

Food Protein-Induced Allergic Proctocolitis (FPIAP): New Insights into Pathogenesis and Implications

Qian Yuan, MD, PhD^{1,*}

¹Food Allergy Center, Massachusetts General Hospital, Harvard Medical School, Boston, MA, 02114, USA

*Correspondence should be addressed to Qian Yuan, qyuan@mgh.harvard.edu

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Abstract

Food protein induced allergic proctocolitis (FPIAP) is one of the earliest presentations of food allergies in infancy. It is a non-immunoglobulin E (IgE)-mediated condition. Although the inflammation is located at the rectosigmoid colon, clinical symptoms are not limited to mucousy, bloody stools and often include gastroesophageal reflux (GER), feeding difficulties, irritability and poor sleep. 16S gut microbiome analyses have demonstrated that dysbiosis of the intestinal microbiota started prior to the onset of FPIAP symptoms and persisted during the active disease phase, suggesting that dysbiosis of the gut microbiome likely plays a role in the pathogenesis of FPIAP. Furthermore, infants with FPIAP have increased risk of developing IgE-mediated food allergies and eosinophilic esophagitis (EoE) later in life, indicating the possible connection between non-IgE-mediated FPIAP and IgE-mediated food allergies. Insights gained in understanding the pathogenesis of FPIAP and its association with other food allergic conditions will not only help in better managing FPIAP but also facilitate designing therapeutic strategies for preventing and treating food allergic conditions and hopefully altering the progress of atopic march.

Keywords: FPIAP, Dysbiosis, IgE, Food allergies, EoE

Introduction

What is FPIAP?

Food Protein Induced Allergic Proctocolitis (FPIAP) is the current accepted term for a non-IgE-mediated food allergic condition in early infancy. FPIAP was previously known as allergic colitis, eosinophilic colitis, cow's milk protein allergy (CMPA), cow's milk protein intolerance (CMPI). The diagnosis is largely based on clinical symptoms, a convincing history and resolution of symptoms with food avoidance [1]. Histological findings of mucosal eosinophilia on rectosigmoid colonic biopsies are seldom used, because the biopsies are rarely performed by pediatric gastroenterologists worldwide. The gold standard confirmatory diagnostic test is a food challenge [2].

The hallmark symptom is mucousy, bloody stools, either hematochezia or the presence of occult blood. Other symptoms often include gastroesophageal reflux (GER), feeding difficulties, irritability, and poor sleeping [3]. In some cases, poor weight gain can also be observed.

Symptoms usually begin within the first three months of life [3,4,10], with a peak onset at 1 month [3]. The symptoms typically resolve by six months [4]. The most common dietary triggers are dairy, egg, soy and corn, with dairy accounting for more than three-quarters of cases [5,6].

The precise prevalence of FPIAP is not well established [1]. Research studies suggest that it is often underdiagnosed [7]. In the first North American study (The Gastrointestinal Microbiome and Allergic Proctocolitis, GMAP) to prospectively assess the prevalence of FPIAP, we reported a cumulative incidence of FPIAP over three years as 17% [3]. In this study, we prospectively enrolled 1,003 healthy newborn infants born between September 2014 and September 2017 from a single pediatric practice in suburban Massachusetts, USA. Diagnosis of FPIAP was made by primary care pediatricians and reviewed by study investigators to confirm inclusion by prespecified criteria, including documented gross or occult blood in the stool without other explanations [3]. However, a study conducted in Israel by Elizur and colleagues [8] on a cohort of 13,234 newborns, born between June 2004 and June 2006

at Assaf Harofeh Medical Center, Zerifin, Israel, and followed until the age of six years, reported that only 0.16 % had FPIAP related to cow's milk consumption. Although the Israeli study focused only on milk-induced FPIAP [8], it is unlikely to account for the difference in estimates, given that milk accounts for the majority of FPIAP cases [7,9]. In a more recent retrospective study from Turkey [10], Senocak and colleagues reported that the frequency of FPIAP was 0.18 % among 64,549 patients over a 10-year period from January 1, 2008, to January 1, 2018. Difference in the study populations may account to the variation in prevalence, and it is also possible that the FPIAP prevalence has increased over the ten years of these studies [1]. Further large-scale studies are needed in this regard.

