



Introduction:

Welcome to the 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 4-10 discussion questions that direct clinical reasoning and/or highlight diagnostic issues. A month 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

About the Author:

I’m a Professor of Clinical Medicine at the Albert Einstein College of Medicine in the Bronx, New York, where my career has centered on medical education for the past 40 years – as a past residency Program Director in Primary Care and Social Internal Medicine at Montefiore Hospital, and global health advisor and program leader at the school. I’ve served on the Boards of Directors of Doctors for Global Health, Doctors of the World USA, and the Global Health Education Consortium. I spend about 3-4 months a year in Uganda working on the Medicine wards of Kisoro District Hospital which, like most hospitals in the world that serve most of the world's population, has (almost) no resources. "At the bedside", I teach Internal Medicine residents and medical students how to assimilate the elements of history, physical exam and epidemiologic probability into a diagnostic impression that, even without definitive testing, can lead to appropriate therapeutic strategies in the field.

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Case 57: Vomiting Forever

A 70 year old man is carried to a bed on the ward unable to walk for 2 days after some diarrhea and an increase in his usual vomiting over the past week.

He has had a long history of abdominal pain since the age of about 20 years old, often waking him from sleep at about 3 AM especially when he was younger, and relieved by food or drink. The pain has been a continual problem throughout his life, with some months better than others.

He has been hospitalized at least 10 times that he can recall for pain and more recently for vomiting which has also waxed and waned in severity (but never disappeared) over the years. The vomiting became a very significant problem 18 years ago and has been bad ever since. He usually vomits about 4-8 hours after eating, three or 4 times a week and often recurrently through the night, and it relieves his abdominal discomfort. The vomitus is most often clear-yellow with undigested food, without blood or mucous. Eighteen years ago, because of inability to keep food down, he lost a tremendous amount of weight, became very weak and had to stop working in the fields. He hasn't worked since, has been "very thin" and walks slowly with a cane. He doesn't drink or take drugs.

At another hospital, he was diagnosed with a "stomach allergy", and another time with "ulcers" for which he's received treatment (?drugs) in the hospital. He usually feels somewhat better after treatment (but never normal) and goes home - until the next *severe* exacerbation in 2-6 months. This has been a particularly bad year with severe bouts of vomiting coming on more frequently and lasting longer. He has been hospitalized 3 times in the past 6 months for periods of days. Surgery has never been recommended to him (that he recalls).

About a week ago, he developed diarrhea, 3-4 times a day - watery, without blood, mucous, or fever. At the same time his vomiting became worse, and he couldn't keep anything down. Two days ago, he was too weak to walk. He can't remember urinating in the past 2 days.

He has had no fevers, cough, history of diabetes, problems with urinating or constipation, dizziness upon standing (except when "very sick"), or other symptoms. He was HIV-tested a year ago, negative. He is fully cared for by his wife and 3 older children who "dig" in the fields.

Physical Exam: Extremely emaciated/cachectic man, "skin-and-bones", unable to stand

BP: 75/60 lying, undetectable sitting; HR 115 lying, 135 sitting; RR 12; T: 96.2

HEENT: prominent bones without soft tissue; eyes: sunken, conjunctiva/sclera, no icterus or pallor

Mouth: no thrush; poor dentition

Neck: no neck veins detectable lying flat, detectable with legs passively elevated; no lymphadenopathy; no thyroid palpable

Lungs: clear

Heart: tachycardic, normal S1, S2; Gr1/6 systolic murmur at base without radiation

Abdomen: no hepato-splenomegaly, masses, tenderness;

Rectal: scant stool, liquid, guaiac negative; prostate not boggy/tender, mildly enlarged, normal contour;

Neuro: mental status intact; Cranial nerves, sensation, cerebellar testing intact

diffuse muscle weakness, 4/5 throughout; no twitching seen.

absent ankle jerks; unable to walk;

1. What is the *frame* of this case from the history and physical exam (i.e. the key clinical features the final diagnosis must be consistent with)?

