



## **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](mailto: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](mailto: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 26 – ACUTE DIARRHEA x 2 Vignettes

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Please read the following brief vignettes and answer the questions that follow:

- A. A 4 year old boy presents with 3 days of diarrhea. At first the diarrhea was watery 5-10 times a day, but now it's worse: small volume stools about 20 times a day, mixed with blood and mucous. He's felt very "hot". Weak and listless, his mother brought him to the hospital after he had a seizure at home.

The physical exam is notable for general apathy, a temperature of 104; heart rate 130; RR 20, not deep; capillary refill 2 seconds; skin turgor intact; and a distended, tender abdomen with visible bowel loops.

**1. What is the "frame" in this case (i.e. the key clinical features the final diagnosis must be consistent with) and the clinical significance of each?**

- *Frequent (20x/day), small volume stools: suggests a colonic origin of the diarrhea*
- *blood and mucous: signs of an invasive organism causing "dysentery"*
- *acute (days) with high fever: suggests a bacterial etiology and a robust inflammatory response*
- *seizure: probably multifactorial, but worrisome*
- *distended tender abdomen with visible loops of bowel: very worrisome, a sign of toxic megacolon with a high risk of perforation*

**2. How volume depleted is the patient likely to be and why?**

*Moderately depleted (probably ~5%), not severely: the lack of vomiting and the small volume stools mean that volume loss is not the major problem in this patient or with dysentery in general. Small bowel diarrhea usually results in greater enteric losses.*

*In a JAMA systematic review, the most useful clinical signs that predicted 5 percent hypovolemia in children were delayed capillary refill time of 2-3 seconds, reduced skin turgor, and deep respirations with or without an increase in absolute respiratory rate, with a combination of signs and symptoms being better at indicating hypovolemia than an individual finding.*

*However, in this patient volume depletion is still likely to be clinically significant: apathy and anorexia decrease oral intake, and insensible losses are exaggerated with high fevers; loss of protein through the gut decreases oncotic pressure*

*exacerbating intravascular volume depletion. Finally, there's 3<sup>rd</sup> spacing into the distended atonic large bowel in this case, further depleting intravascular volume.*

**3. What is the likely diagnosis?**

**What is the epidemiology of the probable causal organism?**

*This is likely to be dysentery caused by Shigella dysenteriae type 1 - the most virulent of the Shigella subspecies and the one that dominates (and is only found in) the developing world. It results in a million deaths a year mostly of children under 5, and is responsible for 5-15% of diarrhea in the developing world. (N.B. Shigella species are responsible for only 1% of the diarrhea cultured in the U.S. However, most Shigella infections in the U.S. never come to medical attention because infection with S. sonnei, the subspecies prevalent in the U.S., usually cause only self-limited, mild-moderate, watery diarrhea.)*

*Mild disease is not usual with S. dysenteriae Type 1, most of which induce the triad of abdominal cramps/ tenesmus, fever and bloody diarrhea. Since only 10-100 organisms are required to induce infection, human-to-human transmission via poor hygiene and hands contaminated with microscopic feces is frequent, and even flies can act as the food-borne vector.*

**4. What severe complications of infection with this organism are evident in this case? Why did the child seize?**

*The 2 complications evident on presentation are toxic megacolon, which is seen in 10% of cases and puts the patient at high risk of perforation, and seizures. Toxic megacolon is associated with a mortality of up to 50%.*

*The seizures are "febrile seizures" in young children, but often co-precipitated by hypoglycemia due to starvation and inadequate gluconeogenesis, and hyponatremia, <125 mmol/L in 50% of children with severe S. dysenteriae in one study from Bangladesh. Hyponatremia is due to both loss of protein and oncotic pressure with resulting intravascular depletion, and ADH secretion "inappropriate" for the degree of volume depletion seen.*

**5. The child becomes increasingly weak despite hydration. On exam, marked pallor is noted. He stops urinating despite stable BP. What may be happening?**

*It's likely that the hemolytic-uremic syndrome (HUS) has set in as a complication of the infection. (HUS is due to "Shiga toxin" produced by S. dysenteriae type 1 binding to glomerular and endothelial cells and causing secretion of very large VWF multimers. The large VWF multimers promote platelet aggregation which leads to both renal injury and hemolysis).*