Atopic march refers to the natural history of allergic diseases as they develop over the course of infancy and childhood [11]. Classically, it begins with atopic dermatitis (AD) and progresses to IgE-mediated food allergy (FA), asthma, and allergic rhinitis (AR) [11]. Each of these conditions carry a complex pathophysiology involving multiple facets of the immune system, in a Th2-predominant immune reaction [11]. Increasing evidence suggests that non- IgE-mediated food allergic diseases are part of the disease spectrum associated with the atopic march [12,13]. Chronologically, FPIAP occurs early in infancy (2 weeks to 2 months) [3,10], followed by atopic dermatitis (eczema), FPIES, IgE-mediated food allergies, and later EoE and asthma. Specifically, children with FPIAP had 1.5 times the likelihood of a history of eczema when compared

with healthy controls [3,14] and had a fivefold increased risk of developing EoE [15]. We believe that FPIAP might be the beginning of the atopic march (**Figure 1**, adapted from reference 12). However, the complex immunological basis of FPIAP and other allergic diseases needs to be further elucidated.

Dysbiosis likely plays a major role in the pathogenesis of FPIAP

There is a growing body of evidence that the gut microbiome plays a key role in the development of IgE- and non-IgE-mediated food allergy [16-23]. The increasing prevalence of food allergies strongly implicates environmental factors – such as antibiotics use, diet, and other exposures – in causing dysbiosis (microbial imbalance), which has already been associated with other forms of pediatric allergy [24,25]. Complex cross-talk between the intestinal microbiome, food antigens, intestinal inflammation, and the innate immune system early in life likely contributes to the mechanisms that determine whether an infant develops healthy immune tolerance or food allergies [26]. In our GMAP prospective FPIAP study, we analyzed 954 longitudinal samples from 160 infants in a nested case-control study (81 who developed FPIAP and 79 matched controls) from 1 week to 1 year of age using 16S rRNA ribosomal gene sequencing [27]. We found key differences in the microbiome of infants with FPIAP, most notably a higher abundance of a genus within the

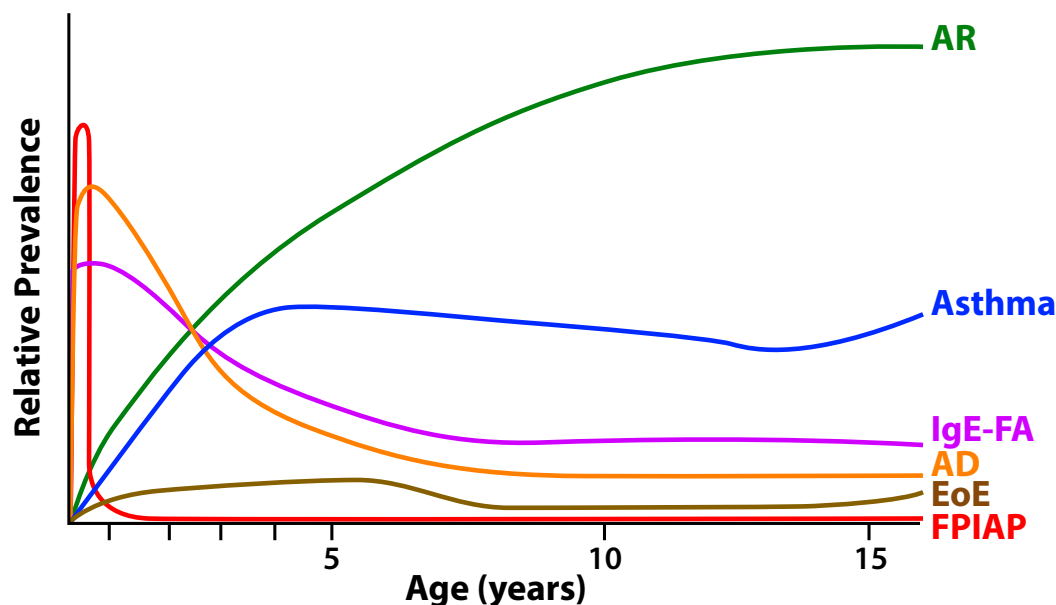


Figure 1. The proposed new atopic march including IgE-mediated atopic disease and non-IgE-mediated food allergy. FPIAP is the earliest manifestation in the atopic march, and EoE is the late manifestation in the march. AR: Allergic Rhinitis; AD: Atopic Dermatitis; EoE: Eosinophilic Esophagitis; FPIAP: Food Protein Induced Allergic Proctocolitis; IgE-FA: Immunoglobulin E-mediated Food Allergy (Adapted from Reference #12).

