



Introduction:

Welcome to CUGH's bi-weekly clinical case-series, "Reasoning without Resources," by Prof. Gerald Paccione of the Albert Einstein College of Medicine. These teaching cases are based on Prof. Paccione's decades of teaching experience on the medical wards of Kisoro District Hospital in Uganda. They are designed for those practicing in low resource settings, Medicine and Family Medicine residents, and senior medical students interested in clinical global health. Each case is presented in two parts. First comes a case vignette (presenting symptoms, history, basic lab and physical exam findings) along with 6-10 discussion questions that direct clinical reasoning and/or highlight diagnostic issues. Two weeks later CUGH will post detailed instructors notes for the case along with a new case vignette. For a more detailed overview to this case-series and the teaching philosophy behind it, see [Introduction to "Reasoning without Resources"](#). Comments or question may be sent to Prof. Paccione at: gpaccion@montefiore.org

Note: If you would like to be notified when a new case is posted (along with instructor notes for the previous one), send your e-mail to Jillian Morgan at jmorgan@CUGH.org.

About the Author:

Dr. Gerald Paccione is a Professor of Clinical Medicine at the Albert Einstein College of Medicine in the Bronx, New York. His career has centered on medical education for the past 35 years – as a residency Program Director in Primary Care and Social Internal Medicine at Montefiore Hospital, and director of the Global Health Education Alliance at the school. He has served on the Boards of Directors of Doctors for Global Health, Doctors of the World USA, and the Global Health Education Consortium. Dr. Paccione spends about 3 months a year in Uganda working on the Medicine wards of Kisoro District Hospital where he draws examples for the case studies.

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CASE 22 – ABDOMINAL PAIN BACK IN TIME

55 year old woman from Kisoro town who has been told of “high sugar” in the past, presents with fever, increasing right upper quadrant (RUQ) pain for 3 weeks, and frequent urination.

Otherwise healthy without any weight loss or chronic symptoms, her problem started about 5 weeks ago with pain in the center of her belly that developed progressively over a half hour accompanied by nausea, anorexia and severe constipation. After many hours the pain moved to the right lower abdomen with severe tenderness and some vomiting (twice), without diarrhea. Her husband bought antimalarial drugs and “antibiotics” and she took both without vomiting, finishing 5 days of antibiotics. The pain in her right lower abdomen lessened but persisted. She was too weak to dig. She began to urinate a lot and drink a lot of water.

She first noticed pain, constant and progressive, in the RUQ under her ribs about a week later (3 weeks ago), with increasing fevers and has lost considerable weight. She has no appetite; hasn't vomited again, has no diarrhea, but often feels nauseated. Over the past few days she has noticed her urine is darker than usual. She and her husband are monogamous, and she hasn't noted vaginal discharge or bleeding. She knows of no others with a similar problem.

P.E. Looks older than age, and uncomfortable but in no acute distress

BP 100/70 → 80/70; 110 → 140; T 102 R 22, not deep

conjunctiva: icteric;

mouth: dry mucous membranes, no thrush

neck: no JVP lying flat; no HJR; no lymphadenopathy

lungs: clear

heart: S₁ S₂ Gr 1/6 SEM

abdomen: RLQ: firm; tender, cylindrical “mass” 7 x 3cm

RUQ: liver ↓ 2 cm, span 13 cm; edge non-tender but winces with gentle punch over the RUQ

rectal: no masses, guaiac negative brown stool

pelvic: no discharge, general discomfort but no cervical motion tenderness, right adnexal fullness/tenderness;

extremities and neurologic exam: normal

U/A: s.g. 1.025, +4 glucose, +3 bili, +3 urobilinogen, (-) protein, trace ketones, (-) leuk. esterase, (-) nitrates

- 1. What is the “frame” of this case (i.e. the key clinical features that the final diagnosis must be consistent with)?
What are the clinical implications of each feature of the frame re-disease process?**

- *older diabetic woman*
[Diabetics are more prone to complications of most infections, particularly abscess formation]
- *5 weeks of illness, with fever, anorexia and weight loss, in no acute distress*
[These are the tempo and symptoms of a chronic infection, e.g. one that produces granulomatous pathology or is walled off by fibrous tissue.]
- *polyuria with diabetes out of control (+4 glucosuria), non-acidotic*
[Suggests underlying inflammation causing diabetes to get out of control, consistent with chronic infection. The trace ketones in the urine and the lack of Kussmaul respirations suggests that ketoacidosis is not present in this type II diabetic. The slight ketonuria is probably due to starvation ketosis.]

