Preanesthetic Assessment of Children With Food Protein–Induced Enterocolitis Syndrome

PREANESTHETIC ASSESSMENT

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LEARNING OBJECTIVES:

At the conclusion of this activity, the reader should be able to:

1. Define food protein–induced enterocolitis syndrome (FPIES)
2. Name the foods that can trigger FPIES episodes
3. Learn about the epidemiology and pathophysiology of FPIES
4. Classify the different types of FPIES
5. Discuss the acute and chronic clinical manifestations of FPIES
6. List the differential diagnoses
7. Describe diagnostic tools
8. Outline the treatment of acute episodes
9. Describe the management of patients with FPIES presenting for surgery
10. Recognize the possible effect of propofol administration in patients with FPIES
Continuing Medical Education

CASE

A 2-year-old girl who weighs 10 kg, with a history of severe food protein–induced enterocolitis syndrome (FPIES), presented for outpatient esophagastroduodenoscopy with biopsies. She is allergic to many foods, including corn-containing products, milk-related compounds, rice, soybean, artificial food dye No. 40, oranges, strawberries, and sunflower oil. Her mother stated that her child had been hospitalized twice for severe FPIES episodes, with protracted vomiting, diarrhea, and dehydration that required IV line placement and hydration for 2 weeks on each occasion. Her mother also stated that an epinephrine injection had not worked at home during these episodes. The child’s last attack was a few months earlier, but she had been doing well by avoiding all trigger foods. She had been born full term by vaginal delivery without complications. There had been no recent upper respiratory infections. She had undergone one anesthetic experience for the same procedure without complications, although the record was not immediately available. Laboratory results indicated mild anemia and hypoalbuminemia but otherwise were within normal range.

Background

FPIES is an underrecognized and frequently misdiagnosed type of food allergy that affects the gastrointestinal (GI) tract, with onset during the first year of life (mean age of initial presentation, 5.5 months). Although it is rarely diagnosed after the first year of life, it can occur at any age.

It is characterized by severe vomiting and/or diarrhea. The typical FPIES is a non–immunoglobulin E (IgE)-mediated food hypersensitivity. The atypical form, comprising 24% of cases, refers to patients who have the classic features of FPIES but have detectable IgE levels to the offending foods. FPIES is more common in boys (54%-60%) than girls.

Classic allergic symptoms of the skin, such as urticaria, pruritus, or angioedema, and of the respiratory tract, such as cough, wheezing, or dyspnea, are typically absent. Workup for sepsis is generally negative. Acute FPIES manifests as profuse, repetitive vomiting (10-20 episodes), often with diarrhea, leading to severe dehydration and possibly shock. In the chronic form, intermittent vomiting and diarrhea cause weight loss and failure to thrive.

Despite the severe reaction that may occur during an acute attack, FPIES can be considered a self-limiting disease since patients are asymptomatic if they are not exposed to the offending foods. In both forms, removal of the offending foods allows resolution of symptoms within a few days.

The most common triggers are cow’s milk and soy, followed by rice and oats. Other foods that can cause FPIES include eggs, poultry (chicken and turkey), meat, beans, vegetables (white potato, sweet potato, and squash), tomatoes, fruits, legumes (peanuts, green peas, lentils, and string beans), different cereals, and seafood. FPIES due to fish or shellfish is seen mainly in older children and adults.

Of note, different countries report distinct percentages of the population affected, types of trigger foods, and times of resolution of symptoms. For example, in the United States, 50% of FPIES patients react to both cow’s milk and soy, while in Australian and Israeli studies, there are no reported cases of patients reacting to both foods. This finding may be due to different feeding practices and genetic factors. FPIES due to eggs is rare in the United States, but it was reported in 10% of an Australian cohort study. An example of the different times of resolution is seen for FPIES due to cow’s milk. In a US referral population, the time of resolution is 50% by 3 years, whereas in an Israeli population cohort, a 90% resolution rate was reported at the same age.