Enterobacteriaceae family and a lower abundance of a family within the *Clostridiales* during the symptomatic period. Some of these significant taxonomic differences were present even before symptom onset. However, there were no consistent longitudinal differences in richness or stability diversity metrics between infants with FPIAP and healthy controls [27]. A rise in *Lactobacillus* and a decrease in *Blautia* were associated with FPIAP resolution [27]. Our data indicate that the developing infant gut microbiome plays a role in FPIAP development and that intestinal dysbiosis is likely critical in its pathogenesis.

In a study from Greece [4], Vallianatou and colleagues studied 61 infants with FPIAP and found that *Enterobacteriaceae* clusters (dominated by *Klebsiella* and *Shigella*) were most commonly represented in samples from symptomatic infants. In contrast, *Bacteroides* and *Bifidobacteria* clusters emerged later in apparently healthy infants. These findings further support the role of intestinal dysbiosis in FPIAP development.

Our GMAP study suggested that breast feeding is protective against the development of FPIAP [3], but a combination of breast milk and formula feeding (compared with infants exclusively breastfed or exclusively formula-fed) was associated with the least likelihood of developing FPIAP. The precise mechanism behind this observation remains under investigation. Other risk factors for developing FPIAP included eczema and a first degree relative with food allergies [3]. Moreover, Vassilopoulos and colleagues found that maternal diet during pregnancy and breastfeeding influenced the development of FPIAP in a Mediterranean population [5]. Increased maternal consumption during pregnancy and lactation of common allergens, whole grain products, homemade food, fish and shellfish, and fruits was associated with a decreased risk of FPIAP [5]. Conversely, a high intake of vegetables, sugar and total fat, and non-stick/grilled cooking was associated with increased risk of FPIAP, as was a high intake of salt and white flour during lactation only [5]. These intriguing findings suggest that not only breastfeeding itself, but also maternal diet during pregnancy and lactation likely plays a role in FPIAP development in infants. Furthermore, FPIAP appeared earlier in premature infants and with a history of NICU hospitalization [10], presumably due to altered gut flora acquisition and its interruption by antimicrobial treatments in the NICU.

Association of FPIAP and IgE-mediated food allergy

FPIAP is a non-IgE-mediated condition, with T cells thought to play a key role in its pathogenesis [12]. Additionally, cytokines such as pro-inflammatory tumor necrosis factor- α (TNF- α) [28] and anti-inflammatory transforming growth factor- β (TGF- β) have been implicated in FPIAP [29,30]. Eosinophils appear to be a common pathologic feature in non-IgE-mediated food allergic diseases [11]. In patients with FPIAP, rectosigmoid biopsy findings typically reveal acute inflammation with five or more eosinophils per high-power field [31].

Eosinophils play a diverse role in our immune function. Traditionally, they are regarded as amplifiers of type 2 immunity, contributing to immune defense against multicellular parasites (e.g., helminths, ticks) and in hypersensitivity reactions (such as asthma and allergic rhinitis) [32]. Type 2 innate lymphoid cells (ILC2) and TH2 cells produce IL-5, which recruit eosinophils into inflammatory sites and activate the production of eosinophils [33]. ILC2s lack antigen-specific receptors but share functional similarities with the TH2 cells and produce the TH2-associated cytokines IL-4, IL-5, and IL-13 [34,35]. Given the immaturity of the adaptive immune system in early infancy, it is plausible that ILC2s may play a role in FPIAP [36]. Cytokines produced by ILC2s may subsequently activate mast cells, impair allergen-specific Tregs [33], promote eosinophilic inflammation, induce TH2 differentiation, and ultimately drive B-cell class switching to IgE [37,38].

