APPROACH TO TWO COMMON CAUSES OF ACUTE ABDOMINAL PAIN IN CHILDHOOD

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I. Appendicitis

A. History (illness progresses with time)
1. Periumbilical pain for 12 hrs or pain in the right lower quadrant
2. Nausea, vomiting, anorexiab
3. Low grade fever
4. Pain with motion, going over bumps in car

B. Physical Examination (varies based on time)
1. Tachycardia
2. Pharynx - R/O pharyngitis with mesenteric adenitis
3. Chest - clear, R/O pneumonia
4. Abdomen - Right lower quadrant peritoneal signs
5. Rectal - tender on right, R/O constipation (usually not needed for diaprosis)
6. Pelvic - R/O Mittelschmerz, R/O pelvic inflammatory disease
7. Can the child jump without pain
8. Examination is best done while the child is distracted
9. Rebound is not a useful sign as most children will react even if it doesn't hurt

C. Laboratory
1. CBC with differential
2. Urinalysis

D. X-rays - if necessary to R/O pneumonia; look for appendicolithiasis
Plain abdominal films are generally NOT useful for abdominal pain, U/S or CT are NOT necessary for the diagnosis of appendicitis.
These tests should be used on a case-by-case basis.

E. Surgical consultation should be utilized in any child with possible appendicitis.

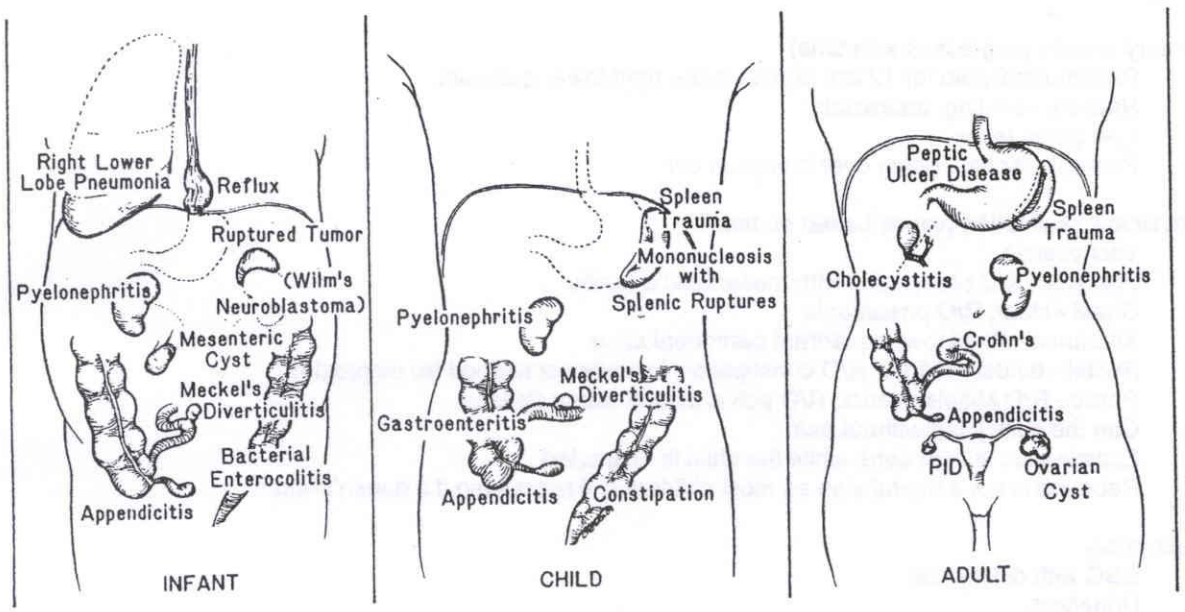
II. Intussusception

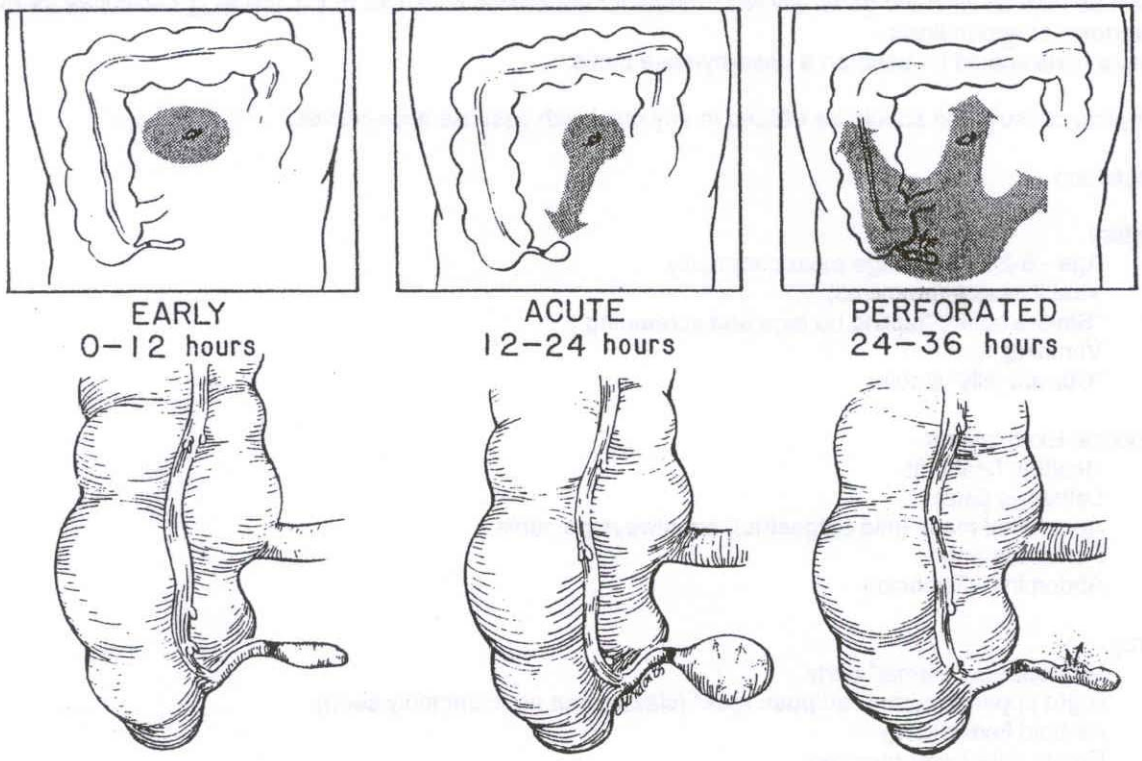
A. History
