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PEDIATRIC Emergency Medicine Reports

The Practical Journal of Pediatric Emergency Medicine

Volume 12, Number 10 October 2007

Patients frequently present to the emergency department with gastrointestinal complaints such as vomiting, abdominal pain, and diarrhea. Identification of a foodborne illness may be very challenging. Foodborne illness (FBI) may cause atypical neurologic symptoms; rapid identification and treatment of those symptoms may decrease patient morbidity and mortality risk. Early identification of an FBI also may help find the source of the disease and prevent further dissemination. The authors comprehensively review foodborne illness, highlighting characteristics that may be utilized to recognize and diagnose this disease process.

— The Editor

Foodborne Illness: Common Biological Contaminants and Pathogens

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Introduction

Foodborne illness (FBI) is a symptom or constellation of symptoms that are largely gastrointestinal. The clinical picture may vary widely from patients who suffer a minor inconvenience to those with life-threatening presentations. FBI results from ingestion of food or consumption of water that is contaminated. Various food groups can be vehicles for transmission of FBI. *(See Table 1.)*

A wide net must be cast to identify causative agents in patients who become symptomatic from ingestion of contaminated food or water. Some agents of foodborne illness rapidly cause symptoms, whereas other agents may take days or even weeks to be evident. This confounds attempts to identify the

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source of the illness. A single patient with symptoms that are suggestive of FBI is unlikely to be recognized unless the treating physician applies considerable skill and judgment. Identification of the source of illness is important for the individual patient and accurate identification of a source of illness may prevent illness in other patients. This article raises awareness for biological contaminants and pathogens that are common in transmission of FBI.

Epidemiology

In 2004, 76 million people in the United States suffered from, 325,000 were hospitalized for, and 5000 individuals died as a result of foodborne disease.[2-5] It is projected that only 1-10% of foodborne illnesses are reported.[6] Hence, FBI may be an underreported contributor to 200,000 annual pediatric hospital admissions and 1.5 million emergency department (ED) visits for "acute gastroenteritis."[1,7] After microbiological diagnosis of several foodborne agents of disease, the hospital admission rate is 14%.[8]

Identification of Foodborne Illness (FBI)

The medical community can facilitate recognition of FBI. This can be globally accomplished by active surveillance of clusters of symptoms, and unusual spikes of similar complaints in the patient population. In the process of determining which disease process is at play for an individual patient with gastrointestinal complaints, physicians should ask specific questions while obtaining the history of the present illness.

Diet. Eating habits modify the patient's vulnerability to foodborne illnesses. Those who prepare their own foods, practice safe food handling techniques, and drink from treated community water systems are less likely to become ill. Those who lack food preparation skills (cross contaminate raw and cooked foods, inadequately heat foods), exhibit poor personal hygiene (fail to wash hands after handling food products), and ingest untreated water are more vulnerable to illness.[9] Those who dine in licensed restaurants, cafeterias, or delicatessens are at increased risk for foodborne illness compared to eating at home. Licensing does not assure that food safety practices are enforced. Nearly 94% of foodborne outbreaks in the United States from 1993 to 1997 occurred in licensed establishments. Foodstuff purchased from unlicensed food services or street vendors magnifies the risk for FBI.[10,11]

The food industry has impacted FBI. Poultry and ground beef have high rates of bacterial contamination.[10] The bacteria are often antibiotic-resistant strains due to the food industries use of antimicrobial agents in feed.[12,13] Agricultural production adjacent to livestock has led to contamination. A 2006 spinach contamination with *E. coli* was attributed to adjacent livestock.[14] When perishable foods are contaminated and widely distributed, as in the 2006 *E. coli* O157:H7 Taco Bell outbreak, there is nationwide exposure to these pathogens in the areas of ultimate distribution.

Parental eating habits influence the pediatric population. In recent years, parents have sought organic or healthy alternatives to processed or mass-produced foodstuffs. Small-batch food products from local farms have been brought with increased frequency into the home. Children have joined their parents in drinking unpasteurized milk or juice, and consumption of cheeses from unpasteurized milk. They have consumed raw shellfish and undercooked meats.

Asking patients with potential FBI the following focused questions may be enlightening:

1. What food and liquids have you consumed recently?
2. Has anyone else consumed the same food or drinks?
3. Has a co-consumer developed any symptoms?
4. Have any of the foods been:
   a. Home canned?
   b. From street vendors?
   c. From restaurants?
   d. Prepared by anyone you know to be ill?
   e. Left out for long periods at room temperature?
   f. Served beyond a perishable date of expiration?
5. Have any of the foods been:
   a. Honey?
   b. Unwashed fruits or vegetables?
   c. Raw or undercooked?
   d. Prepared from a wild animal?
   e. Unpasteurized juices, milk, or cheese from species including cow, sheep, or goat?

Pediatric Emergency Medicine Reports™ (ISSN 1082-3344) is published monthly by AHC Media LLC, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

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GST Registration No.: R128870672

Periodicals Postage Paid at Atlanta, GA 30304.

POSTMASTER: Send address changes to ***Pediatric Emergency Medicine Reports***, P.O. Box 740059, Atlanta, GA 30374.

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Table 1. Contaminated Vehicles for Transmission of FBI (Common to Uncommon)

MEAT	FISH	MILK/CHEESE	EGGS/POULTRY	FRUIT/VEGETABLE/ NUTS	GRAIN/PASTRIES	WATER/JUICES
Staphylococcal toxin	Norovirus	*Salmonella* spp.	*Salmonella* spp.	Staphylococcal toxin	*B. cereus*	Norovirus
Salmonella spp.	Ciguatera	*Shigella* spp.	*Campylobacter* spp.	Norovirus	Staphylococcal toxin	Rotavirus
Campylobacter spp.	Scombroid	*Campylobacter* spp.	Staphylococcal toxin	Rotavirus	*Salmonella* spp.	*Salmonella* spp.
B. cereus	Hepatitis A	*Yersinia* spp.	*C. perfringens*	ETEC	STEC	*Campylobacter* spp.
L. monocytogenes	*Salmonella* spp.	EHEC	*Shigella* spp.	*Salmonella* spp.		*Shigella* spp.
C. perfringens	*V. parahaemolyticus*	*B. cereus*	*L. monocytogenes*	*Shigella* spp.		*Yersinia* spp.
Yersinia spp.	*Plesiomonas* spp.	*C. perfringens*		*Cryptosporidium* spp.		ETEC
EIEC	*V. cholerae*	*L. monocytogenes*		*Cyclospora* spp.		Hepatitis A
STEC	*V. vulnificus*	GABHS		*C. botulinum*		*Cryptosporidium* spp.
Toxoplasma spp.	Shellfish toxins	*Cryptosporidium* spp.		*Giardia* spp.		*Giardia* spp.
	C. botulinum	*Brucella* spp.		*L. monocytogenes*		*E. histolytica*
		STEC				*V. cholerae*
		M. bovis				

