CASE REPORT

Food protein-induced enterocolitis syndrome: a challenging diagnosis

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SUMMARY

Food protein-induced enterocolitis syndrome (FPIES) is a non-IgE-mediated gastrointestinal food hypersensitivity triggered by food proteins. It may present acutely, with repetitive vomiting, diarrhoea and lethargy leading to dehydration and eventually shock or insidiously with intermittent emesis, chronic diarrhoea or failure to thrive. We describe a paediatric male patient with recurrent sepsis-like episodes of fever, lethargy, ashen-grey skin colouration and vomiting followed by diarrhoea. These episodes were triggered by cow's milk formula and grains. Laboratory tests revealed leucocytosis, thrombocytosis, metabolic acidosis and elevated C reactive protein. After exclusion of other differential diagnoses, the diagnosis of FPIES was established on clinical improvement with withdrawal of the offending food and positive oral food challenge. FPIES diagnosis requires a high index of suspicion and is frequently delayed, which contributes to an increased morbidity. This is due to the wide spectrum of clinical presentations and due to the absence of specific diagnostic tests.

BACKGROUND

Food protein-induced enterocolitis syndrome (FPIES) is a non-IgE-mediated gastrointestinal food hypersensitivity, usually triggered by cow's milk or soy protein and more rarely, by other dietary proteins.^{1 2} It represents the severe end of a spectrum of food protein-induced gastrointestinal diseases and usually affects infants with a slight male predominance.^{1 3 4} The only two studies reporting incidence or prevalence of FPIES suggest that FPIES may be more common than previously thought: the incidence of cow's milk FPIES was reported as 0.34% in an Israeli birth cohort; incidence of FPIES to any food was reported as 15.4 per 100 000 per year among infants under 24 months of age from an Australian survey study.^{5 6} The immune mechanism underlying FPIES is not completely understood, although a T-cell dependent mechanism leading to local inflammation and increased intestinal permeability with fluid shift may play a role.^{1 7 8} The diagnosis is assumed in the presence of consistent clinical features with improvement following withdrawal of the causative protein. In recent years, an effort has been made to establish clinical guidelines for FPIES. Leonard and Nowak-Węgrzyn published International Consensus Guidelines for the diagnosis and management of FPIES.⁹ The group proposes adjustments to diagnostic criteria based on recent evidence. The diagnosis of FPIES

requires the presence of the major criteria and at least three minor criteria (table 1).

Due to its non-specific symptoms and the absence of specific confirmatory tests, FPIES is often under-recognised initially. This delays the final diagnosis and increases the morbidity associated with extensive investigations, unnecessary treatments and prolonged hospitalisation.^{1 4 10 11} The recognition of the multiple manifestations of FPIES together with increased awareness for this condition by healthcare providers is necessary to improve its management.^{4 10} We report a case of FPIES occurring in response to cow's milk and grains' protein with some peculiar clinical features.

CASE PRESENTATION

A full-term Caucasian male with an unremarkable prenatal, neonatal and family history, presented at 15 hours of life with hypotonia, weak suction and vomiting. A cow's milk-based formula was used to supplement breast feeding since birth. Initial investigation showed hypoglycaemia (31.0 mg/dL), C reactive protein (CRP) concentration of 0.8 mg/dL and a procalcitonin (PCT) concentration of 0.75 ng/mL. Complete blood count, venous blood gas, metabolic and endocrine studies were all normal. The patient had a negative blood culture and a positive urine culture, containing 10⁵ colony-forming units (CFU) of *Escherichia coli*. Intravenous glucose (maximum of 15.5 mg/kg/min) and antibiotics were started together with bowel rest, with gradual improvement. He was discharged at 3 weeks old with exclusive cow's milk-based formula.

At 2 months of age, the patient presented to the emergency department with fever (39.2°C) and food refusal. He was lethargic with an ashen-grey appearance (figure 1), capillary refill time of 3 s and painless abdominal distension. Full sepsis work-up revealed an elevated white cell count (17.85 ×10⁹/L) with neutrophilia (13 390/μL), thrombocytosis (499 000/μL) and elevated CRP and PCT (4.6 mg/dL and 0.75 ng/mL, respectively). Blood, urine and cerebrospinal fluid (CSF) culture were sterile and there were no findings on metabolic screen. At this point, the patient initiated empirical antibiotics for a presumed infection. He also started intravenous fluids and was left with bowel rest for 4 days. Progressive improvement and apyrexia was observed within 3 days. He was discharged home after 8 days with exclusive formula feeding.