- *initial focal symptoms in right lower abdomen, with partial symptom response to antibiotics*
[The focal symptoms with fever suggest a bacterial infection (over viral or parasitic), as does the response to antibiotics. The RLQ focus suggests appendicitis, ileitis, or (unusually) diverticulitis.]
- *later, right upper quadrant pain with punch tenderness and jaundice*
[Signs of liver involvement by an inflammatory process]
- *2 foci of abdominal tenderness, one, with tender mass in RLQ, and the other, the RUQ, the liver*
[Suggests extension of bacterial infection along tissue planes or via lymph or blood from right lower quadrant to liver.]

2. What is the differential diagnosis of the RUQ pain in this case? What are the clinical “pros and cons” of each of the possibilities, and what is the most likely diagnosis?

- *Viral hepatitis: Viral hepatitis is suggested by symptoms of RUQ discomfort with tenderness, fever and jaundice, preceded by constitutional symptoms like anorexia. Hepatitis would also provoke subclinical diabetes to get out of control. However, symptomatic viral A hepatitis is uncommon in Africa as most are exposed and develop immunity when young, this patient has no risk factors for acquisition of hepatitis B or C, hepatitis C doesn't present with sub-acute progressive symptoms, and hepatitis E is often epidemic. More importantly, viral hepatitis would not explain the early and persistent symptoms of RLQ pain nor mass and tenderness on exam. The height of the fever would also be very atypical for viral hepatitis.*
- *Hepatocellular carcinoma (HCC): HCC is prevalent in Africa primarily as a complication of chronic hepatitis B and also hepatitis C. The prevalence of hepatitis B is markedly higher in Africa than in North America, often acquired in utero or as a child. HCC is 30-100 times more common in Africa than in the West; can cause RUQ tenderness via invasion of the hepatic capsule; and fever. However, with HCC the symptoms are usually more insidious in onset and evolve over a longer period of time than in this patient, fever is unusual (though seen), the liver is hard and nodular on exam. Most importantly, HCC would not explain the early and persistent symptoms of RLQ pain, nor mass and tenderness on exam.*
- *Hydatid cyst disease (Echinococcus granulosus): Hydatid cysts, caused by the larval stage of the echinococcus (cestode) parasite in humans, are prevalent as*

a cause of both liver and lung cysts in areas where livestock are raised in association with dogs.

The larval cysts enlarge slowly over many years without inducing an inflammatory response and often remain asymptomatic. Symptoms, when they do occur, do so because of their size and location: in the liver, insidious RUQ pain and mass, nausea, occasionally biliary obstruction and jaundice. The most serious complication is cyst rupture into the abdominal cavity or lung, often induced by trauma, which can produce anaphylaxis and shock.

Cysts can rarely become secondarily infected, and if this patient came from a nomadic tribe living close to its dogs like the NW Kenyan Turkana or NE Ugandan Karamajong, a bacterial infection of a hydatid cyst would be a plausible diagnostic possibility for the RUQ pain. However, such a complication is very rare, she does not come from such a hyperendemic area, and again, a secondarily infected liver cyst does not explain the right lower quadrant pain and mass or the evolution of symptoms.

- *Amebic Liver Abscess: E. histolytica is an invasive, aggressive protozoan that spreads through feces-contaminated food and water, resides in the colon, and can cause either dysentery or be relatively asymptomatic with occasional symptoms of alternating diarrhea and constipation. Through the portal circulation the amebic trophozoite can also travel to the liver where it kills hepatocytes on contact inducing liquefying necrosis (“anchovy paste” without cells) and hepatic abscess formation. It’s an extremely common cause of hepatic abscess in areas of poor sanitation world-wide.*

Clinically, the symptoms of amebic hepatic abscess usually evolve over 1-2 weeks before presentation, but in 20-50% it’s more than 4 weeks (up to even a year) before the patient seeks medical attention. RUQ pain and tenderness are seen in 85-90% as is fever, often >102F and spiking, and hepatomegaly is seen in 30-50% of cases.

One of the rare complications of amebic colitis is an “ameboma”, a mass of granulation tissue reacting to and walling off amebic infection of the colon, often presenting years after a bout of dysentery with months of non-specific abdominal discomfort with alternating constipation-diarrhea and weight loss, and/or obstructive symptoms mimicking colon cancer.

Ameboma often presents in the cecum or ascending colon and can be associated (rarely) with a coexisting amebic abscess of the liver, which would fit the bi-focal presentation in this patient.

However, unlike this patient, males usually 20-40 years old predominate 10:1 in series of patients with hepatic abscess (for unclear reasons) and jaundice is uncommon (5-30%) of cases. Furthermore, ameboma is rare, occurring in fewer than 1.5% of patients with intestinal amebiasis, and the near simultaneous presentation of symptomatic ameboma and hepatic abscess

would be extremely rare. But most importantly, a close look at the patient's presentation rules out ameboma on clinical grounds: Without any prior symptoms of chronic abdominal disease, the patient experienced mid-epigastric pain that moved over hours to the RLQ - without any diarrhea, but with vomiting, constipation and persistent pain. Although anatomically the (rare) chronic complication of ameboma combined with an amebic liver abscess is conceivable, their simultaneous presentations makes the clinical likelihood remote: clinically it doesn't fit.

