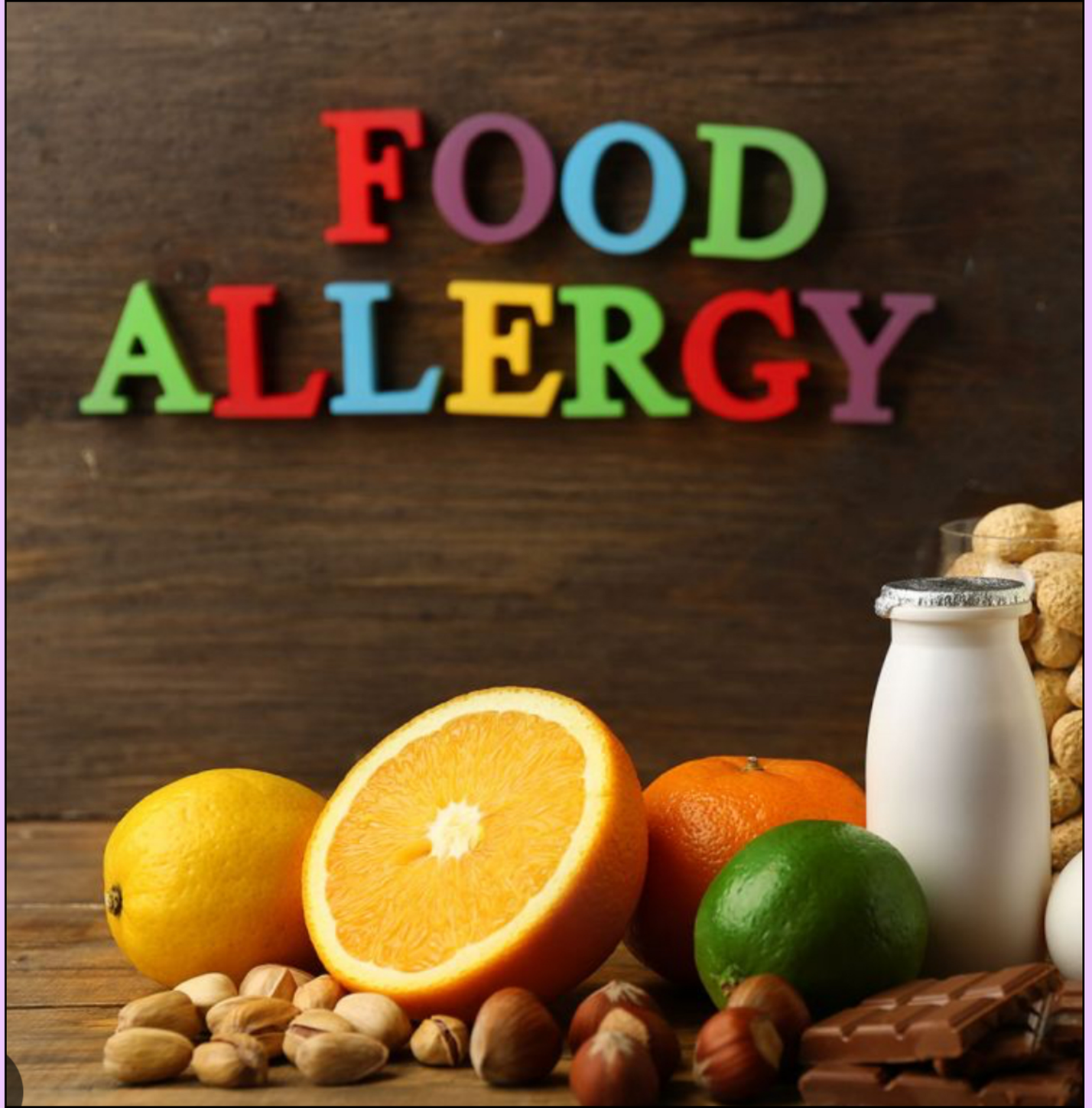
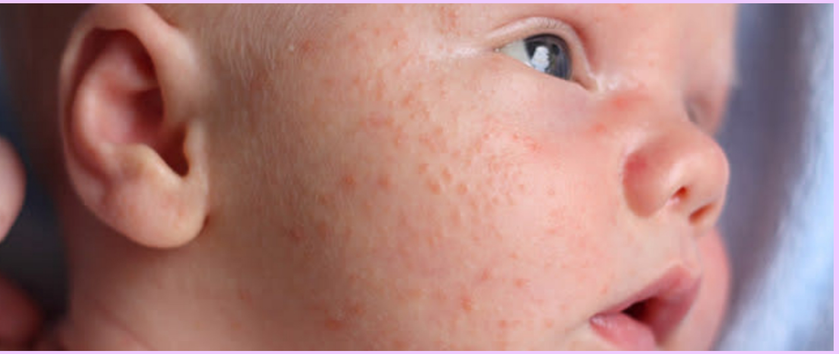
DR.N.ANSARI

M.D


FOOD
ALLERGY

REFERENCES

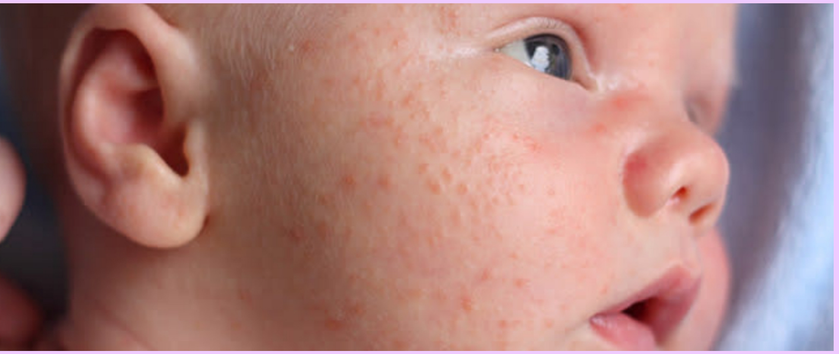
- Uptodate
- Nelson Textbook of Pediatrics
- Pediatric Gastrointestinal and Liver Disease sixth Edition
- Walker’s Pediatric Gastrointestinal Disease Sixth Edition