Significant differences in mechanisms and natural history exist for non-IgE-mediated and IgE-mediated food allergies. It is well established that early introduction of allergenic foods can reduce the risk of IgE-mediated food allergies [39]. In our GMAP study of 903 infants (median age: 42 months; range: 24-59 months), 153 (17%) had FPIAP, and 56 (6%) had IgE-mediated food allergies (28 to peanut, 28 to egg, 12 to tree nuts, and 9 to milk) [3,40]. Among children with FPIAP, 11% developed IgE-mediated food allergies compared with 5% without FPIAP [40]. After adjusting for eczema, children with FPIAP had nearly twice the odds of developing IgE-mediated food allergies (OR = 1.9, 95% CI = 1.0-3.6, P = 0.04), with the strongest association observed for milk IgE-mediated allergy (OR = 5.4, 95% CI = 1.4-20.8, P = 0.01) [40].

Consistent with our GMAP study, a more recent study by Tran and colleagues reported that five patients with FPIAP subsequently developed IgE-mediated allergies to cow's milk and other foods (egg, peanut, tree nuts, soy and wheat) [36].

This apparent association between FPIAP and IgE-mediated food allergy raises concerns about current clinical guidelines for FPIAP management – namely, food elimination (particularly dairy elimination) until 1 year of age. Empiric dietary restriction during the first year of life may inadvertently increase the risk of IgE-mediated food allergies later in life. Further studies are needed to elucidate the precise mechanisms linking non-IgE-mediated FPIAP and IgE-mediated food allergies.

Other implications of FPIAP: The neuro-immune axis

An increased prevalence of Disorders of Gut-brain Interaction (DGBI), formerly known as functional gastrointestinal disorders (FGIDs), has been reported retrospectively in infants diagnosed with FPIAP [42]. In our GMAP study, of the 903 participants in the main cohort, 375 had sufficient data on stooling habits for analysis, and 78 had parent-reported constipation. Sixty-three (17%) of these children were confirmed to have evidence of constipation persisting beyond 3 years of age [42]. The average age of constipation diagnosis was 2.77 years [42]. Children with a history of FPIAP had more than twice the odds of developing

constipation after age 3 compared to those without FPIAP (OR=2.62, 95% CI=1.42–4.74, $p=0.002$) [40]. Stimulant laxative use was also more common among children with FPIAP (OR = 4.68, 95% CI = 1.47–16.04, $P=0.01$) [42].

Constipation was not associated with any history of IgE-mediated food allergy ($p=0.9$) [42]. The precise mechanism of this increased constipation in children with a history of FPIAP in infancy is still unclear. Dysbiosis of the gut microbiota may play a role, changes in gut-brain immune axis may also play a role. Due to the presence of gastrointestinal inflammation in early-life producing abnormal rectal sensory thresholds with post-inflammatory changes to the neuro-muscular function of the pelvic floor causing inadequate rectal and abdominal pressure and/or incomplete anal sphincter relaxation during bear-down maneuvers. This would effectively result in pelvic floor dysfunction and increase the likelihood of withholding behaviors; both key contributing factors in the development of functional constipation in childhood.

Additionally, unpublished observations from the GMAP cohort suggest that children with a history of FPIAP in infancy have a significantly higher risk of developing anxiety and obsessive-compulsive disorder (OCD) by age 10.

Taken together, these findings suggest that early-life gut inflammation in FPIAP may have profound effects on both enteric and central nervous system function. However, the precise molecular and immunological pathways underlying this gut-neuronal interaction require further study.

Conclusion

FPIAP is a non-IgE-mediated food allergic condition that occurs in early infancy, typically within the first three months of life. It is associated with the presence of eczema and a family history of allergies. Dysbiosis of the gut microbiota likely plays a key role in the development of FPIAP. Children with a history of FPIAP in infancy have a higher likelihood of developing IgE-mediated food allergies, EoE, constipation, and anxiety/OCD, indicating possible immunological associations between non-IgE and IgE-mediated food allergic conditions, as well as gut-brain neuroimmune connections. Finally, FPIAP likely represents the first step in the atopic march. A better understanding of the pathogenesis and disease mechanisms of FPIAP will likely facilitate our understanding of allergic condition development and aid in designing novel therapeutic interventions for both the prevention and treatment of food allergies and other allergic conditions.

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QY is the sole contributor for this manuscript in reviewing articles, writing, and editing.

Conflicts of Interest

None.

IRB Approval

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