Exclusively breast-fed children are not exposed to cow’s milk and soy, which are found in many formulas. As a result, allergic reactions may not develop until introduction of these foods later in the first year of life—although rare. Some authors have suggested that breast-feeding is actually protective against FPIES due to cow’s milk and soy since there are no reports of FPIES in exclusively breast-fed infants. The mechanism for this is unknown but may be due to the presence of IgA antibodies in breast milk. In addition, the allergen dose of the breast milk proteins may not reach the trigger threshold of acute FPIES.

Tolerance to offending foods usually develops around 5 years of age and is established by 5.1 years for cow’s milk, 6.7 years for soy, 4.7 years for rice, and 4 years for oats. These times of onset differ from patients with detectable IgE food allergies. These children usually have more protracted courses that last for years. Moreover, patients with FPIES are at increased risk for developing IgE-mediated food allergies later in life.

The exact prevalence of FPIES is unknown. In a large Israeli study, cow’s milk FPIES prevalence was 0.34%, while the prevalence of IgE-mediated cow’s milk allergy was 0.5% in the same study population. The prevalence of FPIES due to other foods is largely unknown.

The majority of patients (65%) react to one food, 26% of them react to 2 foods, and 9% to 3 or more foods, whereas very few react to 6 or more.

Risk factors include cesarean delivery, male gender, FPIES due to another food, and early introduction of cow’s milk. Thirty percent of patients with FPIES have atopy in the form of atopic dermatitis, allergic rhinitis, or asthma, among other morbidities.
Pathophysiology

The exact mechanism of FPIES is not completely understood. It may represent a defect in the protective and immunologic function of the intestinal tract. It is believed that ingestion of food allergens leads to T-cell activation that causes local intestinal inflammation. This inflammation may enhance the intestinal penetrability of the causative allergens and enhance their presentation to the immune system.1,4

Activation of other cells, such as peripheral mononuclear cells, regulatory T cells, eosinophils, neutrophils, and thrombocytes, has been observed.5 Leukocytosis and thrombocytosis are often evident, and they are part of the overall evaluation despite their nonspecific nature.1 Intestinal inflammation is caused by the release of proinflammatory mediators such as tumor necrosis factor-alpha and interferon-gamma. These mediators, in turn, increase intestinal permeability and fluid loss, leading to the various signs and symptoms of FPIES.

In addition to the inflammatory cascade, involvement of humoral and neuroendocrine pathways has been postulated. The latter mechanism was suggested because of the effectiveness of ondansetron in treating FPIES symptoms. Ondansetron, which is a serotonin receptor antagonist, reduces central and peripheral vagus nerve activity.5

The vagus nerve is increasingly recognized as an important tool in the inflammation process. Its afferent fibers sense the inflammatory signals and convey them to the brain. At the same time, the efferent fibers modulate the inflammatory response by controlling acetylcholine release from T cells. These cholinergic signals activate the alpha-7 nicotinic acetylcholine receptor on immune cells.11

Classifications

FPIES is classified as either acute or chronic according to the acuity of symptoms. Acute FPIES clinical reactions start 1 to 3 hours after ingestion of the trigger food. In the case of an acute attack that happens after reintroduction of the offending foods in a known patient with chronic FPIES, classification is labeled as an “acute on top of chronic” attack.1

Chronic FPIES occurs when a patient is exposed to the culprit food on a regular basis. Patients with chronic FPIES suffer from intermittent vomiting, diarrhea, malabsorption that leads to dehydration, weight loss, and failure to thrive. Anemia and hypoalbuminemia are encountered in these patients. They will remain symptom-free if they are not exposed to the trigger foods.1,2,5

Clinical Manifestations

Clinical manifestations depend on the dose and frequency of the ingested trigger food. An acute attack occurs if the patient is exposed to the allergenic food for the first time, reintroduced to it after a period of abstinence, or after an oral food challenge (OFC) test.