1. Age - 6-24 mos of age most commonly
2. Viral illness (adenovirus)
3. "Severe colic" - pulling up legs and screaming
4. Vomiting
5. "Currant jelly" stools

B. Physical Examination
1. Healthy, fat infant
2. Lethargic (late)
3. Abdominal mass (mid epigastric), not always palpable
4. Peritonitis (late)
5. Abdominal distension

C. X-ray
1. May appear "normal" early
2. Right upper quadrant "adipose rose" (classic, but not commonly seen)
3. Air fluid levels (late)
4. Empty right lower quadrant

D. Plan
1. Surgical consultation
2. IV fluids
3. Antibiotics only in child under 6 months of age. If older than 6 months give antibiotics only after diagnosis is confirmed by contrast study.
4. Contrast enema for dx and possible reduction of intussusception (70% success)
5. Surgical reduction or resection, if necessary





APPENDICITIS

TABLE I. DIFFERENTIAL DIAGNOSIS OF APPENDICITIS

	Gastroenteritis	Appendicitis	Urinary Tract Infection
Pain	Diffuse, cramps	Periumbilical right lower quadrant	Flank, dysuria
Vomiting	Coincides with pain	Usually follows pain	Minimal
Diarrhea	Large volumes	Small amounts	Minimal
Fever	Varies	Low grade early	May be high
Course	Intermittently feels better	Worsens with time	Changes slowly
Physical Exam	Soft abdomen, hyperactive, bowel sounds	Peritoneal signs, right lower quadrant, rectal localized to right	Costovertebral angle tenderness