- *Timing of the illness: chronic pain, later chronic vomiting*
 - *Elderly man, abdominal pain/vomiting for 50 years, since early adulthood*
 - *When problem first began, pain would wake him from sleep, relieved by food*
 - *Intermittent, waxing and waning but never gone, requiring hospitalizations over time*
 - *Much worse for past 20 years when vomiting became the dominant problem*
- *Timing/character of the vomiting:*
 - *4-7 hours after meals*
 - *undigested food, no blood*
- *Extreme cachexia; Inability to walk*
- *Diarrhea/increased vomiting for a week*
- *No fevers or other localizing symptoms or signs*
- *Hypotension with orthostasis*

2. What is the *clinical significance* of each of the features selected for “*frame*”?

- *Timing of the illness: The waxing and waning for 50 years “rules out” obstructing malignancies as well as endocrine/toxin/drug disorders that induce vomiting and pain (e.g. DKA, hypercalcemia, uremia, drug side effects) as the etiology of the chronic problem. It suggests either a “benign” cause of intermittent obstruction, or a motility disorder of the gut (intrinsic, neuropathic, or psychologically mediated). That he’s never been symptom-free in his adult life decreases the likelihood of a motility disorder.*
 - *Worse for past 20 years, with vomiting as the main problem: suggests either a worsening of the underlying process, or a (new) complication of that process.*
- *Timing/character of the vomiting: 4-7 hours after meals suggests gastric outlet obstruction, the delay of hours being due to slow breakdown of food in the stomach causing an increase in content osmolarity, gradual osmotic draw of fluid, gastric expansion, and late induction of the emetic reflex.*
 - *undigested food, no blood: again suggests retained food that hasn’t passed far into the digestive system;*
- *Extreme cachexia; inability to walk: the man presents with the picture of end-stage chronic starvation - due to lack of food access to the digestive system, not food malabsorption or hyper-catabolism. His emaciation illustrates the severity of the disease and suggests that any (even mild) “acute process” could be enough to “tip him over” and result in his present bed-bound condition.*
- *Diarrhea/increased vomiting for a week;*
No fevers or other localizing symptoms or signs;
Diarrhea and worsened vomiting for a week, without signs of dysentery, or infection/sepsis elsewhere infer a mild gastroenteritis – but enough to “tip him over”.

3. a) What are the likely *treatable* reasons for this patient's inability to stand/walk?
b) What *immediate* therapy is indicated on admission?
c) What *additional exam maneuvers and tests* (available in a district hospital) are appropriate?

- the patient is severely volume depleted: hypotensive, tachycardic, orthostatic, without visible JVP lying supine (but veins visible with passive leg elevation); much of his acute inability to stand is due to volume depletion compounding chronic starvation.

Along with the volume depletion come (probably severe) electrolyte disturbances which can contribute to the muscular weakness and inability to stand/walk:

- hyponatremia (which can contribute to nausea/vomiting too),
 - hypokalemia (from renal losses of potassium from high compensatory aldosterone levels)
 - probable alkalosis despite the recent diarrhea (which would cause loss of bicarbonate and tend towards a non-anion gap acidosis. However, the diarrhea was mild compared to the vomiting.)
 - hypophosphatemia: if the alkalosis were severe, hypophosphatemia could be another secondarily-induced electrolyte imbalance contributing to muscle weakness.
 - uremia: he's also likely to be in (prerenal) acute renal failure (ARF), and uremia, if present, can contribute to both his weakness and his vomiting.
- immediate therapy should include IV fluid, the first 500 cc of normal saline being given as a fluid challenge over 10-15 minutes to see if BP rises and heart rate falls as expected in volume depletion. (N.B. The alternative explanation for the hypotension, weakness and lack of urine for 2 days is septic shock with acute renal failure due to acute tubular necrosis. Given his chronic malnutrition, the patient is a "set-up" for sepsis, and the cytokines released in sepsis can cause all of his symptoms including diarrhea and vomiting. If it is septic shock causing this picture, his BP should not respond to fluid, and if ATN he shouldn't start urinating.)

If his BP and HR respond, fluids should be continued vigorously, a liter an hour for the next 2-3 hours, with potassium. If he has received 3-4 liters without urinating despite hemodynamic stabilization and appearance of neck veins, consider ATN due to volume-induced hypotension or sepsis. If no improvement in vital signs with fluid or no urine after the above volume repletion, decrease normal saline to 150-200 cc/hour for another 2 liters, hold additional IV potassium and give empiric broad-spectrum antibiotics.