*HUS usually occurs in young children in whom it is probably the most common cause of acute renal failure worldwide. In the developed world HUS is usually caused by shiga toxin produced by E.coli 0157 and follows 10-15% of infections; in Africa, S. dysenteriae is probably the more common precipitating infection though HUS follows less frequently.*

*After the bout of dysentery by S. dysenteriae, HUS follows by 1-5 days. Unlike HUS from E.coli 0157 (in which antibiotics may add to the risk of HUS) it's preventable if Shigella is treated within 3 days of (dysenteric) symptom onset.*

## **6. What other complications are seen after this infection?**

*Shigella is a common precipitant of malnutrition due to severe anorexia, marked catabolism, and protein-losing enteropathy.*

*S. flexneri, the second most virulent shigella species, is associated with reactive arthritis, conjunctivitis and urethritis, with symptoms occurring 1-2 weeks post-dysentery.*

- B. A 17 year old boy presents with 3 days of fever and diarrhea. He was well until 5 days ago when he awoke in the morning and felt too weak to go work the fields. He had 2-3 loose bowel movements and, the day after, watery diarrhea began along with a scant non-productive cough. For the past 3 days he's had diarrhea, 4-8 times a day, watery and non-bloody, moderate volume, without tenesmus. He's vomited once but otherwise has kept food and liquid down. He's felt "hot" with chills each day; his cough is mild and persists but hasn't gotten worse, and his diarrhea has also been about the same since the first day of illness. However, he's felt progressively weaker and was brought to the hospital by family after being too weak to get out of bed.

Physical Exam on admission: T: 96; BP: 72/45; HR 126; RR 20

Mouth/pharynx: moist, no thrush; conjunctiva normal, not pale; no goiter;

Lungs clear;

Heart normal, S1, S2 no murmurs/rubs;

Abdomen: slight distention, normal-decreased bowel sounds, tympanic without shift ;  
no hepato-splenomegaly or masses

Neurologic: grossly normal, diffusely weak (5-/5); no fine tremor

### **1. What are some of the key questions raised by the clinical data in this case, and how should they be further addressed at the bedside?**

- a. Is the patient's low blood pressure normal for him or a sign of shock, either due to severe volume depletion or sepsis?*

*b. Is the diarrhea “primary” or “secondary” i.e. is the bowel the source of the primary problem or is the diarrhea a non-specific response to inflammatory mediators?*

*c. Are there any clues to sources of infection elsewhere?*

*a. Blood pressures in rural Africa are much lower than in the West and it's common to find normal systolic BPs in the 70-90 range in adults, particularly in adolescents. However given the clinical context of extreme weakness and the history of feeling “hot” with chills, the likelihood that this BP represents shock is high; furthermore, the heart rate is excessive particularly in the absence of fever and likely a compensatory response to shock.*

*The history is important, though sometimes can be misleading. If the frequency of the patient's diarrhea is accurate, the volume doesn't seem excessive by report (e.g. <10x/day, moderate amounts) and he's drinking without vomiting. Thus shock due to loss of volume would be unusual. (However, at least in infants/children with diarrhea, the amount of diarrhea reported by the mother isn't predictive of the degree of volume depletion).*

*Probably the best way to tell the etiology of the hypotension at the bedside is via a “fluid challenge” in which 500 cc. of normal saline is infused rapidly over 15 minutes and the response of BP and HR monitored.*

*- If the low BP is due to volume depletion, the BP will rise >10 and the HR fall (by usually more).*

*- If the low BP is due to septic shock, the BP and HR will change minimally;*

*- No change in BP will be seen if the low BP is “normal” for him but if the HR slows down, mild volume depletion could be present;*

*N.B. the tachycardia may have another etiology independent of the BP: if not due to shock or fever, other causes such as severe anemia, anxiety, hyperthyroidism, pericarditis, etc. will have to be explored carefully.*

*b. “Primary” diarrhea is suggested by tenesmus, signs of mucosal invasion such as blood, pus or mucous, and a difficult-to-quantify appraisal of “quantity”: diarrhea that's either extremely frequent (e.g. >15x/day) or extremely voluminous suggests that the bowel is the primary target of the infection.*

*HOWEVER, diarrhea and vomiting can also be “secondary” – non-specific physiologic responses to the cytokines induced by bacterial infection anywhere. Such “secondary” diarrhea has none of the signs of mucosal invasion and is usually <10x/day. Not appreciating this can be fatal, as serious infections can be attributed to “gastroenteritis” and left untreated.*