The acute severe FPIES attack manifests as profuse, repetitive, and sometimes projectile vomiting (10-20 episodes) often with diarrhea, leading to acute dehydration and possibly shock.1,8 The onset of vomiting is typically 1 to 3 hours, while diarrhea is delayed 5 to 10 hours after ingestion of the culprit food.7 Vomiting is the most common presentation of acute FPIES and occurs in almost all patients (95%-100%), followed by lethargy, pallor, dehydration, diarrhea, and hypothermia (Table 1).3,5 Patients appear acutely ill, ashen gray, and listless.5

Chronic FPIES develops when the patient ingests the allergenic food on a regular basis. Patients with chronic FPIES suffer from intermittent vomiting, diarrhea, malabsorption that leads to dehydration, weight loss, and failure to thrive. Anemia and hypoalbuminemia are encountered in these patients. They will remain symptom-free if they are not exposed to the trigger foods.1,2,5

Table 1. Differences Between Acute and Chronic FPIES

<table>
<thead>
<tr>
<th>Acute FPIES</th>
<th>Chronic FPIES</th>
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<tbody>
<tr>
<td>Repetitive vomiting (95%-100%)</td>
<td>Intermittent vomiting</td>
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<tr>
<td>Lethargy (75%-85%)</td>
<td>Diarrhea</td>
</tr>
<tr>
<td>Pallor (67%)</td>
<td>Lethargy</td>
</tr>
<tr>
<td>Dehydration (15%-20%)</td>
<td>Pallor</td>
</tr>
<tr>
<td>Diarrhea (25%-40%), bloody if severe</td>
<td>Dehydration</td>
</tr>
<tr>
<td>Hypotension and shock, if severe (15%-20%)</td>
<td>Weight loss</td>
</tr>
<tr>
<td>Abdominal distention, if severe</td>
<td>Failure to thrive</td>
</tr>
<tr>
<td>Metabolic acidosis, if severe</td>
<td>Abdominal distention</td>
</tr>
<tr>
<td>Hypothermia &lt;36°C (&lt;25%)</td>
<td>Limpness</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>Dusky appearance</td>
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FPIES, food protein–induced enterocolitis syndrome
Diagnosis

Delay in diagnosis may result from the nonspecific clinical features, lack of specific allergy tests, broad differential diagnosis, the perception that grains and vegetable proteins are hypoallergenic (in the case of solid-food FPIES), and the unfamiliarity of this condition among health care providers. It may take up to 5 episodes of FPIES, which may translate to an 8-month delay, before reaching the diagnosis.4

No specific pathognomonic biomarkers or radiological findings are used to diagnose FPIES. Thus, the diagnosis is made by excluding other causes and is largely based on history, clinical criteria, and an OFC test.1-8

The original diagnostic criteria listed by Powell are:

- age younger than 9 months at initial presentation (reaction);
- exposure to the culprit foods that elicit repetitive vomiting and/or diarrhea within 4 hours without any other cause for the symptoms;
- symptoms limited to the GI tract;
- avoidance of the offending food protein that results in resolution of symptoms; and
- a standardized food challenge or isolated reexposure that elicits the typical symptoms.

A food-specific IgE antibody is typically undetectable. Neutrophils are increased in all patients, and the increase is included in the diagnostic criteria for the acute attacks and after OFC tests. Neutrophilia and met-hemoglobinemia, although nonspecific, are supportive for the diagnosis. Skin prick tests are typically negative; atopy patch tests have mixed results, and they are generally not used in establishing the diagnosis.1,2

The OFC test is considered the “gold standard” for diagnosis of FPIES.1-8 The test is not necessary for confirmation, but it is necessary during follow-up care to evaluate the possibility of reintroduction of  

<table>
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<tr>
<th>Table 2. Diagnostic Tests for FPIES and Their Relative Value Are Compared</th>
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<tbody>
<tr>
<td><strong>Diagnostic Test</strong></td>
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<tr>
<td>--------------------------</td>
</tr>
<tr>
<td>OFC</td>
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<tr>
<td>Leukocytosis</td>
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<tr>
<td>Thrombocytosis</td>
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<tr>
<td>Food-specific IgE antibody</td>
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<tr>
<td>Skin prick tests</td>
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<tr>
<td>Atopy patch tests</td>
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<tr>
<td>Fecal tests</td>
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<tr>
<td>Intestinal biopsies</td>
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<tr>
<td>Methemoglobinemia</td>
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<tr>
<td>Metabolic acidosis</td>
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<tr>
<td>C-reactive protein</td>
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<tr>
<td>Radiological findings</td>
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<tr>
<td>Gastric juice leukocytosis</td>
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<tr>
<td>Anemia and hypoalbuminemia</td>
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<tr>
<td>Peripheral blood eosinophilia</td>
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</table>