TABLE 2. COMMON CAUSES OF ABDOMINAL PAIN BY AGE GROUP

Infants	3-11 Year Old	Adolescent
Intussusception	Appendicitis	Appendicitis
Hirschsprung's enterocolitis	Trauma	Pelvic inflammatory disease
Strangulated hernia	Meckel's diverticulitis	Mittelschmerz
Trauma (child abuse)	Pneumonia	Crohn's disease
Meckel's diverticulitis	Bacterial enterocolitis	Enterocolitis
Bacterial enterocolitis	Yersinia	Peptic Ulcer Disease
Pneumonitis	Campylobacter	Cholecystitis
Pyelonephritis	Salmonella	Pneumonia
Mesenteric cysts	Shigella	Trauma
Testicular torsion	Crohn's disease	Ectopic pregnancy
Pancreatitis or pseudocyst	Pancreatitis	Hematocolpos
Intestinal obstruction/ volvulus	Infected mesenteric cyst	Psychosomatic
	Ruptured tumors	
	Pyelonephritis	

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HEMOLYTIC UREMIC SYNDROME

A Handout for Residents by Trudie Sprenkle, MD
Revised by Phillip Tarr, MD, Allison Eddy, MD, and Nancy Bischoff, MD

DEFINITION: Acute nephropathy characterized by a triad of findings: microangiopathic hemolytic anemia, thrombocytopenia, and acute renal failure. It affects multiple organ systems.

CLASSIFICATION:

DIARRHEA-ASSOCIATED HUS (D+HUS)

1. Typically related to *E. coli* 0157:H7, which produce Shiga toxin(s). May also be caused by *Shigella dysenteriae* type 1 (though not in North America)
2. *E. coli* 0157:H7 may be found in undercooked meat, unpasteurized foods/fluids, water and other sources.
3. D+HUS occurs primarily in infants/young children (ages 7m-4y). There are sporadic as well as epidemic cases. Most cases occur in summer and early fall.
4. D+HUS is the most common cause of acquired renal failure that requires dialysis in previously healthy children.
5. Overall prognosis is good with supportive therapy, and relapse is unlikely. (See Prognosis below)

NONDIARRHEA-ASSOCIATED HUS (D-HUS)

1. May be related to *Streptococcus pneumoniae* (neuraminidase-associated), or to other bacterial or viral agents.
2. May be inherited (autosomal dominant or recessive) and related to vWF-metalloproteinase deficiency, drug-associated (Cyclosporin A, oral contraceptives, chemotherapy, etc.), or factor H deficiency.
3. Prognosis is worse than HUS with diarrhea, with increased morbidity and higher incidence of relapse.

CLINICAL FEATURES

History: Prodromal GI illness 3-12 days prior to HUS (mean 6.5 days), followed by (variable) vomiting, crampy abdominal pain and (usually) bloody diarrhea; finally renal insufficiency.
Exam: VS: +/- fever prior to onset of HUS, Htn. CNS: Often irritable, drowsy, less often with seizures or focal neurologic signs. Uncommonly present in CHF. GI: pain (variable degree), bleeding, possible jaundice. SKIN: pallor, petechiae.

LABORATORY FINDINGS

1. HEMATOLOGY:
   Decreased platelet count, increased platelet size; Coombs-negative hemolytic anemia, increased reticulocyte count, fragmented RBCs; PT/PTT usually normal or near-normal; fibrin degradation products increased, fibrinogen normal to increased; WBC's elevated with left shift.
2. SERUM CHEMISTRIES:
   Elevated BUN, Cr, uric acid; K+-variable; Increased LFTs, bili, hypoalbuminemia; increased pancreatic enzymes, hyperglycemia.
3. URINE:
   Urinalysis: proteinuria, heme positive, leukocyte esterase positive, bili +/-
   Micro: red blood cells, white blood cells, and casts (cellular, granular, pigmented, hyaline)

MANAGEMENT: Focus of therapy is supportive once the diagnosis of HUS is established.

1. Correction of fluid/electrolyte abnormalities:
   a. Serial weights, monitor Ins/Outs, HR & BP, orthostatics.
   b. VOLUME DEPLETION repleted with normal saline if indicated, then restrict fluids to insensible losses plus ongoing losses (including urine if not anuric).
   c. Monitor CBC, lytes, urea, calcium and phos daily.
   d. Dialysis if severe fluid overload, hyperkalemia, acidosis, or hyponatremia unresponsive to initial therapy.
   e. Nutritional support: Enteral or parenteral; monitor glucose and triglycerides particularly.