*c. The clues to underlying infection (other than bowel) can be subtle. Whereas cellulitis may be obvious, pneumonia or endocarditis can be easily missed.*

**2. The patient was diagnosed as having either viral gastroenteritis or non-dysenteric bacterial enteritis, and hydrated at 150 cc/hour. His SBP rose to about 77, his HR remained 115-120 and though still weak, he said he felt better with the intravenous infusion. About 3 hours later, he seemed confused to his mother, and the resident measured his SBP at 60, HR 130. Six hours post-admission, the exam by the resident was otherwise unchanged and the attending was called.**

**a) What do you think the attending found?**

**b) What was the diagnosis in this case?**

**c) Name at least 4 “understandable” mistakes made in this case that could have led to the patient’s demise. Why are they “understandable”?**

*a. Careful lung exam by the attending found tubular breath sounds without crackles in the left axilla, quite different from the alveolar breath sounds in the right axilla. The finding was diagnostic of consolidation from community-acquired pneumonia, probably pneumococcal. (When the resident was re-focused on the area, he still found the exam “unremarkable” until he was “tuned into” the differential pitch of the sound and the duration of expiration between the two axillae.)*

*b. The diarrhea this patient experienced was “secondary” to cytokines/sepsis, and the hypotension was due to septic shock.*

*c. The potential for mistakes in this case is enormous:*

- Too readily accepting the “obvious” as causal (and not simply “associated”) without probing further: i.e. low BP with diarrhea = low BP because of diarrhea; extreme weakness in a patient with diarrhea is because of volume depletion ....*
- Not appreciating the presentation of “secondary” diarrhea as part of the sepsis syndrome, and thus not carefully following clues to non-bowel sources of infection. In this case the patient’s scant dry cough was a clue from history to the existence of pneumonia.*
- Not appreciating that fever in most infection is intermittent, and the lack of fever on admission didn’t mean “viral gastroenteritis”.*
- Not giving a “fluid challenge” (but “hydrating” only) and not addressing the origin of the persistent tachycardia.*
- Not knowing what bronchial or tubular breath sounds sound like... and thus listening only for crackles/rales as signs of pneumonia.*

*[The physical signs of consolidation are infrequently appreciated in the U.S. nowadays because they take days to develop and in the U.S. patients come to medical attention before they appear (American patients with pneumococcal PNA show up an average of 2 days after symptom onset and get treated; exam signs of consolidation took 3 or more days to develop post-hospitalization in the pre-antibiotic*

era.) We may listen for “crackles” or “rales”, non-specific and earlier signs of an infiltrate, wet lungs, atelectasis, fibrosis, etc., (and signs with only a fair-moderate observer agreement), but we almost always miss the infrequent signs of consolidation - bronchial or tubular breath sounds.

*Bronchial and tubular sounds take their name from auscultation of normal lung over the major bronchi and trachea (a big “tube”), respectively. Solid or (unobstructed) consolidated tissue transmits sound more efficiently than air, generating sounds that are variably louder, clearer, of higher pitch, and with a shorter I/E ratio (instead of the 2/1 inspiratory to expiratory ratio heard over the peripheral lung/alveoli, it’s 1/1 over the bronchi and 1/2 over the trachea ... i.e. “longer is where air passes last”). They’re easy to miss because they reflect a change in the quality or pitch of the sound, not something that’s altogether different like crackles. Although often accompanied by crackles, the crackles may be subtle and overlooked on cursory exam.]*

- *Not examining the axillae for signs of pneumonia*

*[The lobar bacterial pneumonias, classically pneumococcal, often present with physical and radiologic abnormalities in very circumscribed segments of the lung: indeed, bacteremia and shock can often be traced to infection of one isolated segment of a lobe. Thus, all segments have to be examined carefully, comparing sides - and this includes segments in the axilla bilaterally.]*

*If the signs of extreme weakness despite only moderate diarrhea, inappropriate tachycardia, cough, and axillary consolidation are not “picked up”, it’s easy to interpret this presentation of an afebrile patient with moderate watery diarrhea to be either a viral gastroenteritis or a non-dysenteric bacterial enteritis, both of which should NOT to be treated with antibiotics (in the case of bacterial enteritis, lest we prolong the carrier state). This could be fatal.*

### **Suggested Reading:**

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