Forty-eight hours after discharge, the patient returned to the emergency department, with 1 day

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Table 1 Diagnostic criteria of food protein-induced enterocolitis syndrome⁹

Major criteria	Vomiting in the 1 to 4 hours period after ingestion of the suspect food and absence of classic IgE-mediated allergic skin or respiratory symptoms
Minor criteria	<ul style="list-style-type: none"> ▶ A second (or more) episode of repetitive vomiting after eating the same suspect food; ▶ Repetitive vomiting episode 1 to 4 hours after eating a different food; ▶ Extreme lethargy with any suspected reaction; ▶ Marked pallor with any suspected reaction; ▶ Need for emergency department visit with any suspected reaction; ▶ Need for intravenous fluid support with any suspected reaction; ▶ Diarrhoea in 24 hours (usually 5 to 10 hours); ▶ Hypotension; ▶ Hypothermia.

of profuse watery diarrhoea and fever, without vomiting. Physical examination was similar to the previous episode. Investigation revealed normocytic normochromic anaemia (haemoglobin level 8.6 g/dL) leucocytosis ($19.74 \times 10^9/L$), thrombocytosis ($1653\ 000/\mu L$), metabolic acidosis, elevated CRP (6.28 mg/dL) and elevated PCT (3.84 ng/mL) with normal renal and liver function. CSF analysis revealed pleocytosis (10 leucocytes/ μL , normal glucose and proteins). Abdominal ultrasound showed distension, thickening of the small bowel loops and hyperperistalsis. Blood, CSF and faecal cultures were negative. Urine culture revealed 10^5 CFU of *Klebsiella pneumoniae*, despite normal urinalysis. The patient started broad-spectrum antibiotics, intravenous fluid therapy and was left on bowel rest. Improvement was observed over the next 2 days. Gastrointestinal endoscopies revealed ulceration in the sigmoid. Histology showed oedema of the lamina propria and immune cell infiltrates, composed by a high number of lymphocytes and few eosinophils and neutrophils. Further investigation confirmed a total IgE of 7.39 kU/L,

Figure 1 Ashen-grey appearance of the patient during an acute food protein-induced enterocolitis syndrome episode.

negative-specific IgE and negative skin prick tests to cow's milk protein. After multiple sepsis-like episodes that improved with bowel rest and intravenous fluids, an extensive work-up and exclusion of infectious disease, metabolic diseases and immune deficiency, the diagnosis of FPIES was considered and extensively hydrolysed formula initiated.

At 4 months of age, the diagnosis of FPIES was confirmed after a positive inpatient oral food challenge (OFC) with cow's milk protein. The patient rapidly recovered with intravenous fluid resuscitation and a single bolus of methylprednisolone. After 24 hours of bowel rest with clinical improvement, extensively hydrolysed formula was restarted. One week later, infant cereal containing rice and corn (without milk or gluten) was introduced. Two days after daily intake and 1 hour after the last ingestion, the patient had another sepsis-like episode with hypotension (blood pressure of 55/33 mm Hg), confirming FPIES to grains. After intravenous fluid resuscitation with normal saline bolus (3×20 mL/kg body weight), a single dose of intravenous methylprednisolone (2 mg/kg body weight) and intramuscular epinephrine, the infant was transferred to the intensive care unit (ICU) where he began diarrhoea. Gradual recovery was observed and after 48 hours of bowel rest, extensively hydrolysed formula was restarted. Three days after daily intake and 30 min after last ingestion, feeding with formula elicited similar symptoms and fever was noted during 3 days. Protein hydrolysis was increased to an exclusive amino acid-based formula, which was maintained exclusively until the patient was 6 months of age.

OUTCOME AND FOLLOW-UP

Complementary feeding was progressively introduced as inpatient and was always uneventful. This included the introduction of soy milk at 12 months of age. Hospital-supervised OFC to extensively hydrolysed formula and to cereals were performed at 11 and 21 months of age, respectively, and were both negative. Cow's milk-based formula challenge at 19 and 36 months of age were still positive. At 43 months of age, the patient had an acute FPIES episode with minimal cow's milk amount. Avoidance was resumed, with adequate weight and height development (25th to 50th percentile based on WHO Child Growth Standards). Despite adequate psychomotor development, the patient acquired increased oral feeding aversion and inability to accept new foods. Occupational therapy with sensory integration and speech therapy was started to promote the development of oral motor skills, resulting in clear improvement.