2. GI: Antimotility drugs are contraindicated (they increase both the severity of colitis and the risk of HUS).
3. ID: Antibiotics are contraindicated for the treatment of colitis. However, there are circumstances that arise during HUS that require antibiotics. The negative consequences of antibiotic usage during HUS are probably less than during the pre-HUS stage. We find it helpful to obtain repeat stool cultures at the beginning of the course, to determine if the patient has cleared the infection. The knowledge that an *E. coli* O157:H7 infection has cleared can be quite helpful in management issues, and in relaxing contact precautions.

There are three other ID items that should be kept in mind: (1) children with confirmed or suspected (including children with HUS who do not have a negative culture) *E. coli* O157:H7 infections should be kept under infection control "Contact Precautions." 2) children with HUS from whom an E. coli O157:H7 was not isolated should be considered to potentially be iunfected with a non-O157:H7 Shiga toxin-producing E. coli. These can be deteced by the laboratory, but require a special call to Microbiology (ext. 2585). (3) children at the HUS stage of illness often do not produce much stool. Therefore, requesting a rectal swab, rather than a stool culture, might accelerate the microbiologc workup. (4) C. difficile appears to be present in an unexpectedly high number of children with *E. coli* O157:H7 infection, or with HUS, so keep this possibility in mind.

4. Control of hyertension: attention to volume, antihypertensives.
5. Management of hematologic abnormalities
   a. pRBC tranfusions to keep Hct >20 (negotiable); give folic acid.
   b. platelet transfusions prn clinically-significant bleeding or pre-procedure. Platelet transfusions really need to be justified.

RENAL Prognosis: 65% have no longterm sequelae. 25% have mild sequelae (prolonged proteinuria or mildly decreased creatinine clearance). 0-10% have severe sequelae (refractory HTN, CRF)

EXTRARENAL COMPLICATIONS

1. GI:
   a. Colitis (occurs in ~ 90%): Microthrombi may lead to ischemia, necrosis, and perforation. Antimotility drugs are contraindicated; they increase severity of colitis. Intussusception, rectal prolapse, intestinal perforation, and strictures have been reported. About 10% have nonbloody diarrhea.
   b. Pancreatitis (occurs in up to 20%): defined by enzymes >4x normal plus clinical evidence. Endocrine function affected more than exocrine. Can lead to transient/permanent IDDM in 4-15%. Watch sugars.
   c. Liver effects (occur in ~ 40%): hepatomegaly, increased LFTs, cholestatic jaundice due to hemolysis-rarely clinically significant.
   d. Gall bladder: Cholelithiasis occurred in ~ 10% of patients in the 1993 outbreak.
2. CNS:
   a. Drowsiness and irritability are common but coma/semicoma/stupor, occur in ~ 15%. Seizures in up to 20-40%, usually generalized. Strokes in 4%. Chronic neurologic sequelae in 5%. However, we have had none of these complications since 1993 at this institution.
3. CARDIAC: Congestive heart failure, ischemia or infarction in <1%. Uremic pericarditis, effusions and rarely myocardial involvement may occur.
4. PULMONARY: Hemorrhage/edema in <1%.

PROGNOSIS

1. Usually excellent prognosis in D+HUS. Rate of recovery of renal function is 65-85%.
2. Some studies suggest that 40% with normal CrCl may have decreased GFR and renal functional reserve, but the clinical significance of this is unclear.
3. ESRD occurs in only an estimated 4-10%. May occur during the initial illness or due to progressive renal insufficiency and ESRD requiring chronic dialysis and/or renal transplantation months to years later.
4. Poor prognostic indicators: Nondiarrhea-associated HUS (especially if recurrent/hereditary), Age <1 or >5 years; Prolonged period of anuria; Severe hypertension; CNS findings (coma, seizure, hemiparesis/stroke), Elevated wbc count (>20,000/mm^3). Death occurs in 5-10%, usually from CNS involvement. Death from ARF is now rare.