- The additional exam maneuvers, after hemodynamic stabilization, are two:

a) a "succussion splash" should be elicited to test for gastric outlet obstruction: rock the abdomen back and forth repetitively (at least 2-3 hours after a meal or ingestion of any fluid), while listening with the stethoscope over the stomach. When the rocking abruptly stops, if the sloshing to-and-fro-sound of a fluid wave is still heard, the "succussion splash" is positive. (N.B. if the patient has not vomited);

b) test the autonomic nervous system. As noted above, based on the chronicity of the vomiting, a dysmotility disorder is in the differential and one mechanism behind dysmotility is an autonomic neuropathy affecting the vagus nerve. Since the vagus innervates the heart as well as the gut, vagal neuropathies are diagnosed by reflex heart rate responses to hemodynamic provocations induced at

the bedside, measured by (a running) EKG. Two such autonomic maneuvers are the Valsalva maneuver, and deep breathing:

Valsalva: EKG is recorded continuously before, during and after (for 20 seconds) patient blows into the end of a detached BP cuff raising manometer pressure to 40 mm for 10 seconds; the ratio of the longest RR interval (post-Valsalva “overshoot”) to the shortest RR (at end of strain phase) with this maneuver is normally >1.20; <1.10 indicates impaired baroreceptor reflex and probable autonomic neuropathy;

Deep Breathing Cycles: the ratio of the heart rate changes between expiration and inspiration while breathing slowly at 10 seconds per cycle for a minute: with an intact baroreceptor-vagal reflex, the ratio of the fastest to slowest HRs should be >1.03.

Tests:

- *Urinalysis (on first passage of urine): to check both specific gravity (s.g.) re-history of no urine (high s.g. supports pre-renal) and sediment (muddy brown casts suggest ATN)*
- *EKG: for signs of electrolyte imbalances such as hypokalemia;*
- *Hb/Hct: to assess nutritional anemias from chronic starvation and/or blood loss; if anemic, check blood smear for morphology;*
- *if electrolytes are available, all electrolytes mentioned above would be germane to his acute management;*
- *if a Barium swallow and UGI series are available with repeat films over the next 48 hours, the results would be potentially diagnostic.*

Results:

After 500 cc of normal saline over 15 minutes his SBP rose from 75 to 85 and his HR dropped from 115 to 100, indicative of volume depletion and not septic shock.

After 3 liters of fluid (about 3 hours later) he produced urine:

Urinalysis: first urine was yellow, with a specific gravity of 1.025 and a pH of 6.5 (likely “paradoxically aciduric” i.e. despite systemic alkalemia, the urine is acidotic. This is because much-needed sodium is being resorbed with filtered bicarbonate, the only anion available in lieu of chloride (lost in the vomitus), and hydrogen is being excreted, maintaining the alkalosis; all this will be corrected by giving IV NaCl.) There were no casts seen, nor WBCs/RBCs.

The results suggest that there’s no ATN, and the patient’s anuria for 2 days by history was due to extreme volume depletion.

Abdominal exam, succussion splash: No “succussion splash” on rocking the abdomen back and forth repetitively hours after consuming a meal and fluid;

Autonomic testing, performed after hemodynamic stability was reached, revealed a normal baroreceptor axis.

- The Valsalva maneuver: longest RR/shortest RR (EKG interval), 1.25.
- HR ratio, longest to shortest RR during deep respiratory cycles (6/minute): 1.07
- Orthostasis: as noted, his HR increased markedly when his BP dropped on sitting,

The first 2 maneuvers indicate an intact parasympathetic nervous system response, and the third an intact sympathetic response. Thus, a generalized autonomic neuropathy is not likely to be playing role in his chronic vomiting. N.B. this battery of exam findings is consistent with his history: i.e. no symptoms of urinary difficulties, chronic constipation, or orthostatic dizziness.

EKG: revealed prominent U-waves, suggesting hypokalemia;

Hb/Hct: Hematocrit was spun the 3rd day, well after BP/HR returned to baseline with many liters of fluid: 28.

Smear was examined: suggestion of a bi-morphic picture: both macrocytes and microcytes observed. Results suggest nutritional deficiencies of iron (with both loss from bleeding and malabsorption

possibly compounding intake), and B12 and/or folate, through inadequate intake and possibly malabsorption).