Key: ETEC = enterotoxigenic *E. coli;* EIEC = enteroinvasive *E. coli,* STEC = Shiga toxin-producing *E. coli;* EHEC = enterohemorrhagic *E. coli;* GABHS = group A beta-hemolytic *Streptococcus*

Travel. Exposure to contaminated food or water may be increased with "unsafe" travel. "Unsafe" travel is defined as housing in unfavorable conditions and consumption of foods and liquids with unpredictable standards for cleanliness. Recreational activity during travel can intensify risk. Those who camp, visit a petting zoo, aquarium, or farm where there is handling of reptiles or amphibians are at increased risk for FBI. Those who swim in lakes and rivers are at increased risk for water-borne pathogens.[15,16] Children taken to regions that are endemic for cholera, typhoid, and hepatitis also are at risk for FBI. International travel, particularly to resource-poor countries, intensifies risk.[17,18]

Questions directed to uncover patient travel and recreational exposure include:

1. Have you traveled recently?
2. Have you camped out?
3. Have you consumed lake or stream water?
4. Have you been exposed to reptiles, amphibians, or fowl?

Other Predispositions to FBI. Beyond food, travel, and play, other variables may change the vulnerability of a pediatric patient to FBI. Age has an impact. Those younger than age 5 are at increased risk for illness from pathogenic *E. coli,* including the development of hemolytic uremic syndrome. Small children and infants, who attend daycare, are in proximity to others with fecal incontinence. This predisposes them to specific pathogens associated with diarrheal illness.

In your questioning, pursue recent environmental changes and underlying medical conditions. They predispose patients to two specific pathogens associated with FBI. *(See Table 2.)*

Pathophysiology

The gastrointestinal tract possesses several nonimmunologic barriers that prevent illness from tainted food or water. Potential foodborne pathogens are largely eradicated in the environment of high gastric acidity. Indigenous intestinal flora compete for nutrients of potential pathogens. Nonpathogenic bacteria also lower intestinal pH and produce fatty acids, which inhibit the growth of pathogens. Mucin secreted by the intestine decreases the likelihood of bacterial adherence to the epithelial cells of the gut. Peristalsis minimizes the contact between the mucosal surface and a potential pathogen. It aids to expel pathogenic organisms that are nonadherent to epithelial cells. Further, peristalsis prevents retrograde migration of organisms from the colon to the small intestine.[19]

The gastrointestinal tract is further protected by immunologic defense. Secretary IgA is locally produced when an antigen is exposed to the intestinal surface. Toxins are bound and bacterial growth is inhibited.[20]

Systemic and local defense mechanisms can be overcome to produce foodborne illness. The manifestations result from a toxin or an infection, or a combination of invasion and elaboration of toxin may occur.

Preformed Toxins. Preformed toxin-related FBI results from

Table 2. Predisposition to Foodborne Illness

PREDISPOSITION	COMMON AGENTS
Travel	
International	*E.coli, C. perfringens, Yersinia spp., Vibrio cholerae, Vibrio parahaemolyticus, Aeromonas* spp., *Entamoeba* spp.
Wilderness	*Giardia* spp., *Cryptosporidium* spp.
Environment	
Daycare	Rotavirus, astrovirus, calicivirus, norovirus, *Campylobacter* spp.,*Shigella* spp., *Giardia* spp., *Cryptosporidium* spp.
Swimming, Marine	*Aeromonas* spp.
Swimming, Freshwater	*Giardia* spp., *Campylobacter* spp., *Entamoeba* spp.
Swimming, Pools	*Shigella* spp., *Cryptosporidium* spp.
Domestic animals	*Campylobacter* spp., *Salmonella* spp.
Underlying Medical Illness	
Cystic fibrosis	*Giardia* spp.
Liver disease	*Plesiomonas* spp.
Malignancy	*Plesiomonas* spp.
Hemolytic anemia, sickle cell disease	*Salmonella* spp.
Immunocompromised	*Giardia* spp., *Salmonella* spp., *Cryptosporidium* spp., *Entamoeba* spp.

one of three ways: 1) the toxin is produced by bacteria; 2) the toxin is a natural chemical in a foodstuff; or 3) the toxin is acquired in the course of the food chain.

In the preformed bacterial toxin-mediated illness, a food is contaminated during poor hygienic practice. The food then sits inadequately refrigerated, and the toxin is produced. The ingestion of the preformed toxin leads to symptoms.[21] Common examples include staphylococcal toxin mediated illness in which ingestion of a contaminated high protein content food, such as mayonnaise-containing salad or custard pastry, causes illness, and *B. c*ereus emetic toxin from contaminated rice and potato mix. Another circumstance is "classic" botulism food poisoning, where a home preserved and canned fruit or vegetable is not heated adequately in a pressure cooker.[22] The food is contaminated with spores and toxins are elaborated. Ingestion of the toxin leads to a wide range of illness severity.

Natural chemical constituents are found in various vegetables that are not safe or appropriate for human consumption. Examples include green potatoes, unripe akee fruit, bitter cassava, and various mushrooms of the *Amanita* spp. Inadequate refrigeration of fish permits bacterial growth on the fish flesh. This leads to transformation of histidine to histamine, and no method of food preparation can make it safe to eat (scombroid poisoning).

Consumption of some marine fish or mollusks leads to preformed toxin exposure. Toxins produced by reef algae may get incorporated into large tropical and semitropical fish (ciguatera poisoning) or into filter-feeding shellfish.[23]

Toxins Produced In Vivo. Toxins can be produced within the patient.[24] In the case of certain bacteria, a vegetative cell can be ingested and germinate in the patient's gut. The circumstances surrounding this typically involve a delay between food preparation and the eating of a meal. An example would be meat contaminated with *Clostridium perfringens* that slowly cooled or was left to sit on steam tables that permitted sporulation. Similarly, inadequate refrigeration of meat, dairy, poultry, or boiled rice contaminated with *Bacillus cereus* permits intestinal elaboration of a diarrheal toxin.[25] Ingested *Clostridium botulinum* spores can germinate in the patient's gut, releasing toxin (infantile botulism).[26]

