) Abnormal Urinalysis

This patient's urinalysis showed 1+ protein, 2+ glucose, 3+ ketone, 1+ blood, 20 rbcs, and 8 wbcs. The glucose found in this patient's urine was somewhat surprising since glucose is generally not found in urine until the plasma glucose exceeds 300, but this patient's glucose was measured to be 256 on Chem7. The glucose found in her urine is best explained by the fact that her blood glucose may have been higher at some other point in time, such as immediately after she ate. Simliarly, the ketones found were somewhat surprising, but it is very likely that she had a mild ketosis as a result of her hyperglycemia. Having no eaten for several days probably lowered the glucose threshold above which ketones are produced.

The blood is likely present in her urine since her patient is currently at the end of her menstrual period, and her urine sample could have easily been contaminated with some menstrual bleeding. There is no obvious explanation for the wbcs or protein found in this patient's urine, unless she has a UTI or some other kidney disease. The patient

does not currently have any urinary symptoms, and her creatinine is 0.6, so these abnormalities on urinalysis or not very concerning. Will repeat this urinalysis, and consider further workup if abnormalities persist.

6) Health Maintenance
Will request medical records from PCP in morning during business hours.

7) Prophylaxis
GI – 40 mg PO daily
DVT – SCDs in place to LEs bilaterally

8) Code status
This was not discussed with the patient, so she is a full code by default.

9) Disposition
This patient will be discharged once the obstructions in her common bile duct have been addressed, the underlying cause of her choledocolithiasis has been investigated, and her symptoms have resolved. We anticipate this will take 2-4 days, but it may be longer if ERCP is unsuccessful

KF