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E. COLI O157:H7
Frequently Asked Questions by Physicians (Revised: October, 2001)

E. coli O157:H7 is a gastrointestinal pathogen that can cause diarrhea, bloody diarrhea, and the hemolytic uremic syndrome. Listed below are questions that are commonly asked by physicians regarding this pathogen:

1. **What are the sources for *E. coli* O157:H7?**
   Poorly cooked beef, particularly ground beef, has been the most frequently implicated vehicle. However, as surveillance for this organism increases, a much wider variety of vehicles is now being identified including such items as water, vegetables, unpasteurized milk and cider, mayonnaise, or salami, as well as infected persons. Generally, it is impossible to identify a source in a sporadic case. Source tracing is best performed by public health authorities who should be notified as soon as a case is diagnosed.

2. **What is the incubation period?**
   The average incubation period of *E. coli* O157:H7, in outbreak analysis, is approximately three days, with a range between 1 and 10-12 days.

3. **What are the symptoms**?
   Most frequently, *E. coli* O157:H7 infection is manifest by nonbloody diarrhea which then progresses to bloody diarrhea. Abdominal pain and cramping are usually prominent parts of the disorder. Associated symptoms, which occur variably, are transient fever, abdominal cramping and/or pain in advance of diarrhea, vomiting, and lethargy.

4. **Who should receive a stool culture?**
   The vast majority of diarrheal illnesses does not require microbiologic diagnosis. However, we encourage stool cultures to be obtained on all patients with acute bloody diarrhea, painful, nonbloody diarrhea, diarrhea with fever, acute diarrhea in an immune compromised patient, or diarrhea in a patient who has a family member with a stool culture positive for *E. coli* O157:H7. If you are not certain of your laboratory's practice, it is important to request that *E. coli* O157:H7 be sought.

5. **Are fecal leukocytes helpful or any other rapid tests on the stool helpful in making a diagnosis?**
   Approximately half of the patients with *E. coli* O157:H7 infection do not have fecal leukocytes. Therefore, we do not recommend the use of this test as a screen to predict the presence of this pathogen (or any other pathogen, for that matter). A recently developed rapid test for *E. coli* O157:H7 is specific but not completely sensitive. This test should not be used to replace a stool culture, when either positive or negative.

6. **What therapy should be given to a patient with *E. coli* O157:H7?**
   Hemolytic uremic syndrome, one of the most common causes of acute kidney failure in childhood, is the most important medical consequence of *E. coli* O157:H7 infection. Analysis of data from the 1993 Washington State outbreak failed to demonstrate any benefit of antibiotics in *E. coli* O157:H7 infection. In fact, patients administered antibiotics had a slightly, but not statistically significantly, increased risk of progression to hemolytic uremic syndrome. Moreover, recent data from sporadic cases suggest a strong association between the administration of antibiotics (β-lactams as well as trimethoprim sulfamethoxazole) to children infected with *E. coli* O157:H7 and the development of

HUS. Therefore, we do not recommend antibiotics for *E. coli* O157:H7 diarrhea. We also strongly urge against over-the-counter or prescription medications that might slow the gut, including antimotility, antidiarrheal or anticholingeric agents, or opioid narcotics.

We strongly encourage the admission to hospital of patients with possible or definite *E. coli* O157:H7 infection, and usually administer a 20 ml/kg bolus of normal saline (depending upon the intravascular volume status of the patient). We then maintain IV fluid therapy to replete volume, repeating boluses as necessary. Maintenance fluids and replacement for ongoing losses should be provided as with any other child. **We strongly encourage hospitalization of children with proven or probable *E. coli* O157:H7 infection, and continuation of IV fluid therapy and monitoring, until it is clear that HUS is not developing. Blood pressure should be monitored closely, and children observed carefully for signs of intravascular volume overload.** However, peripheral and eyelid edema are quite common due to hypoalbuminemia.

7. Can I add potassium to the IV fluid?
If the patient's electrolytes have a normal or low potassium, then it is appropriate to add potassium to the IV. The electrolytes should be checked at least daily until resolution.

8. How often are laboratory tests needed?
Unless a rapid deterioration is noted in the laboratory tests or clinical condition, daily determinations are usually adequate.

