

Chronic diarrhea clinical case

Facilitator's copy

Information for learners:

68 y/o patient with history of hypertension, dyslipidemia, asthma, depression, and gastric ulcers comes to the clinic because of diarrhea. She has been having diarrhea for about 6 months. Before the diarrhea started, she had an episode of GI bleed that was secondary to gastric ulcers caused by prolonged NSAID use. She was treated with omeprazole. She recently had a follow up EGD that showed resolution of the ulcers.

Vitals: RR 16, HR 72, BP 123/72, T 36.2, Weight 63.1 kg.

Case discussion

What other questions regarding her symptoms would you like to ask?

What other details about her history do you want to know?

What would you like to know about her physical exam?

What are some of your differential diagnoses?

- Mnemonic "WATERS" can help with some of the causes for acute diarrhea (ingestion of contaminated Water; Antibiotics/medications; Travel; Everyone else in family sick?; Restaurants/food contamination; Systemic disease)

At this point, what other work up would you like to do?

More patient information to provide if participants ask:

Diarrhea started around the same time when she was diagnosed with the gastric ulcers. She describes diarrhea as having about 4 BMs per day, with consistency from soft to watery. Episodes happen shortly after she eats. She also has some cramping pain that improves after having a BM. She has had nausea but no vomiting. No blood in her stool or dark stools. She does not have diarrhea or pain at night or when she hasn't eaten. She has had no changes in her diet, except that she has not had alcohol or caffeine for about 6 months. She avoids lactose b/c she is lactose intolerant. She has not travelled. No other family members have similar symptoms. No fevers or chills. No weight loss.

Rest of ROS negative.

Surgical history: no history of surgeries

Allergies: none

Family history: no family history of CRC, no history of IBD

Medications: Valsartan-hydrochlorothiazide, Simvastatin, Albuterol, Omeprazole (recently discontinued)

Physical exam:

Gastrointestinal: abdomen is non distended, slightly increased bowel sounds, no tenderness to palpation, no masses palpated, no organomegaly. Normal external anal sphincter tone with mild erythema around, no fissures, no blood in rectal exam, no hemorrhoids.

Rest of PE unremarkable.

Initial labs performed: CBC, LFTs, chem7, TSH, ESR, CRP, fecal lactoferrin, celiac serology negative, Giardia, cryptosporidium, stool ova and parasites all negative.

Endoscopy done 2 months prior is normal, healed ulcers. Colonoscopy from earlier this year was not completed b/c poor preparation.

Chronic diarrhea workup and management

How is chronic diarrhea defined? Decrease in stool consistency and increased stool frequency of greater than four weeks duration.

How can chronic diarrhea be categorized according to stool characteristics? Watery, fatty, inflammatory. Watery diarrhea can be subdivided into osmotic or secretory.

What are some of the causes of chronic diarrhea? (see table below)

What are the first steps in the evaluation of a patient with chronic diarrhea? What specific things do you want to ask about the patient's history? What do you want to look for in the physical exam? What lab tests would you initially get?

Detailed history (clarifying what patient defines as diarrhea, duration of symptoms, frequency and characteristics of stool, associated symptoms, presence of incontinence, occurrence during fasting or at night, volume of diarrhea, presence of blood, weight loss, association of stress and depression, travel history, hospitalization, all medications, dietary history, medical history, history of abdominal surgery, family history of CCR, CD, IBD), physical exam and +/- lab testing to help characterize the diarrhea.

Initial lab testing:

CBC and differential, TSH and T4, celiac serologies, serum electrolytes (severe diarrhea, dehydration), stool occult blood, test for giardia, fecal calprotectin or lactoferrin, CRP or ESR.

Other testing to consider: endoscopic evaluation, abdominal imaging, blood testing for malabsorption, other stool testing for infectious disease, fecal osmotic gap, fecal chymotrypsin and elastase, hydrogen breath tests. More rare causes: testing for hormone secreting tumors, screen for laxative abuse.

How can you distinguish between functional from organic causes of diarrhea? Rome criteria for IBS. Functional diarrhea if pain is not present.

What are some alarm features that would prompt you to refer patients for endoscopic evaluation?

Age of onset after 50, rectal bleeding or melena, nocturnal pain or diarrhea, progressive abdominal pain, unexplained weight loss, fever or systemic symptoms, laboratory abnormalities, first degree relative with IBD or colorectal cancer.

What empiric treatments can be used for symptomatic management?

Loperamide (scheduled dosing recommended).