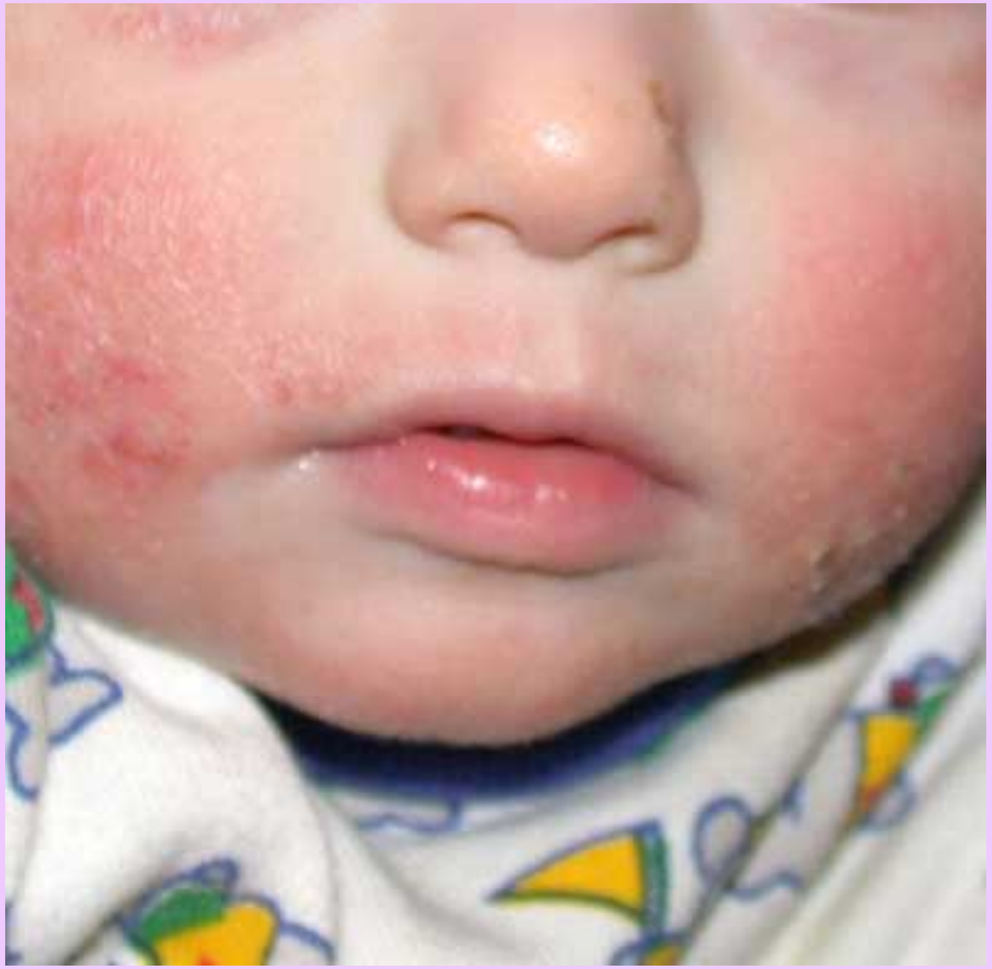


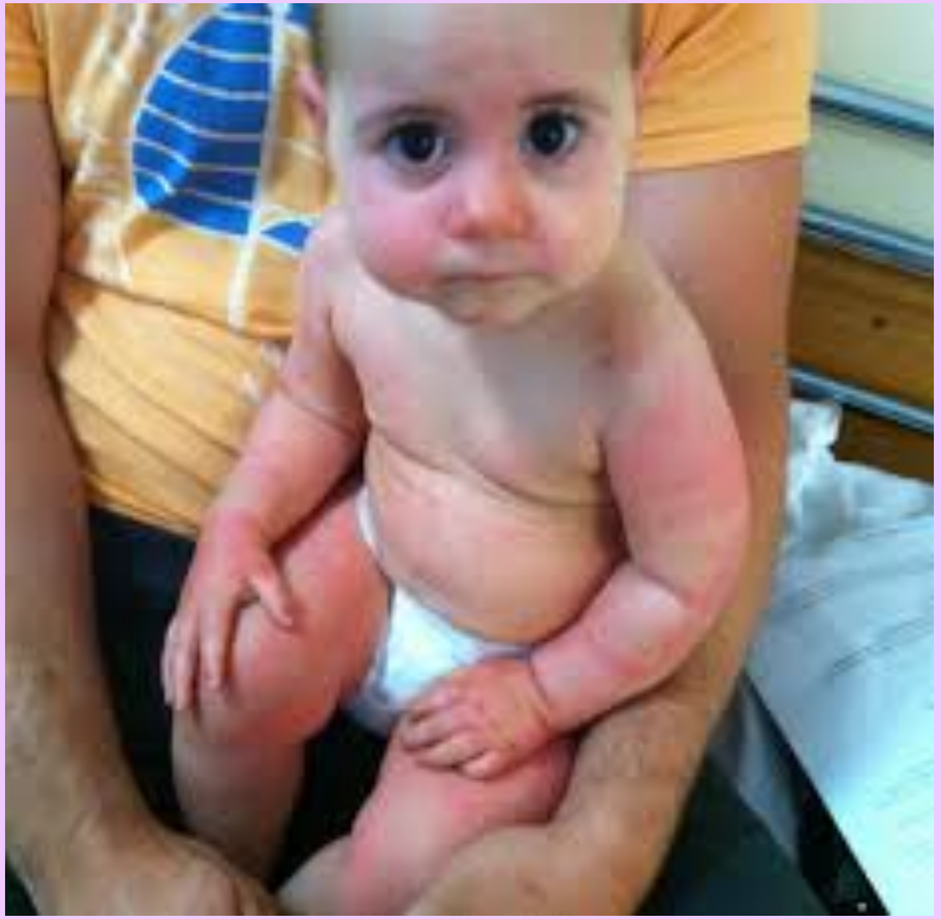
ADVERSE FOOD REACTIONS

Food Intolerance (advese physiologic responses)

Food Allergy (immunologic responses)

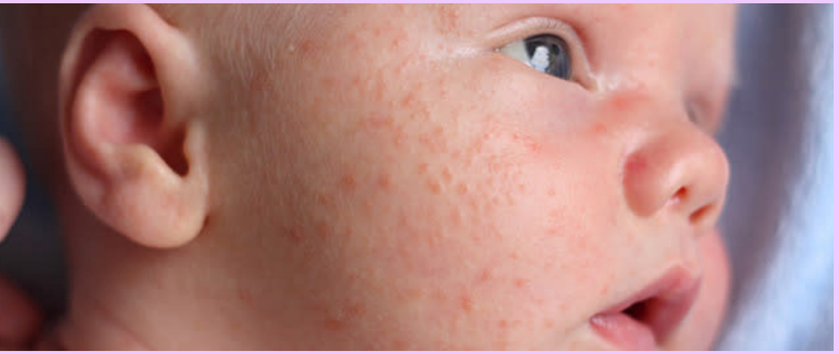






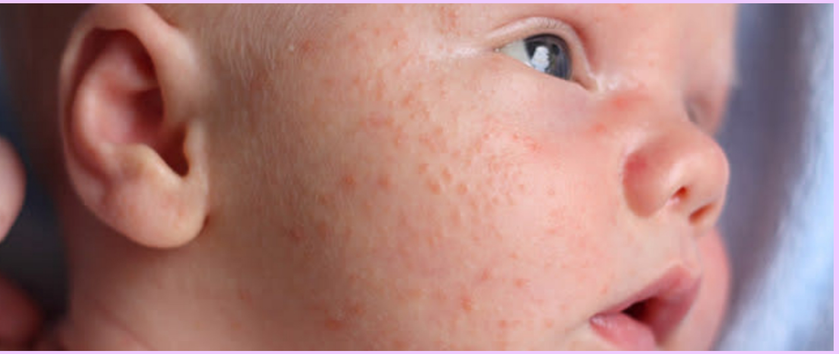
FOOD INTOLERANCES

Food intolerances are nonimmunologic physiologic responses and can include metabolic, toxic, pharmacologic, or other mechanism



FOOD ALLERGIES.

Food allergies are adverse immunologic responses and can be IgE mediated, non–IgE mediated, or mixed



PREVALENCE

- 5 to 10 percent of young children
- peak prevalence at approximately one year of age
- then falls progressively until late childhood, and remains stable at approximately 3 to 4 percent
- Some studies have suggested that the prevalence of food allergy has increased over time (Western lifestyle)

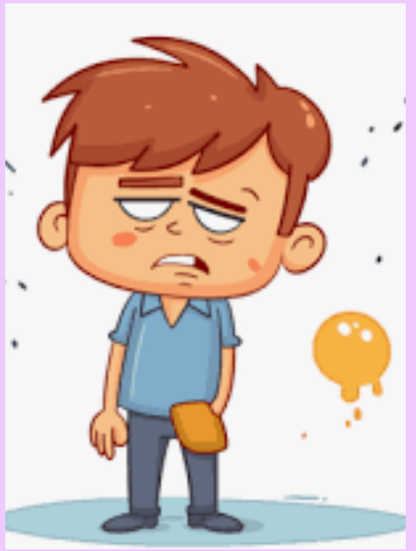
PREVALENCE

Food allergies appear to have increased over the past 3 decades, primarily in westernized/ industrialized countries. Worldwide, estimates of food allergy prevalence range from 1–11% with regional variations.

