

Infants with FPIES to Solid Food Proteins - Chicken, Rice and Oats

Abstract:

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Abstract

We present two cases of Food Protein Induced Enterocolitis Syndrome (FPIES), a non-IgE mediated food hypersensitivity¹. FPIES induces severe vomiting 1.5-to-3 hours post ingestion of the offending food, and may be associated with diarrhoea, hypovolemic shock and acidosis. Avoidance of that food will lead to resolution of symptoms and prevents further episodes.

Introduction

FPIES is rare, and more often misdiagnosed². Up to 20% of patients present in hypovolemic shock³. Clinicians are likely to initially suspect sepsis, metabolic conditions, acute surgical abdomen or gastroenteritis. Children may require fluid resuscitation, and potentially HDU/ICU admission. Infants with FPIES will re-present with similar symptoms if the protein is reintroduced. In most, tolerance develops by 2-3 years of age^{1,4}. The pathophysiology and immune mechanisms underlying FPIES are not fully understood.

Case 1

Eight-month-old male presented twice in 3 weeks with hypovolemic shocked following acute onset of vomiting. He required multiple fluid boluses, and admission to HDU. He was acidotic (pH: 7.30), with thrombophilia (611) and neutrophilia (29.6). Subsequent endocrine, metabolic work-up, ultrasound abdomen and barium study were normal. FPIES was suspected. Food challenges to carrot, sweet potato, followed by chicken ensued. Two hours post ingestion of chicken he had severe vomiting and lethargy, requiring I.V. fluids.

Case 2

Seven-month-old male. Weaned with baby rice at 20 weeks. Three days later was admitted with possible urosepsis. At 22 and 26 weeks of age had profound emesis associated with lethargy and diaphoresis 90 minutes post ingestion of baby rice. Delay in weaning occurred, resulting in anaemia and faltering centiles. At 32 weeks he was admitted for food challenge, and to establish safe foods. He similarly reacted to oats, and required I.V. fluids. Once home he reacted to natural yogurt; subsequent investigation revealed possible contamination with rice flour.

Discussion

â Typical FPIESâ describes infants under nine months; repeated exposure to the offending agent elicits gastrointestinal symptoms; removal of the protein from the diet results in resolution of symptoms. A standardized food challenge provokes vomiting and/ or diarrhoea. FPIES is not exclusive to infants under 9 months; presentation after this period is considered â atypicalâ FPIES. The most common foods implicated are cowâ s milk and soy. Infants who present within the first 2 months of life can have chronic diarrhoea, intermittent vomiting, are more likely to have failure to thrive and hypoalbuminemia. Following dietary exclusion, reintroduction of milk or soy will result in an acute FPIES event. Breast milk appears to confer some protection against FPIES to milk or soy. FPIES to solid food proteins occurs later as weaning occurs after 4 months of age. Both our patients reacted to solid food proteins. This often occurs on first exposure. Health professionals and parents consider rice to be relatively hypoallergenic. However rice is emerging as the most common solid food trigger for FPIES, accounting for up to 70% of solid food FPIES⁵. Rice is more likely to induce a more severe reaction than milk or soy⁵. Due to increasing awareness the number of solid food proteins reported to cause FPIES¹ is rising. Cereals (oats, barley, rye), vegetables (squash, sweet potato, peas), and poultry all may cause FPIES. Children with FPIES to milk or soy can later develop FPIES to solid foods.

Clinical features seen in our patients are similar to those already described: vomiting (up to 100%); extreme lethargy (85%, soon after vomiting starts); pallor (67%); diarrhoea (24%, commencing up to 6 hours post ingestion). Other features include a high neutrophil count, thrombocytosis, as seen in Case 1, and transient methaemoglobinemia⁶. Hypothermia has also been reported⁷. No diagnostic immunological markers have been identified. Specific IgE to food proteins involved are typically negative. IgE testing was performed in Case 2, and was normal. Developing IgE against the offending agent appears to reduce their likelihood of developing tolerance. An upper endoscopy is usually not required. Histology has shown various degrees of villous atrophy that are non-specific and are only present during an acute FPIES event.

The diagnosis of FPIES is clinical, based on clinical criteria and standardized oral challenge⁸. As they are considered high risk such challenges are performed under medical supervision. Awareness and early diagnosis reduces recurrence of life threatening episodes through avoidance. A re-challenge should be considered after two years of age to assess for tolerance.

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