Bile acid binding resins for suspected bile acid malabsorption.

Fiber supplementation for small volume watery diarrhea and fecal incontinence.

Oral calcium supplementation may treat mild chronic diarrhea.

Bismuth subsalicylate – concern for adverse effects with chronic use.

Table 1. Differential Diagnosis of Chronic Diarrhea

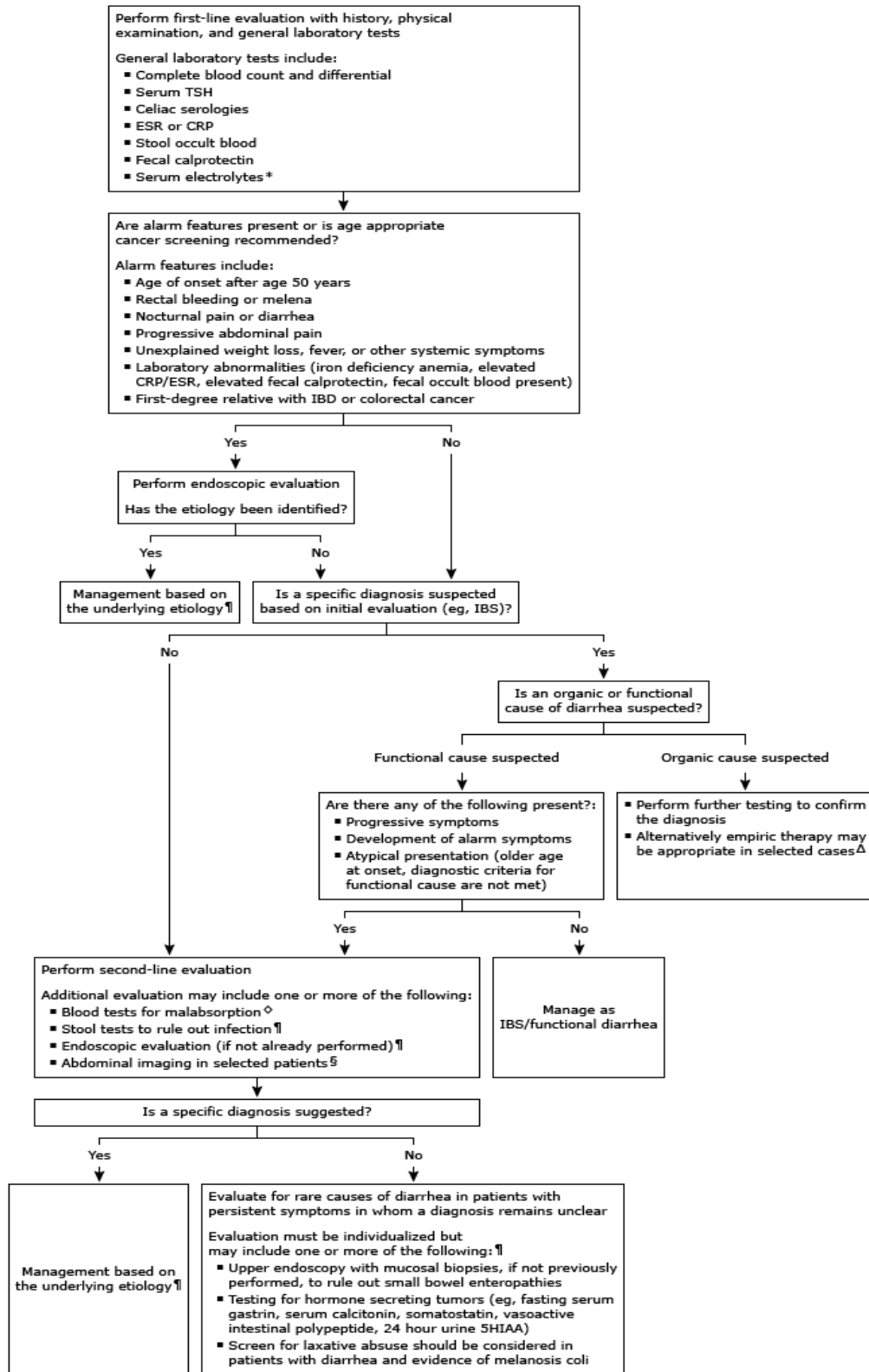
<p>Watery</p> <p>Secretory (often nocturnal; unrelated to food intake; fecal osmotic gap < 50 mOsm per kg*)</p> <ul style="list-style-type: none"> Alcoholism Bacterial enterotoxins (e.g., cholera) Bile acid malabsorption Brainerd diarrhea (epidemic secretory diarrhea) Congenital syndromes Crohn disease (early ileocolitis) Endocrine disorders (e.g., hyperthyroidism [increases motility]) Medications (see Table 3) Microscopic colitis (lymphocytic and collagenous subtypes) Neuroendocrine tumors (e.g., gastrinoma, vipoma, carcinoid tumors, mastocytosis) Nonosmotic laxatives (e.g., senna, docusate sodium [Colace]) Postsurgical (e.g., cholecystectomy, gastrectomy, vagotomy, intestinal resection) Vasculitis <p>Osmotic (fecal osmotic gap > 125 mOsm per kg*)</p> <ul style="list-style-type: none"> Carbohydrate malabsorption syndromes (e.g., lactose, fructose) Celiac disease Osmotic laxatives and antacids (e.g., magnesium, phosphate, sulfate) Sugar alcohols (e.g., mannitol, sorbitol, xylitol) <p>Functional (distinguished from secretory types by hypermotility, smaller volumes, and improvement at night and with fasting)</p> <ul style="list-style-type: none"> Irritable bowel syndrome 	<p>Fatty (bloating and steatorrhea in many, but not all cases)</p> <p>Malabsorption syndrome (damage to or loss of absorptive ability)</p> <ul style="list-style-type: none"> Amyloidosis Carbohydrate malabsorption (e.g., lactose intolerance) Celiac sprue (gluten enteropathy)—various clinical presentations Gastric bypass Lymphatic damage (e.g., congestive heart failure, some lymphomas) Medications (e.g., orlistat [Xenical; inhibits fat absorption], acarbose [Precose; inhibits carbohydrate absorption]) Mesenteric ischemia Noninvasive small bowel parasite (e.g., <i>Giardia</i>) Postresection diarrhea Short bowel syndrome Small bowel bacterial overgrowth (> 10⁵ bacteria per mL) Tropical sprue Whipple disease (<i>Tropheryma whippelii</i> infection) <p>Maldigestion (loss