The vast majority of food allergies are due topeanut, tree nuts, seeds, milk, egg, soy, wheat, fish, and shellfish, with regional variations in prevalence.

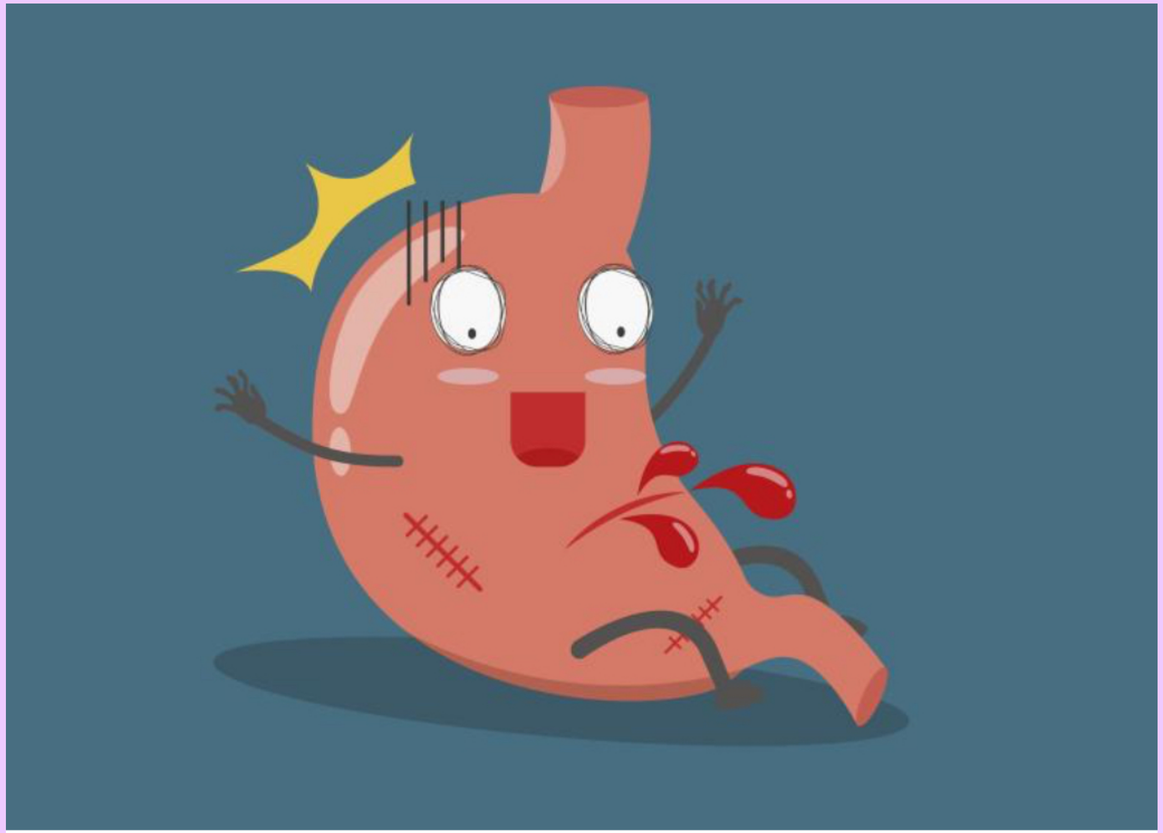
FOOD INTOLERANCE

FOOD INTOLERANCE (NON–IMMUNE SYSTEM MEDIATED, NONTOXIC, NONINFECTIOUS)
Host Factors
Enzyme deficiencies—lactase (primary or secondary), sucrase/isomaltase, hereditary fructose intolerance, galactosemia, alcohol dehydrogenase deficiency
Gastrointestinal disorders—inflammatory bowel disease, irritable bowel syndrome, pseudoobstruction, colic
Idiosyncratic reactions—caffeine in soft drinks (“hyperactivity”)
Psychologic—food phobias, obsessive/compulsive disorder
Migraines (rare)
Food Factors (Toxic or Infectious or Pharmacologic)
Infectious organisms— *Escherichia coli* , *Staphylococcus aureus* , *Clostridium perfringens* , *Shigella* , botulism, *Salmonella* , *Yersinia* , *Campylobacter*
Toxins—histamine (scombroid poisoning), saxitoxin (shellfish)
Pharmacologic agents—caffeine, theobromine (chocolate, tea), tryptamine (tomatoes), tyramine (cheese), benzoic acid in citrus fruits (perioral flare)
Contaminants—heavy metals, pesticides, antibiotics



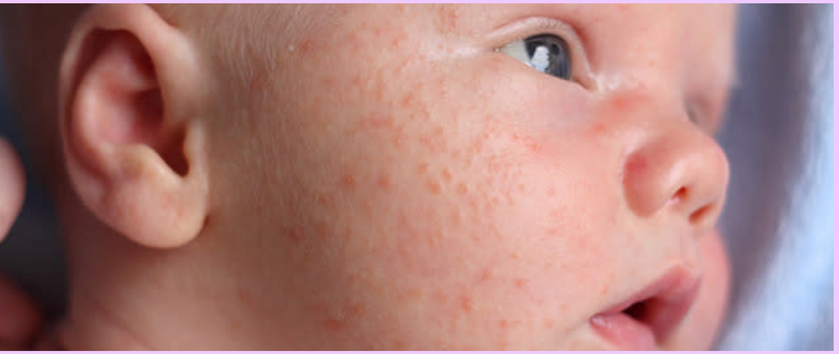
FOOD ALLERGY

- IgE Mediated
- Mixed IgE Mediated and Non-IgE Mediated
- Non-IgE Mediated



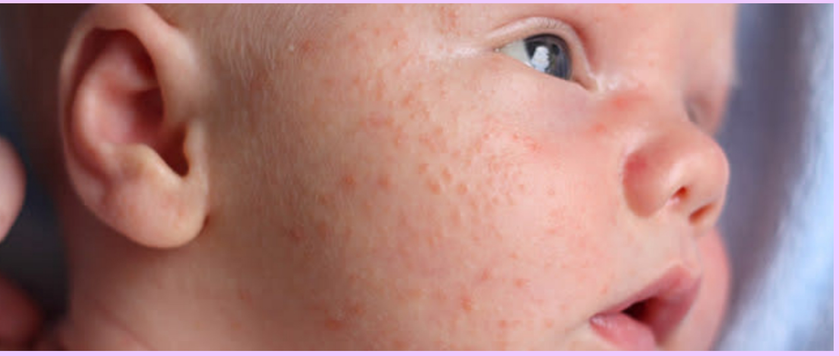
PATHOGENESIS

- Most common in young children and against cows milk and soy protein
- In older children against egg, fish, peanut and wheat
- Allergic reactions begin within minutes up to 2 hours
- Lasting 1-2 hours
- Those reactions causing upper airway obstructions or circulatory collapse are termed anaphylaxy



PATHOGENESIS

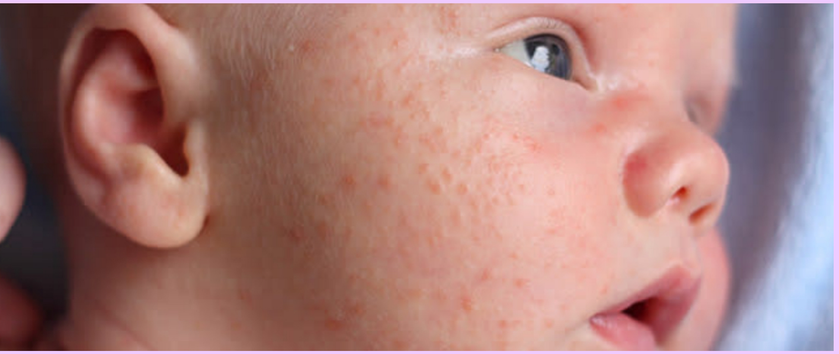
- **IgE Mediated:** In susceptible individuals - food allergens penetrate mucosal barriers-cell-bound IgE antibodies- release mediators -vasodilation, smooth muscle contraction, mucus secretion(allergy)-released cytokines attract and activate eosinophils and lymphocytes-leading to prolonged inflammation.