Electrolytes were not available.

UGI: contact was made with the closest hospital that had barium in stock, and plans were made to send some to Kisoro within 2-3 days.

4. What is the differential diagnosis of chronic vomiting in this patient, and what are the pros and/or cons of each possibility?

The differential diagnosis of chronic vomiting:

- gastric outlet obstruction, partial, due to peptic ulcer disease and duodenal stricture (see below)
- partial intestinal obstruction (stenotic Crohn's disease, intestinal TB/stricture, neoplasm of the SI, ischemic strictures, surgical adhesions, pelvic inflammatory disease in women): the long course without treatment, or the lack of inflammatory symptoms, compatible pain history, surgery or diarrhea make these possibilities unlikely.
- motility disorders:
 - gastroparesis: only about half of the cases of gastroparesis is associated with diabetes (which this patient doesn't have). Gastroparesis and mechanical gastric outlet obstruction can present similarly – without significant pain and with marked dilatation of the stomach and vomitus containing undigested food. (Neuropathic gastroparesis from DM is often associated with pain and vomiting even with an empty stomach.) Gastroparesis, although a chronic disorder, produces symptoms intermittently and patients are usually asymptomatic between episodes. The constancy of this patient's symptoms for the past 18 years as reflected in his chronic emaciation, argues against gastroparesis.
 - chronic intestinal pseudo-obstruction: a motility disorder of the SI may present with nausea and vomiting but other symptoms of SI dysmotility such as abdominal pain and overt distention are usually present, and small bowel mechanical obstruction often the apparent diagnosis. Again, the problem presents more episodically, and this patient's lack of distention and the chronic nature of his problem make pseudo-obstruction unlikely.
- functional vomiting: (defined by Rome II Committee as 1 or more episodes of vomiting on a minimum of 3 separated days in a week for 3 months of the preceding year.) Population-based studies indicate that vomiting occurs once a month or more in ~2-3% of the general population. Although if severe, functional vomiting can induce nutritional deficiencies, it's almost unheard of in this rural African population, and particularly in this elderly male patient.
- cyclic vomiting: clustered episodes of vomiting that last from 1 day-3 weeks (average 6 days) that tend to be stereotypic and separated by asymptomatic periods of 2 weeks-6month. About 25% are associated with migraine, 2/3 with abdominal pain, and occasionally mild fever and diarrhea too. Onset at any age, rare in the elderly. This entity is not consistent with this patient's symptom chronicity.

5. What is the most likely diagnosis and why?

What treatment is necessary?

The clinical diagnosis in this patient is gastric outlet obstruction, partial, due to peptic ulcer disease (PUD) and duodenal stricture.

Although almost never seen in the West nowadays because of the effective treatments available for PUD for the past 20-30 years, prior to the 1980's 12% of patients with PUD presented with gastric outlet obstruction due to either ulcer-induced inflammation and edema, or scarring of the duodenal bulb, the site of most DUs.

*For over 20 years, since broad acceptance of the pioneering work of Warren and Marshall which won the Nobel Prize, we've known that PUD is caused by the bacteria *H. Pylori* which resides in the gastric mucosa. *H. Pylori* infection affects over half the world's population, 5-10% of which develop ulcers. Necessary for ulcers to form is the combination of the bacterially-induced inflammation and the acidity of the stomach.*

If HP affects primarily the distal stomach (antrum) while leaving unaffected the body of the stomach where acid-producing parietal cells are found, hyperacidity results. This is because HP, in order to survive, produces urease in order to form NH_3 to buffer stomach acid, and the local alkaline environment in the antrum eliminates the negative feedback loop that normally restrains gastrin and acid-production proximally. The hyperacid stomach contents spill into the duodenum producing metaplasia in the bulb, allowing the HP to infect the downstream duodenal mucosa. The inflammation of the HP infection and hyperacidity together induce ulceration.

Duodenal ulcers are far more common than gastric ulcers, but both require HP and acid. With gastric ulcers, the HP infection involves the body of the stomach as well the more distal antrum, and thus acid production is diminished. Even with below-normal acid production, acid is necessary for ulcers to form in the stomach. HP eradication cures both forms of PUD, duodenal and gastric.