DISCUSSION

FPIES has a broad spectrum of clinical presentations that may overlap with a vast number of diseases. The phenotype and the severity of FPIES are determined by the frequency and dose of the food trigger ingested, together with the population of origin and the age of the patient.^{8 9} It can have an acute or a chronic presentation.^{5 12}

Acute FPIES commonly presents with repeated vomiting, usually occurring within 1 to 4 hours after food ingestion. Vomiting is accompanied by lethargy, pallor or by an ashen-grey appearance. It ultimately leads to dehydration, hypotension and hypovolaemic shock.^{8–10 12 13} Diarrhoea and abdominal distension may follow within 5 to 10 hours, particularly in severe episodes.^{10 12 14} Acute FPIES episodes typically last about 24 hours from the exposure, and children are usually asymptomatic between episodes.^{8 9}

Chronic FPIES appears to be more common in Japan and Korea, is less well characterised and occurs with daily ingestion

of the offending food.^{8 15} It may present in the first days of life and usually occurs before 4 months of age. It is characterised by intermittent emesis, chronic diarrhoea, abdominal distension, dehydration, poor weight gain and failure to thrive in infants fed with cow's milk or soy formula.^{8 9 16} Chronic FPIES disappears after elimination of the protein antigen. Subsequent feeding with the triggering food will induce an acute FPIES episode.^{8 9 16}

Although the most commonly described triggers are cow's milk and soy protein, FPIES may be induced by any solid food, including grains (rice, oats, barley and corn), meat and poultry, eggs, vegetables, fruit and seafood.^{8 11 17} Despite the reported geographic variation, rice is globally the most common solid food trigger.^{9 14} Most infants with solid food-induced FPIES react to more than one food protein, with the majority being formerly identified with cow's milk and/or soy FPIES.^{11 14 17}

FPIES typically presents within the first 12 months of age, although there are rare and severe cases presenting within the first days of life.^{4 14 16} The age of onset depends on the timing of trigger introduction. Cow's milk and soy-induced FPIES occurs at a younger age (before 6 months of age) compared with solid food-induced FPIES (6 to 12 months of age).⁹ In our case, it is not clear if the first episode at birth was a FPIES episode, although this is possible, considering the early introduction of cow's milk protein.

In both acute and chronic FPIES, laboratory tests can reveal anaemia, hypoalbuminaemia, elevated white cell count with a left shift, eosinophilia, thrombocytosis, metabolic acidosis and methaemoglobinaemia.^{10 12 14 17 18} The immune response in FPIES is not IgE mediated. Therefore, skin prick tests and serum food-specific IgE are usually negative at both diagnosis and follow-up.^{8 9} However, specific IgE testing of the trigger food should be considered due to a higher risk of persistent disease associated with comorbid IgE-mediated sensitisation to triggers in FPIES.¹⁹ Stool examination, radiographic tests and endoscopic evaluation do not provide any specific findings and should therefore be used only in differential diagnosis to exclude other diseases.^{9 17} Differential diagnosis is extensive and infection is the most imperative diagnosis to be excluded in acute FPIES.

FPIES represents a 'spectrum of syndromes' and should be recognised as a heterogeneous disorder with phenotypic geographic variation. The diagnosis implies a high index of suspicion and is often delayed due to its rarity and low awareness, the absence of classic allergy symptoms (ie, respiratory and cutaneous reactions), the non-specific and late presentation and the lack of definitive diagnostic tests.^{10 16} The delay in diagnosis is even more frequent in solid food-induced FPIES, since grains and vegetables are considered hypoallergenic and are usually not perceived as potential triggers.^{9 10}

The diagnosis of FPIES relies on the presence of typical symptoms that improve with discontinuation of the suspected trigger, exclusion of other potential diagnoses and, if required, physician-supervised OFC.^{10 13 14 16 17} Currently, there are no laboratory tests available for a definitive diagnosis. Our patient meets the proposed diagnostic criteria for FPIES described in the International Consensus Guidelines.⁹ However, instead of hypothermia, fever was a prominent feature. Although neutrophilia has been reported, neither increased CRP nor fever is consistently observed in these patients and they can both suggest sepsis and delay the final diagnosis.²⁰ Nevertheless, a few recent studies report the presence of fever associated with CRP elevation at FPIES onset, predominantly in Asian populations.^{20 21} In our patient, a positive urine culture despite normal urinalysis was an additional confounding factor. These features reinforce the

heterogeneous clinical presentation of FPIES and the need for a high index of suspicion to establish the correct diagnosis. In this regard, OFC may be key for diagnosing FPIES in some cases. The establishment of clinical diagnostic criteria has to be done with caution, as this is an under-recognised condition, with a possible bias of presenting symptoms reported.