of digestive function)</p> <ul style="list-style-type: none"> Hepatobiliary disorders Inadequate luminal bile acid Loss of regulated gastric emptying Pancreatic exocrine insufficiency 	<p>Inflammatory or exudative (elevated white blood cell count, occult or frank blood or pus)</p> <ul style="list-style-type: none"> Inflammatory bowel disease <ul style="list-style-type: none"> Crohn disease (ileal or early Crohn disease may be secretory) Diverticulitis Ulcerative colitis Ulcerative jejunoileitis Invasive infectious diseases <ul style="list-style-type: none"> <i>Clostridium difficile</i> (pseudomembranous) colitis—antibiotic history Invasive bacterial infections (e.g., tuberculosis, yersiniosis) Invasive parasitic infections (e.g., <i>Entamoeba</i>)—travel history Ulcerating viral infections (e.g., cytomegalovirus, herpes simplex virus) Neoplasia <ul style="list-style-type: none"> Colon carcinoma Lymphoma Villous adenocarcinoma Radiation colitis
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*—Fecal osmotic gap = 290 – 2 × (stool sodium + stool potassium). It helps differentiate secretory from osmotic diarrhea. Normal fecal osmolality is 290 mOsm per kg (290 mmol per kg). Although measurement of fecal electrolytes is no longer routine, knowing the fecal osmotic gap helps confirm whether watery stools represent chronic osmotic diarrhea (fecal osmotic gap greater than 125 mOsm per kg [125 mmol per kg]) or chronic secretory diarrhea (fecal osmotic gap less than 50 mOsm per kg [50 mmol per kg]).¹

Information from references 1 and 2.

Proposed algorithm for workup:

Suggested approach to the evaluation of chronic diarrhea in adult patients in resource-rich settings



TSH: thyroid-stimulating hormone; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; IBD: inflammatory bowel disease; IBS: irritable bowel syndrome; HIAA: hydroxy indole acetic acid.

* Serum electrolytes should be evaluated in patients with severe diarrhea, or when there is concern for dehydration or electrolyte abnormalities.

¶ Refer to appropriate UpToDate topic on chronic diarrhea.

Δ Empiric therapy may be appropriate when comorbidities limit diagnostic evaluation or when a diagnosis is strongly suspected (eg, lactose restriction for suspected lactose intolerance, bile acid binding resin for post-cholecystectomy diarrhea).