9. When does HUS occur and when do I know that my patient has recovered without this complication?
Approximately 7 days (± 2 days s.d.) after onset of diarrhea. Patients whose diarrhea has resolved for 2 days without laboratory evidence of HUS are very unlikely to develop this complication. An elevated WBC count (>13,000/ mm^3) suggests elevated risk for HUS, but this is not a completely sensitive, or very specific, indicator of such risk. A decreased platelet count is usually the first abnormality to be noticed in the progression to HUS, and the first abnormality to correct. The platelet count usually falls in children even in the absence of HUS, and its return to or towards normal can be used to give assurance that HUS will not develop. We usually obtain an additional CBC on the day after discharge.

10. What percent of children with *E. coli* O157:H7 infection develop HUS?
Approximately 10% of children under age 10 develop full blown HUS (Hct < 30%, creatinine above upper limit of normal for age, platelet count< 150,000/mm3). A similar percentage of infected children develop partial HUS (two of these three criteria are met).

The average age of children with HUS in Seattle is 4 years (+/- 4 years s.d.). Unfortunately, there is no test that can completely exclude the possibility that some infected children will develop HUS.

11. How do I report a patient with *E. coli* O157:H7 or with HUS?
Communicable disease reporting lines are listed below for the respective counties of residence of the patients.

Adams County	(509) 659-3315
Asotin County	(509) 758-3344
Grant County	(509) 754-6060 (option #0)
King County	(206) 296-4782
Pierce County	(253) 591-6410 (option #4)
Skagit County	(360) 336-9385
Snohomish County	(425) 339-5225
Thurston County	(360) 786-5470
Whatcom County	(360) 738-2503
Yakima County	(509) 575-4040(option #8)

When you report a case, please have available:

- Patient's name
- Patient's parent(s) name(s)
- Patient's home telephone number
- Patient's address
- Patient's date of birth
- Patient's sex
- Your name
- How you can be reached
- Laboratory which reported the culture being positive

12. How contagious is *E. coli* O157:H7?

Secondary cases do occur; in the 1993 outbreak, approximately 10% of the Washington State cases were classified as secondary by the Health Department. Child to child, child to adult, adult to child, and nosocomial transmission, have occurred and have each been documented. Any patient with diarrhea should exercise good hygiene (hand-washing, separate utensils). People with diarrhea should not return to settings where transmission is likely to occur, such as child care, day care, health care, or food service until the diarrhea has resolved. In the case of *E. coli* O157:H7, many health departments recommend two negative cultures prior to returning to child care, health care, or food service. This decision is best left to the local public health authority. The 2000 AAP Red Book recommends contact precautions for hospitalized patients who are infected with *E. coli* O157:H7.

13. What does one do with a patient who looks quite good, when the report of the stool culture becomes known?

Unfortunately, there is an imperfect relationship between the severity of the enteric prodrome and the development of HUS. We therefore urge the same diagnostic and therapeutic precautions regardless of gastrointestinal symptom severity.

14. Can antibiotics prevent an incubating infection?

There are no data to answer this question. However, there have been several cases of children and adults who were taking antibiotics when they contracted *E. coli* O157:H7 infection, and these medications did not prevent a severe course. Furthermore, antibiotics do not seem to affect post diarrheal carriage of *E. coli* O157:H7. We do not recommend the use of these agents to prevent infection in exposed individuals. It is probable that most people who ingest *E. coli* O157:H7 do not get sick.

This information is only intended for the use of physicians caring for patients with possible or confirmed *E. coli* O157:H7 infection. These statements are based on published and unpublished data, and represent the opinions of the author. Physicians retain individual professional judgment regarding the applicability of this information to and for their care of specific patients. Physicians are encouraged to telephone us (206) 526-2521) if there is a need for further elaboration, or if other questions arise.

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CHILDREN'S HOSPITAL & MEDICAL CENTER RESOURCES FOR PHYSICIANS

Division of Gastroenterology & Nutrition Dennis L. Christie, M.D. Karen F. Murray, M.D. David L. Suskind, M.D.	(206) 987-2521
Nephrology Fellow on call	(206) 987-2131
Infectious Disease Fellow on call	(206) 987-2131
Emergency Room Physician	(206) 987-2222