FPIES, food protein–induced enterocolitis syndrome; IgE, immunoglobulin E; OFC, oral food challenge
offending foods. The test is usually delayed 12 to 18 months after the acute attack to check the development of tolerance. It is done under medical supervision in a controlled setting after IV cannulation.

Radiological changes are nonspecific and include air–fluid gut levels, nonspecific narrowing and thumbprinting of the rectum and sigmoid colon, thickening of the plicae circulares, and intramural gas. Thumbprinting is a radiographic sign of wall thickening of the large bowel, usually caused by edema, and is related to an infective or inflammatory process (colitis). The normal haustra become thickened at regular intervals, appearing like thumbprints projecting into the aerated lumen. These signs may result in misdiagnosis during an attack of FPIES, instead resulting in suspicion of an acute abdominal condition, such as appendicitis, and lead to unnecessary surgery.

Methemoglobinemia is produced by increased heme oxidation caused by the elevation of nitrites. The latter is the result of reduced catalase activity associated with severe intestinal inflammation.

Endoscopic biopsies are not routinely required, except in persistent severe chronic disease that is not responding to diet modification. Biopsies aim to exclude other GI pathology. Various diagnostic tests and their value in establishing the diagnosis are listed in Table 2. Differential diagnoses are listed in Table 3. Rapid and complete resolution of symptoms after IV hydration is a characteristic of acute FPIES and typically not seen in other conditions, such as sepsis, metabolic disorders, or surgical emergencies.

Treatment

The mainstay of management for chronic FPIES is avoidance of the culprit food. Infants who have FPIES due to cow’s milk and/or soy may be either exclusively breast-fed or switched to hypoallergenic formulas, such as casein hydrolysate or amino acid–based formulas. They usually return to a normal state of health in 3 to 10 days after eliminating the offending foods from the diet. Patients should be followed by OFC tests every 12 to 18 months to evaluate the possibility of reintroduction of the allergenic foods and development of tolerance.

Due to the unfamiliarity of FPIES in urgent care centers, letters explaining the presentation and management of FPIES, provided to the families, can be helpful. In particular, parents must be advised to carefully read the contents of food items. For example, one might not expect peas to be included in ice cream, but it can be an ingredient in vegan compounds of iced foods.

For acute FPIES, management includes the following:

- The first line of treatment is hydration requiring vigorous IV resuscitation (10-20 mL/kg) of balanced electrolyte solution for severe attacks or oral rehydration, if tolerated, for mild ones.
- The second line of treatment is 1 mg/kg per day of methylprednisolone (maximum, 60–80 mg), which is recommended for severe reactions in order to decrease presumed cell-mediated intestinal inflammation.
- Bicarbonate is used to treat metabolic acidosis.
- Vaspressors such as epinephrine are given for severe hypotension or shock, but they are not effective in improving the symptoms of vomiting and lethargy.
- Ondansetron (0.2 mg/kg/dose) has been shown to be effective in treating emesis along with IV fluids.
- Methylene blue (1-2 mg/kg) should be administered to treat methemoglobinemia.
- Oxygen supplementation is indicated in patients with cyanosis, shock, or those with severe methemoglobinemia.