IgE Mediated
Cutaneous—urticaria, angioedema, morbilliform rashes, flushing, contact urticarial
Gastrointestinal—oral allergy syndrome, gastrointestinal anaphylaxis
Respiratory—acute rhinoconjunctivitis, bronchospasm
Generalized—anaphylactic shock, exercise-induced anaphylaxis

PATHOGENESIS

- Non-IgE food allergies: lymphocytes, primarily food allergen–specific T cells, secrete excessive amounts of various cytokines that lead to a “delayed,” more chronic inflammatory process
-
-

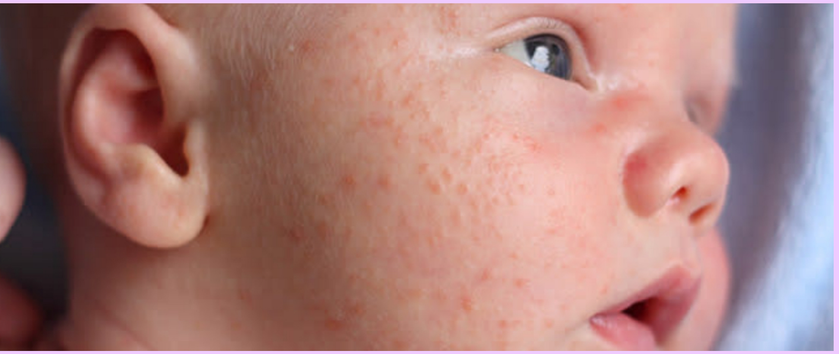


PATHOGENESIS

Non–IgE Mediated
Cutaneous—contact dermatitis, dermatitis herpetiformis (celiac disease)
Gastrointestinal—food protein–induced enterocolitis, proctocolitis, and enteropathy syndromes, celiac disease
Respiratory—food-induced pulmonary hemosiderosis (Heiner syndrome)
Unclassified

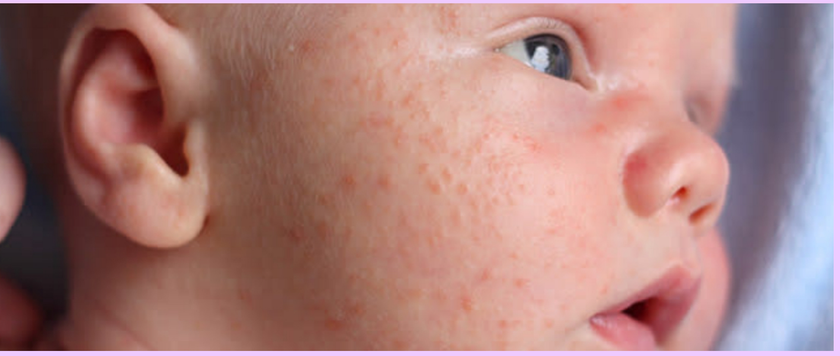
PATHOGENESIS

- Mixed IgE and cellular responses to food allergens can also lead
  to chronic disorders



PATHOGENESIS

Mixed IgE Mediated and Non–IgE Mediated
Cutaneous—atopic dermatitis, contact dermatitis
Gastrointestinal—allergic eosinophilic esophagitis and gastroenteritis
Respiratory—asthma

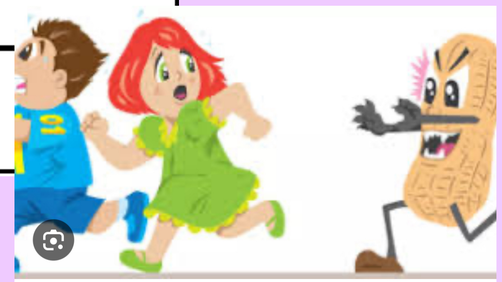


FOOD ALLERGENS

- 90% are: *milk*, *egg*, *peanuts*, *tree nuts*, *fish*, *soy*, and *wheat*

NATURAL HISTORY OF FOOD ALLERGY

FOOD	USUAL AGE AT ONSET OF ALLERGY	CROSS REACTIVITY	USUAL AGE AT RESOLUTION
Hen's egg white	0-1 yr	Other avian eggs	7 yr (75% of cases resolve)*
Cow's milk	0-1 yr	Goat's milk, sheep's milk, buffalo milk	5 yr (76% of cases resolve)*
Peanuts	1-2 yr	Other legumes, peas, lentils; coreactivity with tree nuts	Persistent (20% of cases resolve)
Tree nuts	1-2 yr; in adults, onset occurs after cross reactivity to birch pollen	Other tree nuts; co-reactivity with peanuts	Persistent (9% of cases resolve)
Fish	Late childhood and adulthood	Other fish (low cross-reactivity with tuna and swordfish)	Persistent †
Shellfish	Adulthood (in 60% of patients with this allergy)	Other shellfish	Persistent
Wheat*	6-24 mo	Other grains containing gluten (rye, barley)	5 yr (80% of cases resolve)
Soybeans*	6-24 mo	Other legumes	2 yr (67% of cases resolve)
Kiwi	Any age	Banana, avocado, latex	Unknown
Apples, carrots, and peaches §	Late childhood and adulthood	Birch pollen, other fruits, nuts	Unknown



PREVENTION OF FOOD ALLERGY

RECOMMENDED
Infant-safe forms of peanut, egg introduced around age 6 mo, not before 4 mo
Other allergens may be introduced around this time as well
Allergy testing before introduction not usually needed (see text)
Infants with severe eczema or egg allergy may benefit from evaluation for early peanut introduction at 4-6 mo
Diverse infant diet
UNPROVEN/NOT RECOMMENDED
Hydrolyzed formulas
Maternal allergen avoidance during pregnancy or lactation
Purposeful delay in introducing allergens to infants