Host Invasion. FBI may be caused by an infectious invasion of the intestine by bacteria, viruses, or protozoa. These agents are transmitted by direct contact with humans or animals that harbor these organisms. They are transmitted by the fecal-oral route, or by contaminated food or water. The mode of transmission is dependent on the particular pathogen and the host immunity. Bacteria that gain entry to the gut have fimbria or pili that allow them to adhere to the mucosa. Adherence causes destruction of the microvilli brush border in underlying cell cytoplasm.[27] Viruses damage the absorptive cells of the microvilli of the small bowel, thereby decreasing the available surface area for absorption of fluid and electrolytes. Shortening and blunting of these villi cause transient malabsorption until the microvilli are repaired. Protozoans attach to the mucosa of the small intestine where they cause blunting of the villi. There is variable invasion of colonic glands, and in some circumstances protozoa may enter the lamina propria layer of the colon. Examples of host invasion causing FBI are enteroinvasive *E. coli* and *Salmonella* food poisoning. Examples of common viral and protozoan agents include rotavirus, norovirus, *Giardia* spp., and *Entamoeba* spp.[28]

Host Invasion and Enterotoxin. Some agents that produce FBI can invade mucosal cells, creating cellular damage or death as well as elaborate toxins. The toxins can stimulate intestinal secretion of fluid and electrolytes, alter smooth muscle activity in the intestine, cause release of neurotransmitters, and produce systemic effects. Examples of dual toxicity are *Vibrio parahaemolyticus* (from ingestion of raw or uncooked crab, oysters, shrimp, or lobster) and enteroaggrative *Escherichia coli* (contaminated water, dairy, or meat products).

Table 3. Causes of FBI with Hyperacute Manifestations (< 6 hours)

CAUSATIVE AGENT	PROMINENCE OF MANIFESTATIONS				
	Fever	N/V	Abdominal Pain	Diarrhea	Other
B. cereus emetic toxin	-	+++	+	+/-	--------
Scombroid	-	-	++	++	Headache, flushing
S. aureus toxin	-	+	++	+	Headache, prostration
Cigua toxin	-	+	++	+	Headache, sweating
Shellfish (neurotoxin, paralytic, diarrheal, amnesic)	-	+	+	+	Headache, tingling, numbness, myalgia, paralysis
Amanita mushrooms	-	+++	+++	-	Hallucinations
Pufferfish	-	+	-	-	Perioral paresthesia, dizziness, weakness

Key: N/V = nausea and vomiting

Table 4. Causes of FBI with Acute Manifestations (6-24 hours)

CAUSATIVE AGENT	PROMINENCE OF MANIFESTATIONS				
	Fever	N/V	Abdominal Pain	Diarrhea	Other
B. cereus enterotoxin	-	+/-	++	+++	--------
C. perfringens	-	-	+++	+	--------
V. parahaemolyticus	-	+	+	+++	--------
Norovirus	+	+	+	++	--------
Rotavirus	+++	+	-	+++	--------
C. botulinum	-	+	-	+	Blurred vision, dry mouth, dysarthria, descending paralysis

Key: N/V = nausea and vomiting

Clinical Features

Clues to FBI may come when a prominent symptom or constellation of symptoms occur with a suggestive travel history, dietary history, family contacts, or zoonotic exposure. The chief complaint or pattern and evolution of symptoms may help discriminate the likely cause of FBI.

Chief Complaint. Most patients with FBI present with an isolated or a combination of gastrointestinal complaints.[28] Common symptoms include anorexia; nausea; non-bilious vomiting; non-foul-smelling, watery diarrhea, with or without passage of mucus and/or blood; tenesmus; and abdominal pain. Less common symptoms include constitutional symptoms such as malaise, fever, chills, or prostration. Uncommon symptoms include aches (back, joints, muscles), paresthesia, visual impairment (inability to focus, "wandering eyes," diplopia), difficulty speaking, difficulty ambulating, paralysis, or seizures. Some symptoms, such as increased thirst, weakness, blunted affect, and decreased urine output, result from dehydration.

History of Present Illness. FBI presents in one of four patterns: acute gastrointestinal (GI), chronic gastrointestinal, acute neurologic, or histamine syndrome.

Acute GI. The most common pattern of FBI is an acute syndrome with a duration of 1-2 days. The caretaker and patient are cognizant of the time that illness begins. In hyperacute cases of FBI (within 6 hours of exposures) and in acute cases (6-24 hours), the first manifestations are dramatic and typically are gastrointestinal. *(See Tables 3 and 4.)* The illness may be heralded by one of three symptoms of varying frequency, depending upon the underlying cause. Most often, there is a constellation of gastrointestinal symptoms.

Vomiting occasionally may be concurrent with or follow abdominal pain, but most often vomiting precedes abdominal pain. The vomitus is first of undigested food. No matter how protracted, emesis remains nonbilious. Anorexia and nausea are prominent associated symptoms.

Abdominal pain is of variable intensity ranging from mild to severe. It often is generalized or periumbilical. The pain is crampy in nature. Occasionally, stool passage is a painful, aggravating factor. Generally, pain is alleviated somewhat by assuming a left lateral decubitus, curled-up posture and by passage of flatus or stool.

Diarrhea is an uncommon, isolated gastrointestinal symptom with FBI. Increased stool water and increased frequency of stooling generally follow abdominal pain and/or vomiting. The stools are non-foul smelling and contain no visible mucus or blood, especially if caused by a foodborne agent or viral pathogen. Mucoid or bloody diarrhea can follow infection with invasive pathogens that damage the lining of the bowel.

Chronic GI. Less commonly, there is a more gradual onset of

Table 5. Causes of FBI with Insidious Manifestations (1-2 days)

CAUSATIVE AGENT	PROMINENCE OF MANIFESTATIONS				
	Fever	N/V	Abdominal Pain	Diarrhea	Other
Salmonella spp.	++	+	++	++	--------
Shigella spp.	+++	+	+	+++	Tenesmus
Campylobacter spp.	++	+	+	+ (bloody)	--------
Yersinia spp.	+	+	+++	++ (bloody)	--------
V. cholerae spp.	-	-	-	++++	--------
V. vulnificus spp.	+	-	+	+++	--------
EIEC	++	+	+	+++	--------

Key: N/V = nausea and vomiting; EIEC = enteroinvasive *E. coli*

Table 6. Causes of FBI with Delayed Manifestations (> 2 days)

CAUSATIVE AGENT	PROMINENCE OF MANIFESTATIONS				
	Fever	N/V	Abdominal Pain	Diarrhea	Other
STEC	+/-	-	+++	+ (bloody)	--------
GABHS	++	++	++	-	--------
Cryptosporidium spp.	+/-	+	+	+++	Fatigue
Cyclospora spp.	+/-	+	+	+++	Fatigue
Giardia spp.	-	-	+	+++	--------
Brucellosis spp.	+	-	+	++	Headache, weakness, arthralgia, myalgia
Hepatitis A	+	+	+	-	Jaundice
E. histolytica	+/-	+/-	++	++ (bloody)	Tenesmus
EHEC	+	+	+	+ (bloody)	--------

Key: N/V = nausea and vomiting; STEC = Shiga toxin-producing *E. coli;* GABHS = group A beta-hemolytic *Streptococcus;* EHEC = enterohemorrhagic *E. coli*

not-so-severe manifestations and a duration of 2-4 days. Patients and their caretakers rarely recall the time of day that their illness began. The insidious manifestations can be tied to FBI with a careful history that reveals exposure in the last 1-2 days. *(See Table 5.)* Occasionally, a FBI will take from 2 to 14 days to create symptoms. *(See Table 6.)* The duration of symptoms in chronic FBI may be protracted.