DUs bathed in acid produce pain, and the classic DU history is a burning pain that occurs on an empty stomach... thus 3-4 hours post-prandially and in the middle of the night. It's relieved by consuming food or drink. However, many with PUD have no pain, and pain patterns are atypical at least 30% of the time.

In the years prior to potent anti-acid regimens with H2-blockers followed by PPIs, and then finally the "H. pylori revolution" and antibiotic cure of the ulcer-forming diathesis, obstruction was a common presentation of complicated PUD in the West - along with perforation, bleeding and posterior penetration. DUs induce gastric outlet obstruction most commonly through scarring the duodenal bulb and narrowing the gastric outlet. In a surgical series of 58 patients from Nigeria (East Af. Med. J. 1999; 76:690), 50% of outlet obstructions were due to scar alone, 25% to scar and adhesions, 15% to adhesions alone, and 10% to edema. The adhesions were thought secondary to past micro-perforations.

As in this patient, the vomiting in gastric outlet obstruction is post-prandial and delayed - usually many hours after meals, and often the following morning upon arising. During this hiatus, the food emulsifies and the increased osmolarity of stomach contents draws in water and stretches the stomach inducing vomiting. The history of undigested food in the vomitus is classic for outlet obstruction, indicating that food hasn't gained access to the intestines.

Over time, whatever component of the obstruction was due to edema resolves, usually partially, and the patient can go on until the next exacerbation. The stomach slowly expands and never fully empties. Bezoars can form. Patients usually adapt by eating less and less. Nutritional deficiencies and gross malnutrition can become evident, as in this patient.

*This patient NEEDS SURGERY! In the past a vagotomy with drainage procedure was curative. Food needs to bypass the scarred duodenum and reach the intestines. Immediately, “putting the stomach to rest” via NG suction and treatment of *H. Pylori* with antibiotics and of acid with an IV PPI would be indicated. Given his age and inanition, surgery carries a significant mortality risk, although the alternative, medical therapy which has failed many times over the past 50 years in him, would be worse.*

Result: Barium arrived and an UGI series showed an enormous dilated stomach that filled the abdomen, with a trickle of barium seen in a thoroughly distorted duodenum. A film taken 48 hours later showed more than half the barium still in the stomach, and a narrow tortuous channel for a duodenum. (The stomach was so huge and much of it at the pelvic brim that a “succussion splash” would be hard to generate against its distended walls – clearly a “false negative” exam.)

6. What is the epidemiology of the underlying disease in Africa, and the implications of this patient’s presentation on the social dimensions of health care in Africa?

H. Pylori is thought to be the most common infection in Africa. Although half the world is infected by HP, in Africa as in most of the developing world, half are infected by age 10, and upwards of 80-90% throughout lifetime. Although most infections are chronic unless eradicated by antibiotics, there’s an unknown proportion that are spontaneously eliminated by the body’s natural immunity. Because the first hospital-based reports of PUD from Africa suggested a lower incidence/prevalence of ulcer disease, an “African enigma” was thought to exist: more HP, but less ulcer disease. We now think that that “enigma” was a product of biased data, and that there is no diminution of HP-induced clinical symptomatology, just less access to care and good clinical science.

This patient is a tragic example of health disparities between the developed and the less-developed worlds, a relatively recent phenomenon of the late 19th and 20th Centuries. Usually, we think of disparities involving infectious diseases and malnutrition: malaria, TB, HIV, cholera, strife-induced starvation, etc. but in this case, it’s a common chronic curable disease that has plagued him throughout his adult life and crippled him for the past 18 years. He sought care late, had no access to definitive diagnosis for decades, and was often misdiagnosed and mismanaged. In the meantime, he withered away and has been unable to work for nearly 2 decades. His clinical presentation was from another era of the disease: although gastric outlet obstruction and other complications of PUD are a thing of the past in the West, they’re still very relevant to present-day practice in rural Africa.

Finally, when surgery was offered the patient and his family balked at the (modest) cost of the procedure at the nearest hospital that could perform it. The team patiently explained the competing risks over days, and raised money for them. In the end, they accepted, and returned to their village with cans of oral high-energy calorie supplementation to prepare for the operation. Unfortunately, he never returned for follow-up or for the operation.

Suggested Readings:

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