Symptoms

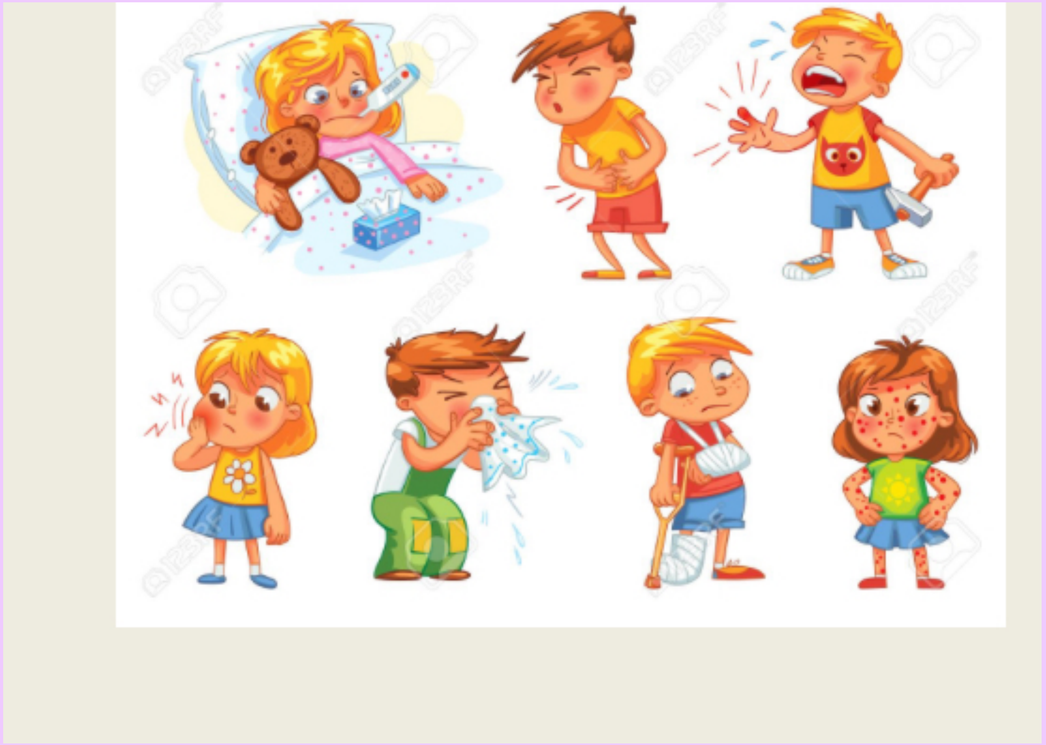

ALLERGY

CLINICAL MANIFESTATIONS

TARGET ORGAN	IMMEDIATE SYMPTOMS	DELAYED SYMPTOMS
Cutaneous	Erythema Pruritus Urticaria Morbilliform eruption Angioedema	Erythema Flushing Pruritus Morbilliform eruption Angioedema Eczematous rash
Ocular	Pruritus Conjunctival erythema Tearing Periorbital edema	Pruritus Conjunctival erythema Tearing Periorbital edema
Upper respiratory	Nasal congestion Pruritus Rhinorrhea Sneezing Laryngeal edema Hoarseness Dry staccato cough	
Lower respiratory	Cough Chest tightness Dyspnea Wheezing Intercostal retractions Accessory muscle use	CoughDyspneaWheezing
Gastrointestinal (oral)	Angioedema of the lips, tongue, or palate Oral pruritus Tongue swelling	
Gastrointestinal (lower)	Nausea Colicky abdominal pain Reflux Vomiting Diarrhea	Nausea Abdominal pain Reflux Vomiting Diarrhea Hematochezia Irritability and food refusal with weight loss (young children)
Cardiovascular	Tachycardia (occasionally bradycardia in anaphylaxis) Hypotension Dizziness Fainting Loss of consciousness	
Other	Uterine contractions Sense of “impending doom”	

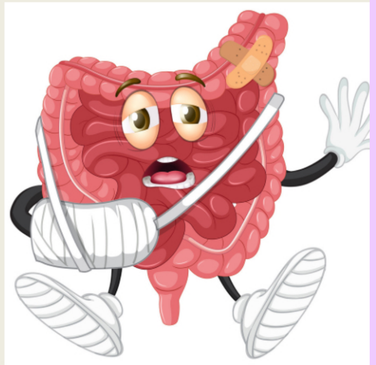
CLINICAL MANIFESTATIONS

GI food allergies are often the first form of allergy to affect infants and young children, and typically manifest as chronic irritability, vomiting or “spitting-up,” diarrhea, and poor weight gain. Cell-mediated hypersensitivities without IgE involvement (non-IgE) predominate, making standard allergy tests such as skin-prick tests and in vitro tests for food-specific IgE antibodies of little diagnostic value



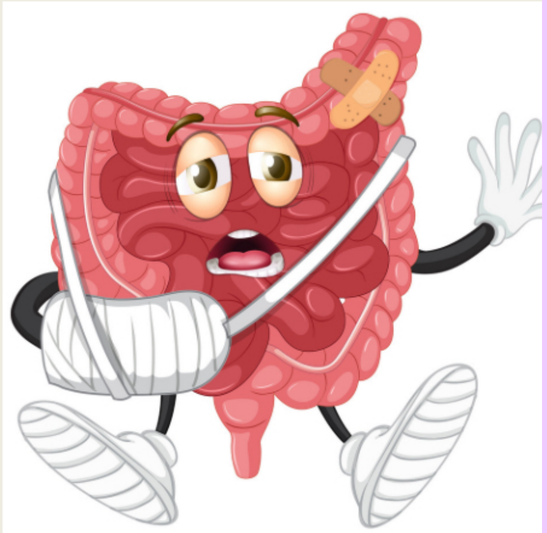
GASTROINTESTINAL MANIFESTATIONS

Pollen-food allergy syndrome (oral allergy syndrome) is an IgE-mediated hypersensitivity to certain uncooked or unprocessed plant-based foods that occurs in many older children who have pollen-induced allergic rhinitis. Symptoms are usually confined to the oropharynx and consist of the rapid onset of oral pruritus; tingling and angioedema of the lips, tongue, palate, and throat; and occasionally a sensation of pruritus in the ears and tightness in the throat. Symptoms are generally short-lived and are caused by local mast cell activation following contact with fresh raw fruit and vegetable proteins that cross react with birch tree pollen (including but not limited to apple, carrot, potato, celery, hazelnuts, peanuts, kiwi, cherry, pear), grass pollen (potato, tomato, watermelon, kiwi), mugwort weed pollen (celery, fennel, mustard, peach), and ragweed pollen (banana, melons such as watermelon and cantaloupe).



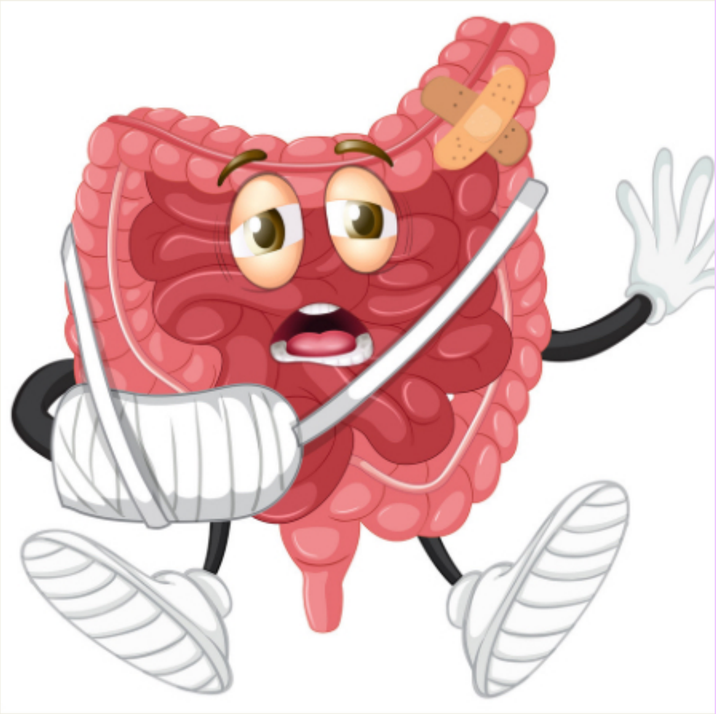
GASTROINTESTINAL MANIFESTATIONS

GI food allergies are often the first form of allergy to affect infants and young children and typically manifest as irritability, vomiting or “spitting-up,” diarrhea, and poor weight gain. Cell-mediated hypersensitivities without IgE involvement predominate, making standard allergy tests such as skin-prick tests and in vitro tests for food-specific IgE antibodies of little diagnostic value. The non–IgE-mediated GI food-allergic disorders food protein–induced enterocolitis syndrome (FPIES), food protein–induced allergic proctocolitis (FPIAP), and eosinophilic GI disorders



GASTROINTESTINAL MANIFESTATIONS

Acute GI allergy generally manifests as acute abdominal pain, vomiting, or diarrhea that accompanies IgE-mediated allergic symptoms in other target organs.