Neurologic. If verbal, the pediatric patient may be able to express abnormal sensations. The sensation of weakness is prominent with botulism, ciguatera, paralytic shellfish, and mercury intoxication. Numbness and tingling of the lips, tongue and throat are seen in paralytic shellfish, pufferfish, and ciguatera. Reversal of hot or cold is seen with ciguatera and neurologic shellfish poisoning.[29,30]

Histamine Syndrome. Within 10 minutes to an hour after ingestion of a contaminated fish, patients with scombroid develop itching or a burning sensation of the skin associated with skin blotches, blisters, or hives.[31,32]

Physical Examination. It is more likely that uncovering the agent causing the FBI will come from the patient's history rather than the physical examination. Several effects of FBI on physical examination findings are discriminatory.

General Appearance. The general appearance will be significantly altered by a patient's pain, and a poor perfusion state. Excessive and distracting pain emanate from head, muscles, joints, or abdomen. Extra abdominal pain is prominent with FBI from *Shigella* spp., *Vibrio parahaemolyticus,* and rotavirus. Patients with FBI from *Yersinia* spp., *Campylobacter* spp., *Clostridium perfringens,* and *B. cereus* diarrheal toxin have heightened abdominal complaints that alter general appearance. Excessive fluid loss through hyperemesis or profusely watery stools blunts the patient's affect, skin turgor, and capillary refilling time. A dehydrated state is far more common with FBI from staphylococcal food poisoning, *Vibrio cholerae,* and *Vibrio parahaemolyticus.*

Vital Signs. Modest increases in heart rate, respiratory rate, or increased depth of respiration reflect acidosis and dehydration that may accompany foodborne illnesses. These vital sign changes are rarely discriminatory. Decreasing respiratory drive, respiratory distress, and/or apnea are discriminatory. These events may occur in the course of botulism or paralytic shellfish poisoning. Hyperpyrexia (≥ 40ºC) in the context of FBI suggests *Shigella* spp. enteritis or *Salmonella* spp. enteritis with occult bacteremia. Extreme tachycardia and/or hypotension suggests septic shock or *Vibrio cholerae.*

Skin. Foodborne hepatitis A is generally nonicteric, but on occasion may cause jaundice. Facial and upper thoracic petechiae are common following forceful vomiting and are nondiscriminating. Diffuse petechiae in the context of FBI suggest hemolytic uremic syndrome. Hives or flushing suggest scombroid poisoning. Typically, flushing is most prominent on the face and upper body, sometimes with sharp demarcation between reddened and unaffected skin. Diaphoresis suggests ciguatera or mushroom poisoning.

HEENT (head, ears, eyes, nose, throat). There can be conjunctival suffusion (increased blood supply and redness from the conjunctival vessels) with scombroid. Decreased salivation and dry mouth occur in botulism. Increased salivation occurs with mushrooms and cadmium poisoning.

Abdomen. Distention and hyperactive bowel sounds are prominent with many causes of FBI. Hepatic percussion tenderness suggests hepatitis A or *Entamoeba* spp. liver invasion. Right upper quadrant pain and mass may accompany *Salmonella* infection as a result of hydrops of the gallbladder. Abdominal voluntary guarding with or without rebound tenderness may be seen in the right lower quadrant with *Campylobacter* spp. or *Yersinia* spp.

Neurologic. Absent or a persistently high-pitched cry, inability to console, poor eye contact, and decreased interaction with the environment are constituents of toxicity. Toxicity is seen with *Vibrio parahaemolyticus, Vibrio cholerae, Shigella* spp., *Listeria monocytogenes,* botulism, and staphylococcal food poisoning. Decreased motor activity, weakness, and hypotonia suggest ciguatera fish poisoning or botulism. Cranial nerve abnormalities, manifested by difficulty with swallowing and vision, with fixed and dilated pupils, are seen with severe botulism. Paralysis of respiratory muscles suggests paralytic shellfish poisoning or severe ciguatera. Ascending paralysis suggests pufferfish poisoning. Descending paralysis suggests botulism.

Diagnostic Studies

In the majority of cases of suspected foodborne illness, diagnostic testing is of limited utility. Laboratory assessments have value in several circumstances.[33]

Dehydration. When there is a history of ≥ 10% acute weight loss, poor oral intake, reduced urine output, significant tachycardia, decreased capillary refilling time or decreased skin turgor and dry mucous membranes, CBC (complete blood count) and BMP (basic metabolic profile) are useful to bolster clinical suspicion of moderate to severe dehydration. The presence of hemoconcentration or electrolyte abnormalities may modify fluid treatment and disposition. Findings of thrombocytopenia or azotemia would require further evaluation for hemolytic uremic syndrome, such as coagulation studies and enzyme immunoassay to detect serum antibodies to *E. coli* 0157:H7 lipopolysaccharide.

Bloody Diarrhea. Patients with frank bloody diarrhea or positive Hemoccult test would benefit from a CBC and stool culture. The presence of leukocytosis does not discriminate likely pathogens, but the presence of a normal total white blood cell count with many immature forms suggests *Shigella* spp., *Salmonella* spp., or *Yersinia* spp. Rectal swab should be submitted for stool culture. The laboratory must be informed that you are trying to isolate *Campylobacter* spp., *Yersinia* spp., *Salmonella* spp., *Shigella* spp., *E. coli* 0157:H7, and *Vibrio* spp.

Acute Abdomen. The presence of shuffling gait, inability to climb onto the examining table, involuntary guarding, rebound tenderness, absent bowel sounds, positive psoas or obturator sign, or abdominal mass constitute manifestations of the "acute abdomen." With the presence of an acute abdomen, the evaluation should be as per the physician's routine for a similar clinical circumstance.[34,35] These typically include submission of urine and appropriate consultation.

Toxic, Hyperpyrexic. Ill appearance should induce the clinician to search for systemic infection. The exact laboratory tests vary by practitioner routine and the patient's clinical state. Young age, immunosuppression, and illness following exposure to an invasive pathogen should be associated with a more aggressive search for invasive disease.

Suspicion for Catastrophic Cause. When there has been a clinical course that is highly suggestive of a particular catastrophic cause for FBI, the diagnosis should be pursued. Examples of diseases include botulism, where botulism toxin should be searched for in the stool. Fluorescent antibody techniques for cholera and *E. coli* 0157:H7 should be pursued in the appropriate clinical context.