- **Abdominal Tuberculosis:** Abdominal TB must be considered in every patient in Africa with chronic abdominal pain, fever, and constitutional symptoms. Consistent with this patient's presentation, TB often involves the ileum and/or cecum with right lower quadrant pain, tenderness, constipation and a palpable mass (which could be either hyperplastic granulomatous cecal tissue or a matted bunch of nodes). However, the RUQ findings would be quite unusual for TB: TB in the liver is most often asymptomatic, induces hepatic micro-granulomas without jaundice, and the RUQ symptom evolution is too marked and rapid for TB.

- **Pyogenic Liver Abscess with preceding Appendicitis:** This 55 year old diabetic patient had a typical presentation of a disease that is common world-over, but which causes significant mortality in Africa due to the lack of access to care and surgeons: appendicitis.

Although less common in rural African populations than in whites, appendectomies are the most common emergency abdominal surgeries performed in Africa. However, the surgeries are more complicated: 20-40% of patients have peritonitis and/or abscess at presentation due to delay in accessing care and consequent perforation. Occasionally, parasitic migration or infestation (e.g. ascaris, schistosomiasis) leads to appendiceal obstruction and infection.

This patient developed mid-epigastric pain initially which moved to the RLQ as the inflammation from the appendix reached the parietal peritoneum. Nausea, intermittent vomiting, and a sensation of constipation are classic accompaniments of appendicitis. She tolerated oral antibiotics (?type) which probably helped prevent the spread of the infection but were incapable of curing the disease and a walled-off abscess developed in the RLQ leading to her persistent pain, diabetes out of control, and weight loss. (Although appropriate IV antibiotics might well have treated appendicitis, the recurrence rate is >30% within a year.)

A hepatic abscess then developed as mixed aerobic/anaerobic bacteria entered the portal system and spread to the liver from the intra-abdominal focus. (Nowadays in the West, the most common abdominal source of hepatic abscess

is diverticulitis, which is rare in Africa, but it used to be appendicitis, with delayed treatment, in the West as well.) Thus the pain shifted to the right upper quadrant, the liver became tender, and jaundice developed.

In addition to intra-abdominal foci of infection and spread via the portal circulation, biliary disease (cholecystitis, cholangitis sometimes caused by ascaris obstructing the cystic and common bile ducts), sepsis, and penetrating trauma are all underlying causes of pyogenic liver abscess, although up to 40% are idiopathic.

As in this case, patients with pyogenic abscess are most frequently older than 50, and have co-morbidities such as diabetes. They're more often sicker on presentation than patients with amebic abscess, with jaundice and signs of sepsis and shock. (The jaundice can be multifactorial, caused both by anatomic obstruction of focal biliary ducts and chemical interference with excretion of conjugated bilirubin by bacterial endotoxin and cytokines, the "hyperbilirubinemia of sepsis".) Most patients with pyogenic abscess have multiple abscesses in the liver, whereas amebic abscess is most frequently single – but the overlap in clinical presentation between the two makes differentiation on clinical grounds alone hazardous.

3. How would you manage this patient?

This very ill patient, with an intra-abdominal abscess complicated by probably multiple hepatic abscesses needs:

- *Antibiotics, IV, broad-spectrum, which cover aerobic (Klebsiella, E. coli) and anaerobic intestinal flora: metronidazole (which would cover ameba as well) plus either a fluoroquinolone or 3rd generation cephalosporin (or if not possible, a 1st-2nd generation cephalosporin with an aminoglycoside).*
- *Insulin management of diabetes, with rapid infusion of IV fluids to preserve cardiovascular and renal function;*
- *After stabilization, an USG, locally if available or referral to a center with USG capability, to both assist diagnosis and to guide percutaneous drainage of the likely hepatic abscess(es) and surgery for the likely appendiceal abscess. (Pyogenic abscesses are ideally drained, but many (probably <50%) can be treated medically.) In this high risk diabetic, drainage is likely to reduce her risk of mortality with medical treatment alone.*

Suggested Readings:

Johannsen, E.C., Madoff, L.C. Infections of the Liver and Biliary System in Principles and Practice of Infectious Diseases, Mandell, G, Bennet, J., Dolin, R. 6th Ed. 2005, Ch.69: 951
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Seeto RK, Rockey DC. Pyogenic liver abscess: changes in etiology,
management, and outcome. *Medicine* 1996;75:99 –113.