FOOD PROTEIN–INDUCED GASTROINTESTINAL SYNDROMES

Food Protein–Induced Gastrointestinal Syndromes

	FPIES	FPIAP	FPE	EOSINOPHILIC GASTROENTEROPATHIES
Age at onset	1 day to 1 year, later for fish and shellfish	1 day to 6 months	Dependent on age of exposure to antigen, cow's milk and soy up to 2 yr	Infant to adolescent
FOOD PROTEINS IMPLICATED				
Most common	Cow's milk, egg, oat, rice	Cow's milk, soy	Cow's milk, soy	Cow's milk, wheat, egg white, soy, peanut, seafood
Less common	Soy, chicken, turkey, fish, pea, peanut, avocado, sweet potato	Egg	Wheat, egg	Meats, corn, rice, fruits, vegetables, legumes
Multiple food hypersensitivities	>50% both cow's milk and soy if younger than 6 mo; 40–50% react to more than one grain, 30% react to more than one fish	40% both cow's milk and soy	Rare	Common
Feeding at the time of onset	Formula	>50% exclusive breastfeeding	Formula	Formula
ATOPIC BACKGROUND				
Family history of atopy	40–70%	25%	Unknown	~50% (often history of EoE)
Personal history of atopy	30%	22%	22%	~50%
SYMPTOMS				
Emesis	Projectile, repetitive, severe	No	Intermittent	Intermittent
Diarrhea	Severe in chronic FPIES	No	Moderate	Moderate
Bloody stools	Occasionally severe	Moderate	Rare	Moderate
Edema	Acute, severe	No	Moderate	Moderate
Shock	15%	No	No	No
Failure to thrive	Moderate	No	Moderate	Moderate
Differential diagnosis	Infection: viral, bacterial Necrotizing enterocolitis, GI obstruction (ileus, pyloric stenosis, Meckel diverticulum); gastroesophageal reflux disease; very early onset inflammatory bowel disease, seizure disorder, metabolic disorder, cardiac disease, anaphylaxis	Rectal fissure, bleeding disorder, vit K deficiency, GI infection e.g., *Shigella,* inflammatory bowel disease	Celiac disease, primary immunodeficiency, inflammatory bowel disease	Gastroesophageal reflux disease, recurrent vomiting due to other causes, parasitic and fungal infections, congenital rings, Crohn disease, periarteritis, allergic vasculitis, connective tissue diseases, bullous pemphigoid, pemphigoid vegetans, graft-versus-host disease, achalasia, drug hypersensitivity, celiac disease, vasculitis, carcinoma, hypereosinophilic syndrome
LABORATORY FINDINGS				
Anemia	Moderate	Mild	Moderate	Mild-moderate
Hypoalbuminemia	Acute	Rare	Moderate	Mild-severe
Methemoglobinemia	May be present	No	No	No
ALLERGY EVALUATION				
Food skin-prick test	Majority negative †	Negative	Negative	Positive in ~50%
Serum food allergen IgE	Majority negative †	Negative	Negative	Positive in ~50%
Total IgE	Normal	Negative	Normal	Normal to elevated
Peripheral blood eosinophilia	No	Occasional	No	Present in <50%
BIOPSY FINDINGS				
Colitis	Prominent	Focal	No	May be present
Lymph nodular hyperplasia	No	Common	No	Yes
Eosinophils	Prominent	Prominent	Few	Prominent; also neutrophilic infiltrates, papillary elongation, and basal zone hyperplasia
Food challenge	Emesis in 1-4 hr; diarrhea in 5-8 hr (in a subset)	Rectal bleeding in 6-72 hr	Vomiting, diarrhea, or both in 40-72 hr	Vomiting and diarrhea in hours to days
Treatment	Protein elimination, 80% respond to casein hydrolysate and symptoms clear in 3-10 days; rechallenge under supervision in 0.5-2 yr	Protein elimination, symptoms clear in 3 days with casein hydrolysate; resume/continue breastfeeding on maternal antigen-restricted diet; reintroduce at home after 9-12 mo of age	Protein elimination, symptoms clear in 1-3 wk; rechallenge and biopsy in 1-2 yr	Protein elimination, good response to casein hydrolysate, excellent (>90%) response to elemental diet; symptoms clear in 2-3 wk, excellent acute response to oral steroids but with high rate of relapse following discontinuation; in EoE 30–50% response to proton pump inhibitors, 70% to swallowed corticosteroids; rechallenge by introducing food at home and biopsy in 1-2 yr
Natural history	Cow's milk: 60% resolved by 2 yr Soy: 25% resolved by 2 yr	Resolved by 9-12 mo	Most cases resolve in 2-3 yr	Typically a prolonged, relapsing course
Reintroduction of the food	Supervised food challenge	At home, gradually advancing from 1 oz to full feedings over 2 wk	Home, gradually advancing	Home, gradually advancing

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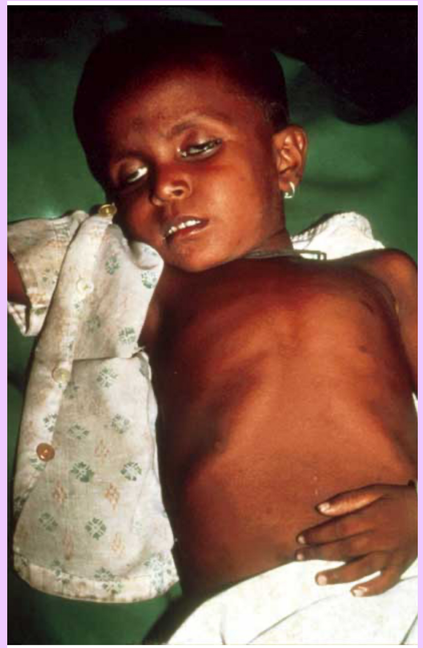
FPIES

FOOD PROTEIN–INDUCED ENTEROCOLITIS SYNDROME

- Food triggers : Globally, cow’s milk is the most common trigger of FPIES, although in countries with higher rates of breastfeeding rather than formula feeding, complementary foods introduced into infants’ diets early are also reported. Commonly reported triggers include soy, oat, rice, vegetables (avocado, sweet potato), fruits (banana), egg, fish, chicken, turkey, peanut, tree nuts, and fish. Most infants (50–75%) react to one food; however, about 10–15% report more than three food triggers.
- Pathophysiology: FPIES is characterized by a strong inflammatory response with significant elevation of CRP, neutrophils, and platelets.



FPIES

FOOD PROTEIN–INDUCED ENTEROCOLITIS SYNDROME

- Clinical manifestations: FPIES typically manifests in the first year of life in an acute form as projectile, repetitive vomiting within 1-4 hours of food ingestion, frequently accompanied by lethargy, pallor (or dusky appearance), and low muscle tone; in a smaller subset, vomiting is followed by watery diarrhea in 5-10 hours . Prolonged ingestion of the causal allergen may result in abdominal distention, bloody diarrhea, anemia, and failure to thrive, referred to as chronic FPIES. Acute FPIES is considered to be an allergic emergency because hypotension occurs in approximately 5–10% of patients after allergen ingestion, which initially may be attributed to sepsis.
- Diagnosis: Acute FPIES is diagnosed based on the recognition of a constellation of symptoms,and allergy tests detecting food-specific IgE are typically negative. OFCs are rarely required for the confirmation of the initial diagnosis, but are utilized for evaluating resolution of FPIES. Chronic FPIES is diagnosed based on the chronic GI symptoms that resolve within days to weeks following elimination of the allergen and recur acutely within 1-2 hours following a subsequent feeding.

FPIES

FOOD PROTEIN–INDUCED ENTEROCOLITIS SYNDROME

Emergency management: Severe FPIES reactions are considered allergic emergencies due to the risk of hypotension (in extreme cases, hypovolemic shock), dehydration, and metabolic derangements including acidemia and methemoglobinemia . Acute management entails vigorous intravenous hydration. Additional therapies include intravenous or intramuscular ondansetron as an antiemetic, and a single dose of steroid (e.g., methylprednisolone) may be administered due to a strong inflammatory response.

Mild to moderate reactions can be managed with oral rehydration and oral ondansetron.