Prolonged Course. When a patient has been symptomatic longer than a 3-5 day period and supportive care has not led to resolution of symptoms, the emergency physician should pursue a definitive diagnosis. Laboratory examination should include submission of several sets of stool specimens for culture, and ova and parasite examination.

Public Health. In a case of isolated illness, laboratory evaluation is not particularly cost effective. When an outbreak in the community has occurred, testing benefits the public. Acquire tests of stool for viral, bacterial, and protozoan infection; and serology for hepatitis profiling. The projected contaminated food, if retrieved, can be cultured.

Differential Diagnosis

Patients with FBI present with diverse gastrointestinal complaints. They may present to the ED hours to days after an abrupt onset, or delayed after insidious complaints have not resolved with self treatment. Patients may seek care at a time of escalating symptoms, at a time of plateau, or during a period of resolving complaints. As a result, the appearance and physical findings are highly variable. Given the wide constellation of symptoms, the differential diagnosis is wide ranging. The differential is narrowed when focus is placed on specific elements of the patient's history.

The differential diagnosis for the patient with non-foodborne anorexia, nausea, and abdominal pain who has a prominence of diarrhea includes infectious, inflammatory, structural, and metabolic conditions. These conditions can be divided by the presence or absence of blood in the stool, by either visual or Hemoc-

Table 7. Differential Diagnosis for FBI with Prominence of Diarrhea

NON-BLOODY DIARRHEA	BLOODY DIARRHEA
Infectious	***Infectious***
Occult bacteremia	Bacterial enteropathogens
Viral gastroenteritis	Pseudomembranous enterocolitis
	Parasitic infection
Inflammatory	***Inflammatory***
Disaccharidase deficiency	Ulcerative colitis
Allergic gastroenteropathy	Crohn's disease
	C. difficile colitis
Structural	***Structural***
Hirschsprung's enterocolitis	Intussusception
Ganglioneuroma	
Toxic	
Organophosphate	

cult examination. *(See Table 7.)*

The differential diagnosis for non-foodborne gastrointestinal complaints with a prominence of vomiting includes extraintestinal infection, intraabdominal infection, obstructive disease states, endocrine disorders, and toxic and traumatic conditions. *(See Table 8.)*

The differential diagnosis for non-foodborne gastrointestinal complaints with diffuse neurologic symptoms including visual impairment; increased oral secretions or contrasting, dry mouth; headache; numbness; abnormal sensations including altered heat and cold detection; weakness to paralysis; or seizures are limited to several infectious diseases, intoxicants, or inflammatory or autoimmune diseases. *(See Table 9.)*

Management

Initial Stabilization. In limited circumstances of foodborne illness (i.e., botulism, pufferfish, ciguatera, scombroid, shellfish neurotoxin, bacterial enteropathogens), a life-threatening presentation demands a primary survey directed toward the vital areas of airway, breathing, circulation, and neurologic instability. Perform endotracheal intubation for airway protection in FBI with hypovolemic shock, septic shock, decreased level of consciousness, loss of gag reflex, or prolonged seizure activity. Provide ventilatory support for those with fatigued muscles of respiration or impending respiratory failure. Administer epinephrine and corticosteroids for angioedema. Treat bronchospasm with inhaled beta$_2$ agonists. Infuse intravenous normal saline at 20 mL/kg as rapidly as possible to treat a tenuous perfusion state. With reassessments in between infusions, provide up to 60 mL/kg. If the circulatory status is unresponsive, treat with pressors.

Refined Fluid Therapy. In the majority of patients with FBI, fluid and electrolyte status are the only management concerns. The therapeutic principle for those with dehydration from FBI is to restore perfusion and cellular functioning by providing rehydrating fluids while monitoring the patient's progress. The type of fluid given, route of fluid administration, and speed of administration rests with the initial assessment of the degree of dehydration. Oral glucose-electrolyte rehydration solutions are highly effective in the treatment of mild and moderate dehydration.[36-39] A rapid rate of early repair is provided by giving from 40 to 90 mL/kg of a 75 or 90 mmol Na/L solution over 4-6 hours. This bolus is followed by maintenance solutions with concentrations from 40 to 50 mmol/L. Total daily volumes range from 150 to 250 mL/kg.

Intravenous fluids are given to patients who are unable to tolerate oral rehydration therapy.[40] Intravenous fluids are provided routinely for those with severe dehydration who need rapid restoration of intravascular volume. Intravenous fluids also are suitable for lesser degrees of dehydration. Following bolus therapy of normal saline, 5% dextrose with one-half normal saline is the ideal maintenance fluid beyond the stabilization phase. The rate of fluid administration and the specific content of potassium supplementation should be tempered by serum electrolyte measurements and assessment of urine output. Renal perfusion is assumed to be adequate with the passage of urine at a rate of 1-2 mL/kg/hr.

Other Pharmacotherapy. Various pharmacologic agents may be of utility, whereas others are of marginal benefit or are overtly harmful.

Antiemetics. There are no studies of ondansetron or dexamethasone with FBI. However, patients with repeated vomiting may benefit from either drug.[41,42] In a study of vomiting children with acute gastroenteritis, ondansetron given by mouth or intravenously (0.15 mg/kg to a maximum of 8 mg) prior to oral challenge reduced intravenous fluid rates and hospital admissions.[43] The positive impact of ondansetron with acute gastroenteritis is likely to be more pronounced with patients who have mild to moderate dehydration as defined as a serum $CO_2 \geq 15$ mEq/L.[41] In acute pediatric gastroenteritis, there is an improved tolerance of oral fluids but no reduction in hospital admissions with the use of dexamethasone.[44]

Opioid Agonists. In adults, paregoric, diphenoxylate/ atropine (Lomotil), and loperamide (Imodium) have been shown to diminish fecal urgency, frequency, and volume of diarrheal stools. In children, a scant amount of evidence exists regarding the efficacy of loperamide.[45] In vitro, opioid agonists predispose to ileus and increase the likelihood of intestinal perforation. In uncontrolled studies, paregoric and diphenoxylate/atropine have been noted to prolong fever and duration of diarrhea. Diphenoxylate/atropine has been associated with iatrogenic and accidental death in the pediatric population.[46] Toxic megacolon has been reported with loperamide. Because of the deficiency of evidence for the benefit of these drugs and the potential for harm, none of the opioid agonists are recommended in children.[17,47,48]

Non-opioid Compounds. Kaolin and pectin (Kaopectate) use is without harm but does not reduce the frequency and water content of stools. Bismuth subsalicylate (Pepto-Bismol) decreases intestinal secretions and has some antibacterial effects. Bismuth subsali-