Table 3. FPIES May Be Confused With Several Other Pathologic Conditions

<table>
<thead>
<tr>
<th>Differential Diagnoses of FPIES</th>
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<tbody>
<tr>
<td>Allergic proctocolitis and enteropathy</td>
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<tr>
<td>Anaphylaxis</td>
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<tr>
<td>Congenital methemoglobinemia</td>
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<tr>
<td>Eosinophilic gastroenteropathies</td>
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<tr>
<td>Food poisoning</td>
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<tr>
<td>Gastroenteritis (viral or bacterial)</td>
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<tr>
<td>Gastroesophageal reflux disease</td>
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<tr>
<td>Hirschsprung disease</td>
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<td>Idiopathic pyloric hypertrophy</td>
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<tr>
<td>Ileus</td>
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<tr>
<td>Inflammatory bowel disease</td>
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<tr>
<td>Intussusception</td>
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<tr>
<td>Metabolic disorders</td>
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<tr>
<td>Necrotizing enterocolitis</td>
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<tr>
<td>Neurologic disorders</td>
</tr>
<tr>
<td>Primary immunodeficiency disorders</td>
</tr>
<tr>
<td>Sepsis</td>
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<tr>
<td>Volvulus</td>
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FPIES, food protein–induced enterocolitis syndrome
Preoperative Management

Patients with acute FPIES may be scheduled for diagnostic surgeries such as exploratory laparotomy or radiological procedures (computed tomography, magnetic resonance imaging, etc). In chronic disease, patients may present for endoscopic procedures or for unrelated surgeries. History should include emphasis on any complications from anesthesia in the extended perioperative period and not just the immediate post-anesthesia care unit (PACU) stay. Previous anesthesia records should be reviewed, including medications given. Any recent OFC results also should be reviewed.

Children with atopy may be at greater risk for developing adverse respiratory reactions under general anesthesia because of the increased risk for laryngospasm, bronchospasm, breath holding, and desaturation. Latex precautions should be in place for children who may have FPIES due to foods with cross-reactivity to latex, such as bananas, kiwis, and avocados.

Laboratory tests that should be reviewed include electrolytes, complete blood count, and protein levels. In the case of a recent acute attack, electrolytes should be monitored and corrected before proceeding to surgery. Intravenous cannulation should be accomplished in the ward or emergency department before induction of anesthesia to ensure adequate hydration and allow any indicated drug administration. Levels of methemoglobin should be measured by a co-oximeter, and methylene blue should be given if the level of methemoglobin exceeds 20%.

Vasopressor support, such as epinephrine, is used to treat severe hypotension and shock. Patients may be started on antibiotics if infection is suspected, and corticosteroids may be administered to treat the presumed intestinal inflammation. Patients may need additional doses of ondansetron (0.1–0.2 mg/kg) if vomiting persists.

Patients with active vomiting during an acute attack are considered to have a full stomach and be at risk for aspiration. Rapid sequence induction is conducted in these cases. All necessary emergency medications and equipment should be readily available. Surgeries are mostly performed during an acute attack if FPIES is misdiagnosed as an acute abdominal condition or if a known patient with FPIES does not improve after fluid resuscitation and there is suspicion of another underlying pathology. Otherwise, no surgeries should be performed during acute attacks.

Intraoperative Management

In addition to standard monitoring by the American Society of Anesthesiologists, continuous blood pressure monitoring by arterial cannulation is helpful in guiding resuscitative measures and vasopressor titration in severe cases. In addition, the monitor can be used for frequent laboratory tests such as arterial blood gas analyses, electrolytes, acid–base deficit, and lactate levels.

As an induction agent, propofol is of concern because of its soy and egg yolk contents. Propofol is an alkylphenol derivative (2,6-diisopropylphenol) that contains soybean oil (10%) and egg lecithin (1.2%). Lecithin is a highly purified egg yolk phosphatide that is not the allergenic component. Most patients with egg allergy are allergic to the egg white component. The soybean oil is refined during propofol preparation by removing the allergenic proteins.

Documented anaphylactic reactions to propofol have been attributed to its isopropyl or phenol groups. As a result, propofol can be used safely in non-FPIES patients with soy and egg allergies. This might not be the case in susceptible soy (and maybe egg) FPIES patients, as propofol may cause typical acute attacks and should be used cautiously. All other induction and inhalation agents are considered safe. Antibiotics, muscle relaxants, and other commonly used anesthesia medications have been used successfully in patients with FPIES.