Epinephrine autoinjectors and oral antihistamines are not prescribed for home management; however, vasopressors may be used for treatment of shock in the medical setting.

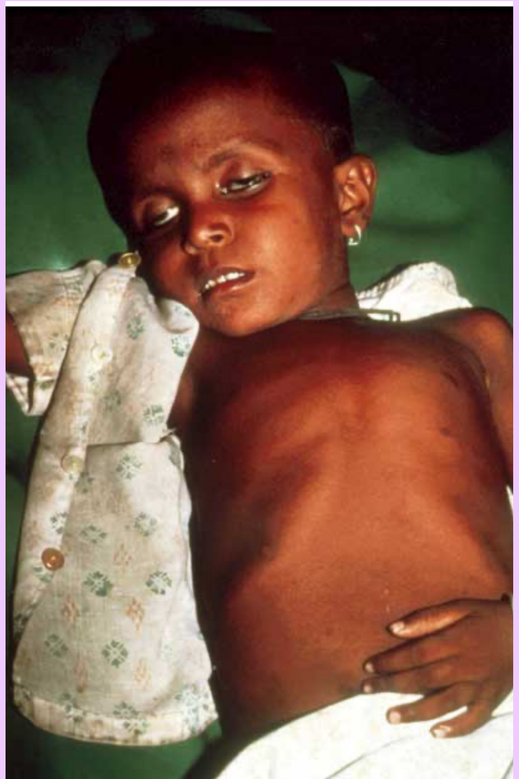
FPIES

FOOD PROTEIN–INDUCED ENTEROCOLITIS SYNDROME

Dietary management: Breastfeeding mothers rarely need to restrict the foods that trigger symptoms in an infant following direct feeding, unless the infant exhibits symptoms of acute or chronic FPIES during breast milk feeding or has impaired growth.

Hypoallergenic infant formulas (extensively hydrolyzed or amino acid) are recommended in non-breastfed infants to avoid cow's milk and soy. Timely introduction of solids is important for nutrition and for the development of oromotor skills.

Following acute FPIES reactions to a solid food, foods from an unrelated food group can be chosen for introduction. Tolerance to one food from a food group usually indicates a favorable likelihood of tolerance to the related food discusses practical guidelines for dietary management of FPIES.

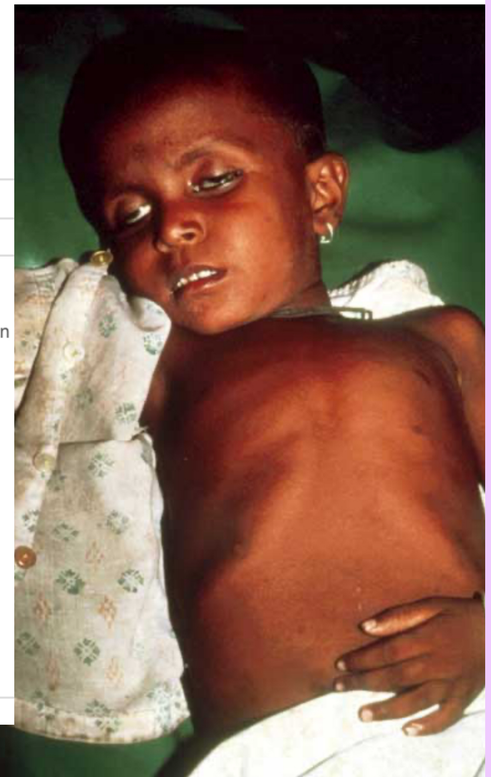


FPIES

FOOD PROTEIN–INDUCED ENTEROCOLITIS SYNDROME

ACUTE FPIES *	
MAJOR CRITERIA (BOTH MUST BE MET), PLUS	MINOR CRITERIA (≥3 OCCURRING WITH EPISODE)
Vomiting 1-4 hr after suspect food ingestion	≥2 episodes with same food
Absence of immediate, IgE-mediated allergic symptoms (hives, itching, swelling, wheezing, cough)	One episode with a different food
	Lethargy
	Pallor
	Need for ER visit
	Need for IV fluid support
	Diarrhea within 24 hr (usually 5-10 hr)
	Hypotension
	Hypothermia

CHRONIC FPIES †	
SYMPTOMS AND SEVERITY	CRITERIA
Milder (lower doses with intermittent ingestion):	Resolution of symptoms within days to weeks after elimination of offending food(s)
Intermittent vomiting and/or diarrhea	Acute recurrence of symptoms (vomiting in 1-4 hr, diarrhea in <24 hr, usually 5-10 hr) when the food is reintroduced, following a period of elimination
Growth faltering	Confirmatory OFC required for conclusive diagnosis; if OFC not performed diagnosis remains presumptive
No dehydration or metabolic acidosis	
Severe (higher doses with chronic ingestion):	
Intermittent but progressive vomiting and watery diarrhea (occasionally with blood)	
Poor weight gain or failure to thrive	
Possible dehydration and metabolic acidosis, anemia, hypoproteinemia, neutrophilia, thrombocytosis	

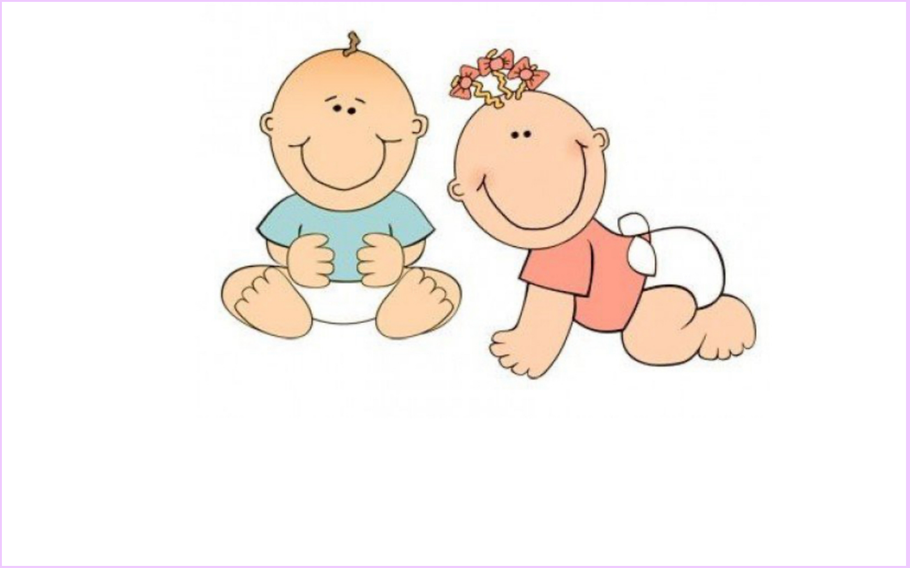


FPIAP

FOOD PROTEIN–INDUCED ALLERGIC PROCTOCOLITIS

presents in the first few months of life as blood-streaked stools in otherwise healthy infants that are breastfed and/or formula-fed . Blood loss is typically mild, but can occasionally result in anemia. The most commonly implicated dietary triggers are cow’s milk and soy proteins, followed by egg; their elimination, either by maternal dietary restriction if breastfeeding or by use of hypoallergenic formulas, leads to symptom and gross blood resolution within 48-72 hours in most infants. FPIAP is diagnosed clinically based on the presence of blood in the stool.

in cases of mild to moderate FPIAP, many authorities recommend a trial of the culprit food 2-3 months following symptom resolution to determine whether the infant has “outgrown” the sensitivity.



FPE

FOOD PROTEIN–INDUCED ENTEROPATHY (FPE)

often manifests in the first several months of life as diarrhea, often with steatorrhea and poor weight gain . Symptoms include protracted diarrhea, vomiting in up to 65% of cases, failure to thrive, abdominal distention, early satiety, and malabsorption. Anemia, edema, and hypoproteinemia occur occasionally. Cow’s milk sensitivity is the most common cause of FPE in young infants, but it has also been associated with sensitivity to soy, egg, wheat, rice, chicken, and fish in older children.