Table 8. Differential Diagnosis for FBI with Prominence of Vomiting

Extra-intestinal infection	**"Obstructive" disease**
Otitis media	Malrotation
Pneumonia	Intussusception
Streptococcal pharyngitis	Hernia
Meningitis	Renal stone
Occult bacteremia	Aerodigestive foreign body
Intraabdominal infection	**Endocrine**
Viral gastritis	Diabetes mellitus
Appendicitis	Adrenal insufficiency
Primary peritonitis	**Toxic**
Mesenteric adenitis	Caustic ingestion
Urinary tract infection	Heavy metal intoxication
Pelvic inflammatory disease	**Traumatic**
Pancreatitis	Black widow bite
Biliary tract disease	Occult trauma

Table 9. Differential Diagnosis for FBI with Prominence of Neurologic Complaints

INFECTION	INTOXICATION	INFLAMMATORY/AUTOIMMUNE
Echovirus 071	Nitrites	Guillain-Barré syndrome
West Nile virus	Cadmium	Tick paralysis
St. Louis equine virus	Mercury	Multiple sclerosis
Eastern equine virus	Lead	Periodic paralysis
California virus	Organophosphates	Myasthenia gravis
Herpes simplex virus	Nicotine	Lambert-Eaton syndrome
Herpes zoster		
Rabies		
Wound botulism		
Epstein-Barr virus		

cylate has shown positive in vitro effects with several organisms including *Salmonella* spp., *Campylobacter* spp., *C. difficile,* and norovirus.[49] Bismuth subsalicylate has been shown to be effective with patients with enteritis secondary to enterotoxigenic *E. coli.*

Antihistamines. Intravenous diphenhydramine and/or cimetidine may shorten the course and ameliorate the symptoms of scombroid poisoning.[50]

Osmotic Agents. Intravenous mannitol (1 g/kg infused over 0.5-4 hours), if provided within the first 24 hours of the onset of ciguatera poisoning, may improve neurologic manifestations caused by cigua toxin.[23]

Antitoxin. An equine-derived antitoxin is available from the CDC to treat patients with foodborne botulism. The trivalent toxin can prevent the progression of neurologic dysfunction if administered early in the course of the illness.[28] No antitoxins are available for any of the shellfish toxins, tetrodotoxicity of pufferfish poisoning, Shiga toxin, or non-botulism food poisonings. Antitoxin is of no benefit in infantile botulism.

Antibiotics. Antimicrobials may be of theoretical, but unproven benefit, supported by clinical experience, or contraindicated by clinical outcomes. Antimicrobials are theoretically indicated in the management of infantile botulism, as there is an active infection from *C. botulinum.* However, it is difficult to measure the clinical impact of intravenous penicillin, the drug of choice. Response as measured clinically (reduction in fever, abdominal discomfort, or diarrhea) and bacteriologically (reduce stool shedding or eradication of organisms from the bloodstream) is seen with antibiotic treatment of *Campylobacter* spp., enterotoxigenic *E. coli, C. difficile, V. cholerae,* and amebic dysentery. Positive clinical and bacteriologic response is less clear with *Yersinia* spp. Antibiotic treatment fails to hasten clinical recovery and may prolong the period to resolution with *Salmonella* spp.[51,52]

Disposition

The majority of patients with FBI evaluated in the ED are suitable for immediate discharge or discharge home after assessment and/or management of fluid depletion. There are occasions, however, when admission to the hospital is advisable. They include toxic appearance; expectant antibiotic therapy for occult bacteremia or documented extraintestinal focus of infection; severe dehydration; laboratory abnormalities that include electrolyte imbalance, azotemia, hypoglycemia, thrombocytopenia, hemolysis, neutropenia; failed oral challenge in the ED; concern for acute abdomen; neurologic impairment; past medical history, such as immunocompromise; psychosocial, such as chronic poor parenting, acute parental illness, long distance to hospital, and inadequate transportation or follow-up; and need for continued healthcare observation.

Surveillance and Reporting

Foodborne disease surveillance and reporting are important public actions. The ED is a participant in the process. When an outbreak is suspected, the ED staff should contact by phone or e-mail the local or county health department in the jurisdiction of the event. The regional health department will investigate and forward a written case report to the state health department.

Should a diagnosis of FBI be confirmed in the ED, the state health department requests notification. Reporting requirements are mandated by individual state regulation. Details on specific state reporting requirements are available from state health departments. Additional information is available electronically at www.cste.org/nndss/reportingrequirements.htm and www.cdc.gov/epo/dphsi/phs/infdis2003.htm.

Members in the state health department implement disease control measures. They will conduct a more thorough epidemiologic investigation. The state coordinates laboratory testing that is unavailable locally. They may enact closure of local food establishments or alter central distribution of foodstuffs. The out-

come is a curtailment of the outbreak. The state will collaborate with the Centers for Disease Control and Prevention (CDC). The CDC acts as a common agent for collecting information and reporting of nationally notifiable diseases. Reports of these diseases are transmitted to the CDC each week and provisional data are published in the *Morbidity and Mortality Weekly Report*.

Summary

Foodborne diseases frequently occur and are associated with multiple pathogens. The affected patient may present with a wide array of signs and symptoms. It is often unclear if a patient has foodborne illness based on history, physical, and readily available diagnostic tests. Supportive care reduces morbidity. The emergency physician should report potential FBI to local health authorities when two or more patients present with an illness that may have resulted from ingestion of a common food or fluid. Public health authorities may be able to curtail further outbreak of foodborne illness.

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CME Questions

91. A 6-year-old male presents with 4 hours of crampy abdominal pain and nonbilious vomiting. Onset followed consumption of an egg salad sandwich at a picnic. Foodborne illness should be considered when:
    A. There is no known outbreak of gastrointestinal illness within the boy's community.
    B. The food consumed smelled "off."
    C. Another person who consumed the same food becomes ill.
    D. When a CBC, urinalysis and abdominal x-rays are negative.

92. A 5-year-old female presents with a two-day history of nausea, periumbilical abdominal pain, and 7-8 episodes of non-bilious vomiting and multiple episodes of non-bloody diarrhea, with the last episode of vomiting upon arrival to the ED. The mother had recent "stomach flu." All family members consumed bottled beverages and had grilled hamburgers before the onset of symptoms. Examination reveals a temperature of 100.5º orally, heart rate 160, respiratory rate 24, blood pressure 100/60, capillary refilling time 3 seconds. Her abdomen is soft, nontender. The most appropriate initial management would be:
    A. No diagnostics; oral rehydration in the ED.

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge.

To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a credit letter. When your evaluation is received, a credit letter will be mailed to you.