Although OFC is the gold standard for FPIES diagnosis, infants with a typical history usually do not need confirmatory challenges because of the risk of severe reactions.^{9 10 14} OFC should be performed only if the history is ambiguous, a specific food trigger is not recognised, the time course of symptoms is atypical, symptoms persist regardless of presumed offending food exclusion or to determine whether FPIES has been resolved before reintroduction of the trigger food.^{14 16} OFC protocols for FPIES are different from those for IgE-mediated food allergy. In FPIES, the protein challenge is administered in three equal doses over 30 min. This is due to the difficulty in establishing the threshold trigger dose, as acute reactions are consistently delayed. In cases of positive IgE to the challenge food, OFC should be performed according to the IgE-mediated food allergy protocols.^{8 11 13 14}

The management of an acute reaction includes aggressive intravenous fluid resuscitation for patients presenting with hypovolaemic shock which occurs in 15% of cases.^{11 12} For mild to moderate episodes, oral rehydration can be done instead, if tolerated.^{9 14 17} A single dose of intravenous methylprednisolone (1 mg/kg; maximum of 60 to 80 mg) could be administered to decrease the cellular inflammatory response, although more studies are needed to support this attitude.^{9 11} Some patients might require a more aggressive approach, with advanced haemodynamic supportive therapy and admission into the ICU, as seen in this case.⁹ Epinephrine is not generally recommended unless an associated IgE-mediated food allergy is identified.^{12 14} Ondansetron appears to be effective in shortening emesis, but more and better designed trials are needed to determine its safety and efficacy.²²

Long-term management consists of strict dietary elimination of the causative food together with nutritional guidance and an emergency treatment plan for acute accidental exposures.^{4 14} Infants with both cow's milk and soy FPIES should be supplied with casein-based extensively hydrolysed formula, though 10% to 20% may require an amino acid-based formula, as observed in our patient.^{4 9 14 16} For infants with cow's milk FPIES and unknown soy tolerance, soy formula should be offered under medical supervision and vice versa. While concomitant sensitisation to cow's milk and soy has been described in 20% to 40% of US patients, this has been notably absent in reports from other nations. Breast feeding should be continued when possible, as FPIES to proteins through breast milk is rare.^{4 11 14}

The age of resolution of FPIES diverges widely among the literature and seems to depend on the culprit food, the coexistence of atopic disease and the country of origin.^{9 17} Most patients outgrow FPIES by the age of 3.¹⁷ A population-based cohort study performed in Israel revealed that 50% of patients with cow's milk-induced FPIES had tolerance by 1 year, 75% by 18 months, 89% by 2 years and 94% by 3 years.⁵ A more extended course has been reported in patients with solid food FPIES or with positive food-specific IgE.^{8 10 14}

OFC to food trigger should be performed under the physician's supervision to determine FPIES resolution before reintroduction of the food into the diet.^{8 14} The timing of such reintroduction varies and has not been systematically studied. Recent reviews recommend follow-up challenges every 12 to 18 months in patients without recent reactions, although Korean

data suggest that challenges can be performed earlier.^{9 23} It seems reasonable to delay OFC in cases where the quantity of food required to induce symptoms is smaller, as seen in our patient. It might be prudent to perform measurement of serum food-specific IgE levels and/or skin prick tests to determine the timing of OFCs, which should be delayed in case of positive test.^{8 14}

Introduction of solid foods in infants with cow's milk/soy-induced FPIES should not be delayed.^{9 16} In the presence of reaction to multiple foods, complementary food should be postponed until 6 months of age and should be carefully introduced in a supervised environment.^{9 10 13 16} Due to the potential severity of acute episodes as well as strict avoidance requirement for the trigger protein, nutritional counselling should be offered to ensure balanced nutritional intake.^{8 10 14}

Empiric solid food introduction delay should be avoided to minimise nutritional deficits and feeding disorders during infancy. Even with timely introduction of solid foods, many infants have oral feeding aversion related to previous traumatic experiences associated with FPIES reactions, behaviours often enhanced by parental apprehension, as noticed in our case.^{16 24} Counselling parents about strategies for enhancing feeding and oral motor skills is critical in these infants.

Learning points

- ▶ Food protein-induced enterocolitis syndrome (FPIES) is a non-IgE-mediated food allergy triggered by food proteins that remains under-recognised.
- ▶ FPIES diagnosis is challenging and frequently delayed due to the lack of specific clinical and laboratory findings, eventually mimicking a constellation of other disorders. Therefore, it should rely on the history, exclusion of other causes and eventually in an oral food challenge.
- ▶ Establishing specific diagnostic criteria should be made with caution due to variable presentation among different populations.
- ▶ It is imperative to increase awareness to this condition to prevent morbidity from repeated reactions, unnecessary work-ups, treatments and hospitalisations.
- ▶ Further research is required to elucidate the pathophysiology and natural history of FPIES to improve the care of these patients.

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