Patients undergoing an acute attack (eg, a patient who has been misdiagnosed as suffering from an acute abdominal condition and is now undergoing laparotomy) also are at high risk for developing hypotension after induction if they are not resuscitated adequately. Generous maintenance IV hydration should be continued intraoperatively in addition to replacement of any loss of blood and urine. Vasopressors should be continued in the intra- and/or postoperative period as indicated. Neuraxial techniques should be avoided in hemodynamically compromised patients.

Postoperative Management

Postoperative medical care of patients with FPIES with acute severe attacks should be resumed in an intensive care unit setting, especially those receiving vasopressors or who are hemodynamically unstable.

Patients who are scheduled for outpatient surgeries should be observed for an adequate time in the PACU before discharge. Should symptoms develop after discharge, parents should be aware to bring the patient back for further medical management immediately. Any new allergies should be documented in the patient’s medical record.

Case Management

After taking the history and reviewing the laboratory results, the patient was transferred to the operating room. General anesthesia was induced with sevoflurane in oxygen and nitrous oxide. A vein was cannulated and sedation continued with propofol boluses (total, 50 mg). The procedure went well and the recovery period appeared unremarkable. The patient was discharged home once the discharge criteria were met.

Three hours later, the patient developed repetitive vomiting and diarrhea. She was returned to the hospital and admitted for IV hydration for 2 days because of typical FPIES exacerbation.
Conclusion

Our patient was among the rare cases who had documented allergies to more than 6 foods. Although propofol has not been associated with allergic symptoms in non-FPIES patients who are allergic to soy, it was the presumed medication that caused the adverse symptoms in this patient since it was the only one given other than sevoflurane.

Fortunately, the majority of anesthesia medications can be used safely in patients with FPIES. This case shows that a careful medical history, review of the anesthesia records, and familiarity of this condition are paramount in conducting safe anesthesia in patients with FPIES.

The FPIES Foundation is available to supply further information to families and health care workers (www.thefpiesfoundation.org). It networks with other important and related organizations that all provide information, including:

- the American College of Allergy, Asthma, and Immunology
- Food Allergy Research & Education
- the Allergy and Asthma Foundation of America
- the Food Allergy Support Group of Minnesota
- the American Academy of Allergy, Asthma & Immunology
- the National Institute of Allergy and Infectious Diseases
- AllergyHome.org

FPIES was recently given an official diagnosis code: K52.21.

References

Post-Test:

1. The most common presentation of acute food protein–induced enterocolitis syndrome (FPIES) is:
   a. hypotension
   b. vomiting
   c. bradycardia
   d. pallor

2. Treatment of acute FPIES includes all of the following, except:
   a. IV hydration
   b. ondansetron
   c. vasopressors
   d. platelet transfusion

3. FPIES is typically diagnosed by medical history and:
   a. fecal tests
   b. specific immunoglobulin E antibodies
   c. an oral food challenge test
   d. development of thrombocytosis

4. Differential diagnosis of FPIES includes all of the following, except:
   a. croup
   b. sepsis
   c. viral gastroenteritis
   d. necrotizing enterocolitis

5. Which medication contains a soy derivative?
   a. Thiopental
   b. Propofol
   c. Ketamine
   d. Etomidate

6. Which of the following will be an expected finding in patients with severe acute FPIES?
   a. Metabolic alkalosis
   b. Leukopenia
   c. Methemoglobinemia
   d. Thrombocytopenia

7. In acute FPIES, ingestion of an offending food will cause vomiting:
   a. immediately
   b. after 1 to 3 hours
   c. after 24 hours
   d. after 1 week

8. Which of the following is not involved in FPIES pathology?
   a. Neutrophils
   b. Eosinophils
   c. T cells
   d. B cells

9. Which radiological finding is least likely to be present in acute FPIES?
   a. Air–fluid gut levels
   b. Small-bowel distention
   c. Pneumothorax
   d. Thumbprinting of the rectum

10. Which medication is contraindicated in patients with acute FPIES?
    a. Nitroglycerin
    b. Bicarbonate
    c. Methylene blue
    d. Methylprednisolone