CME Objectives

The CME objectives for *Pediatric Emergency Medicine Reports* are to help physicians:

a.) Quickly recognize or increase index of suspicion for specific conditions;

b.) Describe the epidemiology, etiology, pathophysiology, historical and physical examination findings associated with the entity discussed;

c.) Correctly formulate a differential diagnosis and perform necessary diagnostic tests;

d.) Apply state-of-the-art therapeutic techniques (including the implications of pharmacologic therapy discussed) to patients with the particular medical problems discussed;

e.) Provide patients with any necessary discharge instructions.

B. BMP, IV fluids in the ED.
C. BMP, x-ray views of the abdomen; IV fluids in the ED.
D. BMP, blood and stool cultures; IV fluids as inpatient.

93. A 12-year-old male presents with a 12-hour history of anorexia, nausea, three episodes of nonbilious vomiting and increasingly severe abdominal pain. He had no exposure to contagion. He had consumed chicken salad along with other family members, who remain well. Exam reveals a temperature of 99ºF orally, heart rate 88, respiratory rate 16, blood pressure 106/60, capillary refilling time 2 seconds. He has a nondistended abdomen with voluntary guarding in the right lower quadrant. There is no rebound tenderness. The most appropriate initial management is:
A. No diagnostics; oral rehydration in the ED.
B. CBC, BMP; IV fluids in the ED.
C. CBC, BMP, surgical consultation; IV fluids in the ED.
D. Measure Staphylococcus aureus toxin in a stool specimen.

94. An 11-year-old female, who spent the night with her scout troop at a campsite, presents with a six-hour history of periumbilical abdominal pain and grossly bloody diarrhea. Vital signs are as follows: Temperature 97.5º orally, heart rate 100, respiratory rate 16, blood pressure 94/58. She is nontoxic, comfortable and has a benign abdominal exam. The patient's bloody diarrhea could not be associated with which of the following?
A. Enteroinvasive *E. coli.*
B. *Shigella* spp.
C. *Staphylococcus aureus* toxin.
D. *Campylobacter* spp.

95. An adolescent presents with numbness of the face, tingling of the lips, nausea and vomiting after eating at a church-sponsored festival. Vital signs are as follows: Temperature 98º orally, pulse 68, respiratory rate 16, blood pressure 106/74. She has normal cranial nerves, symmetrical motor strength and deep tendon reflexes. Her manifestations are most suggestive of:
A. Guillain-Barré syndrome.
B. Myasthenia gravis.
C. Botulism.
D. Ciguatera intoxication.

96. A 4-month-old male is brought for evaluation of lethargy. The child had been previously well with the exception of constipation. His development has been normal. His immunizations are up to date. He had spent the previous three days with grandmother while mother was on business trip. Grandmother and mother are in attendance and seem appropriately concerned. Vital signs reveal a temperature of 100º rectally, heart rate 116, respiratory rate 32 and unlabored, blood pressure 90/50 and brisk capillary refill. The patient makes poor eye contact and has generalized hypotonia. The abdomen is non-tender. The additional history that is most likely to uncover the cause of his condition is:
A. Recent head injury.
B. Drinking formula that was left out overnight.
C. Drinking tea with honey.
D. Exposure to an invasive pathogen.

97. Which of the following FBIs is associated with nausea, vomiting, perioral paresthesias, dizziness, and weakness?
A. Saureus
B. Scombroid
C. Pufferfish
D. Yersinia

98. Which of the following may be associated with itching or burning sensation of the skin, associated with skin blotches, blisters, or hives?
A. EHEC
B. GABHS
C. EIEC
D. Scombroid

99. Which of the following agents should *not* be used in children with diarrhea?
A. Acetaminophen
B. Ibuprofen
C. Pedialyte
D. Diphenoxylate/atropine

100. Which of the following may be beneficial to a patient with ciguatera poisoning?
A. Antitoxin
B. Mannitol
C. Diphenhydramine
D. Cimetidine

Answers: 91. C; 92. B; 93. C; 94. C; 95. D; 96. C; 97. C; 98. D; 99. D; 100. B

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In Future Issues: **Sports-related Injuries**

PEDIATRIC Emergency Medicine Reports
The Practical Journal of Pediatric Emergency Medicine

Foodborne Illness

Contaminated Vehicles for Transmission of FBI (Common to Uncommon)

MEAT	FISH	MILK/CHEESE	EGGS/POULTRY	FRUIT/VEGETABLE/NUTS	GRAIN/PASTRIES	WATER/JUICES
Staphylococcal toxin	Norovirus	*Salmonella* spp.	*Salmonella* spp.	Staphylococcal toxin	*B. cereus*	Norovirus
Salmonella spp.	Ciguatera	*Shigella* spp.	*Campylobacter* spp.	Norovirus	Staphylococcal toxin	Rotavirus
Campylobacter spp.	Scombroid	*Campylobacter* spp.	Staphylococcal toxin	Rotavirus	*Salmonella* spp.	*Salmonella* spp.
B. cereus	Hepatitis A	*Yersinia* spp.	*C. perfringens*	ETEC	STEC	*Campylobacter* spp.
L. monocytogenes	*Salmonella* spp.	EHEC	*Shigella* spp.	*Salmonella* spp.		*Shigella* spp.
C. perfringens	*V. parahae-molyticus*	*B. cereus*	*L. monocytogenes*	*Shigella* spp.		*Yersinia* spp.
Yersinia spp.	*Plesiomonas* spp.	*C. perfringens*		*Cryptosporidium* spp.		ETEC
EIEC	*V. cholerae*	*L. monocytogenes*		*Cyclospora* spp.		Hepatitis A
STEC	*V. vulnificus*	GABHS		*C. botulinum*		*Cryptosporidium* spp.
Toxoplasma spp.	Shellfish toxins	*Cryptosporidium* spp.		*Giardia* spp.		*Giardia* spp.
	C. botulinum	*Brucella* spp.		*L. monocytogenes*		*E. histolytica*
		STEC				*V. cholerae*
		M. bovis				

Key: ETEC = enterotoxigenic *E. coli;* EIEC = enteroinvasive *E. coli,* STEC = Shiga toxin-producing *E. coli;* EHEC = enterohemorrhagic *E. coli;* GABHS = group A beta-hemolytic *Streptococcus*

Predisposition to Foodborne Illness

PREDISPOSITION	COMMON AGENTS
Travel	
International	*E.coli, C. perfringens, Yersinia spp., Vibrio cholerae, Vibrio parahaemolyticus, Aeromonas* spp., *Entamoeba* spp.
Wilderness	*Giardia* spp., *Cryptosporidium* spp.
Environment	
Daycare	Rotavirus, astrovirus, calicivirus, norovirus, *Campylobacter* spp.,*Shigella* spp., *Giardia* spp., *Cryptosporidium* spp.
Swimming, Marine	*Aeromonas* spp.
Swimming, Freshwater	*Giardia* spp., *Campylobacter* spp., *Entamoeba* spp.
Swimming, Pools	*Shigella* spp., *Cryptosporidium* spp.
Domestic animals	*Campylobacter* spp., *Salmonella* spp.
Underlying Medical Illness	
Cystic fibrosis	*Giardia* spp.
Liver disease	*Plesiomonas* spp.
Malignancy	*Plesiomonas* spp.
Hemolytic anemia, sickle cell disease	*Salmonella* spp.
Immunocompromised	*Giardia* spp., *Salmonella* spp., *Cryptosporidium* spp., *Entamoeba* spp.