Celiac disease , the most severe form of FPE, occurs in about 1 per 100 of the U.S. population, although it may be “silent” in many patients . The classic form is characterized by extensive loss of absorptive villi and hyperplasia of the crypts, leading to malabsorption, chronic diarrhea, steatorrhea, abdominal distention, flatulence, and weight loss or failure to thrive.

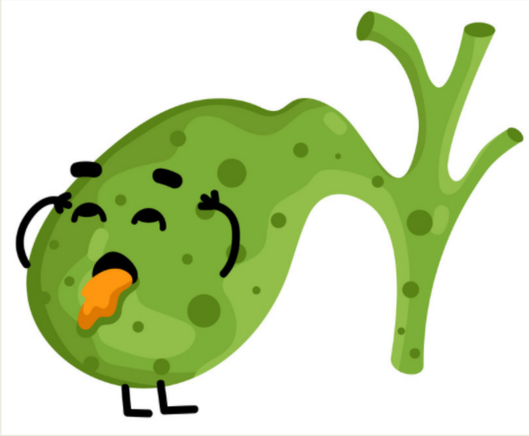
FPE

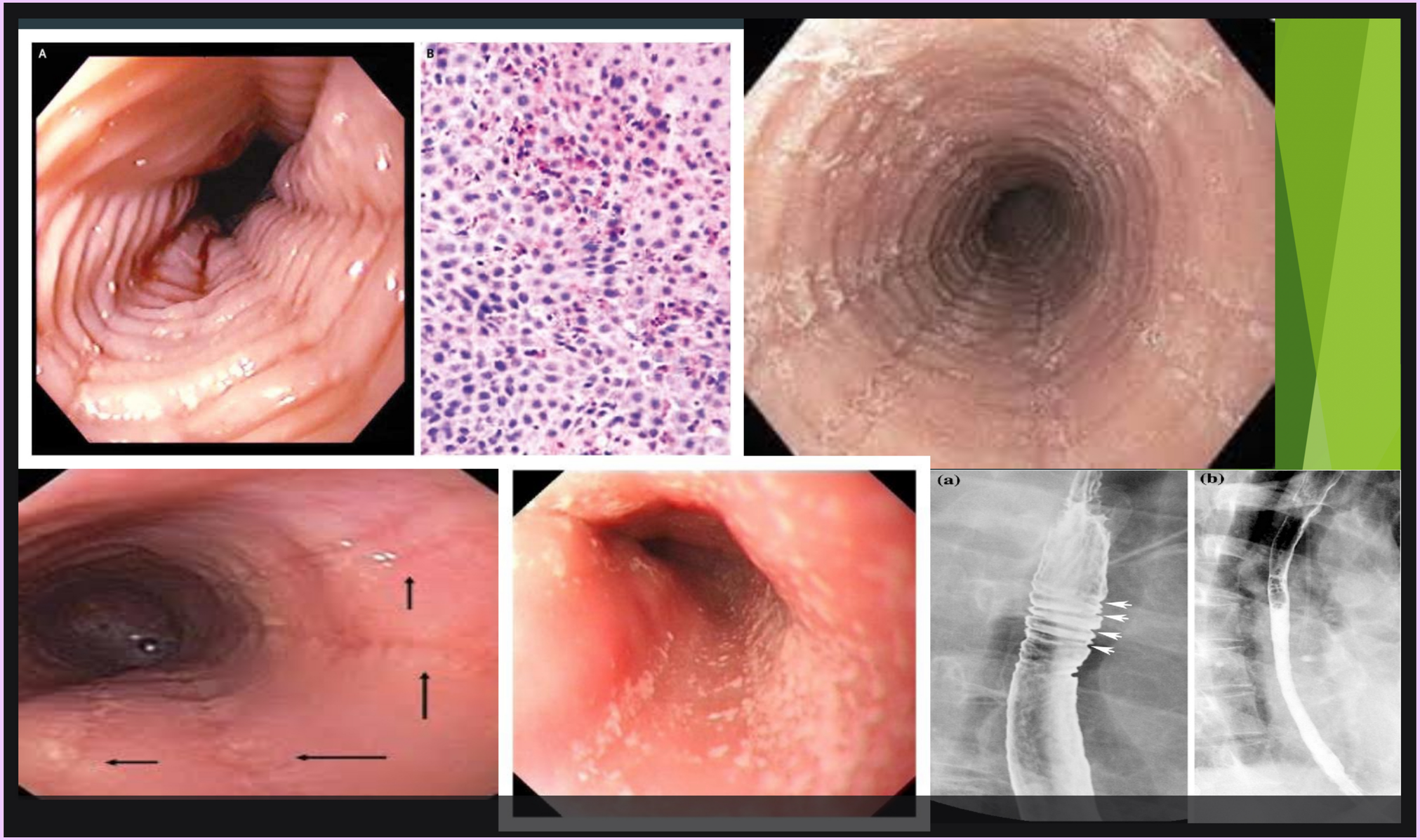
The classic form is characterized by extensive loss of absorptive villi and hyperplasia of the crypts, leading to malabsorption, chronic diarrhea, steatorrhea, abdominal distention, flatulence, and weight loss or failure to thrive. Oral ulcers and other extraintestinal symptoms secondary to malabsorption may occur.

EOE

EOSINOPHILIC ESOPHAGITIS.

EoE may present from infancy through adolescence, more frequently in boys . EoE is a cell-mediated disorder, which is often associated with IgE-mediated food allergies in infants and young children, and manifests as chronic GER, intermittent emesis, food refusal, abdominal pain, dysphagia, food impaction, irritability, sleep disturbance, and failure to respond to conventional GER medications. EoE is a clinicopathologic diagnosis. The diagnosis is confirmed when 15 eosinophils per high-power field are seen on esophageal biopsies. Treatment is possible with elimination of dietary allergens but management with medications is typically included . *Eosinophilic gastritis and gastroenteritis are additional eosinophilic GI disorders that are far less common and can occur at any age*

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Eosinophilic gastritis often presents with nausea and abdominal pain or bloating, while eosinophilic enteritis may also present with nausea, abdominal pain or bloating with additional diarrhea, anemia, or protein loss. Eosinophilic colitis may present with loose stool or blood in stool associated with abdominal cramping/pain.

Generalized edema secondary to hypoalbuminemia may occur in some infants with marked protein-losing enteropathy

DIAGNOSIS

A thorough medical history is necessary to determine whether a patient’s symptomatology represents an adverse food reaction , whether it is an intolerance or food allergic reaction, and, if the latter, whether it is likely to be an IgE-mediated or a cell-mediated response

TREATMENT

Appropriate identification and elimination of foods responsible for food hypersensitivity reactions are the most established and validated management strategies for food allergies. Complete elimination of common foods (milk, egg, soy, wheat, rice, chicken, fish, peanut, nuts) is very difficult because of their widespread use in a variety of processed foods.

Egg allergy is not a contraindication for vaccination with measles, mumps, rubella, or influenza vaccines, but remains a concern for the yellow fever vaccine where referral to an allergist is recommended.

TREATMENT

Children at risk of food-induced anaphylaxis should be given self-injectable epinephrine and a written emergency plan in case of accidental ingestion)

An FDA-approved peanut oral immunotherapy (OIT) agent is commercially available for use in children. Combining OIT with anti-IgE treatment (omalizumab) or other biologic agents is under study and may improve safety or efficacy compared to OIT alone. Furthermore, extensively heated milk or egg in baked products are tolerated by the majority of milk and egg–allergic children.

Regular ingestion of baked products with milk and egg may accelerate resolution of milk and egg allergy.

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M.D