Causes of FBI with Hyperacute Manifestations (< 6 hours)

CAUSATIVE AGENT	PROMINENCE OF MANIFESTATIONS				
	Fever	N/V	Abdominal Pain	Diarrhea	Other
B. cereus emetic toxin	-	+++	+	+/-	--------
Scombroid	-	-	++	++	Headache, flushing
S. aureus toxin	-	+	++	+	Headache, prostration
Cigua toxin	-	+	++	+	Headache, sweating
Shellfish (neurotoxin, paralytic, diarrheal, amnesic)	-	+	+	+	Headache, tingling, numbness, myalgia, paralysis
Amanita mushrooms	-	+++	+++	-	Hallucinations
Pufferfish	-	+	-	-	Perioral paresthesia, dizziness, weakness

Key: N/V = nausea and vomiting

Causes of FBI with Acute Manifestations (6-24 hours)

CAUSATIVE AGENT	PROMINENCE OF MANIFESTATIONS				
	Fever	N/V	Abdominal Pain	Diarrhea	Other
B. cereus enterotoxin	-	+/-	++	+++	--------
C. perfringens	-	-	+++	+	--------
V. parahaemolyticus	-	+	+	+++	--------
Norovirus	+	+	+	++	--------
Rotavirus	+++	+	-	+++	--------
C. botulinum	-	+	-	+	Blurred vision, dry mouth, dysarthria, descending paralysis

Key: N/V = nausea and vomiting

Causes of FBI with Insidious Manifestations (1-2 days)

CAUSATIVE AGENT	PROMINENCE OF MANIFESTATIONS				
	Fever	N/V	Abdominal Pain	Diarrhea	Other
Salmonella spp.	++	+	++	++	--------
Shigella spp.	+++	+	+	+++	Tenesmus
Campylobacter spp.	++	+	+	+ (bloody)	--------
Yersinia spp.	+	+	+++	++ (bloody)	--------
V. cholerae spp.	-	-	-	++++	--------
V. vulnificus spp.	+	-	+	+++	--------
EIEC	++	+	+	+++	--------

Key: N/V = nausea and vomiting; EIEC = enteroinvasive *E. coli*

Causes of FBI with Delayed Manifestations (> 2 days)

CAUSATIVE AGENT	PROMINENCE OF MANIFESTATIONS				
	Fever	N/V	Abdominal Pain	Diarrhea	Other
STEC	+/-	-	+++	+ (bloody)	--------
GABHS	++	++	++	-	--------
Cryptosporidium spp.	+/-	+	+	+++	Fatigue
Cyclospora spp.	+/-	+	+	+++	Fatigue
Giardia spp.	-	-	+	+++	--------
Brucellosis spp.	+	-	+	++	Headache, weakness, arthralgia, myalgia
Hepatitis A	+	+	+	-	Jaundice
E. histolytica	+/-	+/-	++	++ (bloody)	Tenesmus
EHEC	+	+	+	+ (bloody)	--------

Key: N/V = nausea and vomiting; STEC = Shiga toxin-producing *E. coli;* GABHS = group A beta-hemolytic *Streptococcus;* EHEC = enterohemorrhagic *E. coli*

Differential Diagnosis for FBI with Prominence of Diarrhea

NON-BLOODY DIARRHEA	BLOODY DIARRHEA
Infectious	***Infectious***
Occult bacteremia	Bacterial enteropathogens
Viral gastroenteritis	Pseudomembranous enterocolitis
	Parasitic infection
Inflammatory	***Inflammatory***
Disaccharidase deficiency	Ulcerative colitis
Allergic gastroenteropathy	Crohn's disease
	C. difficile colitis
Structural	***Structural***
Hirschsprung's enterocolitis	Intussusception
Ganglioneuroma	
Toxic	
Organophosphate	

Differential Diagnosis for FBI with Prominence of Vomiting

Extra-intestinal infection	**"Obstructive" disease**
Otitis media	Malrotation
Pneumonia	Intussusception
Streptococcal pharyngitis	Hernia
Meningitis	Renal stone
Occult bacteremia	Aerodigestive foreign body
Intraabdominal infection	**Endocrine**
Viral gastritis	Diabetes mellitus
Appendicitis	Adrenal insufficiency
Primary peritonitis	**Toxic**
Mesenteric adenitis	Caustic ingestion
Urinary tract infection	Heavy metal intoxication
Pelvic inflammatory disease	**Traumatic**
Pancreatitis	Black widow bite
Biliary tract disease	Occult trauma

Differential Diagnosis for FBI with Prominence of Neurologic Complaints

INFECTION	INTOXICATION	INFLAMMATORY/AUTOIMMUNE
Echovirus 071	Nitrites	Guillain-Barré syndrome
West Nile virus	Cadmium	Tick paralysis
St. Louis equine virus	Mercury	Multiple sclerosis
Eastern equine virus	Lead	Periodic paralysis
California virus	Organophosphates	Myasthenia gravis
Herpes simplex virus	Nicotine	Lambert-Eaton syndrome
Herpes zoster		
Rabies		
Wound botulism		
Epstein-Barr virus		

Supplement to *Pediatric Emergency Medicine Reports*, October 2007: "Foodborne Illness: Common Biological Contaminants and Pathogens." *Authors:* **Jonathan I. Singer, MD, FAAP, FACEP,** Associate Program Director for Emergency Medicine, Professor of Emergency Medicine and Pediatrics, Boonshoft School of Medicine, Wright State University, Dayton, OH; **Jason Pickett, MD,** Resident Physician, Emergency Medicine Residency, Boonshoft School of Medicine, Wright State University, Dayton, OH; **Pat Chhuon, MD,** Resident Physician, Emergency Medicine Residency, Boonshoft School of Medicine, Wright State University, Dayton, OH. *Pediatric Emergency Medicine Reports' "Rapid Access Guidelines."* Copyright © 2007 AHC Media LLC, Atlanta, GA. **Senior Vice President and Group Publisher:** Brenda Mooney. **Editor-in-Chief:** Ann Dietrich, MD, FAAP, FACEP. **Associate Publisher:** Lee Landenberger. For customer service, call: **1-800-688-2421.** This is an educational publication designed to present scientific information and opinion to health care professionals. It does not provide advice regarding medical diagnosis or treatment for any individual case. Not intended for use by the layman.