Food Protein Induced Enterocolitis Syndrome to Quinoa

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Abstract

Food protein induced enterocolitis is a non-IgE, cell mediated food allergy. It is not a well-known condition, but it can be severe in some children. The predominant feature is vomit 1 to 4 hours after consuming the offending food. There is limited evidence of allergic reactions to quinoa and generally it has been considered to be safe for children diagnosed with food induced enterocolitis (FPIES) to other foods. There is not enough data on FPIES to solid foods since these are rarely considered as potential triggers for this entity. After a review of the literature we found that there are no reported cases of FPIES to quinoa. We report a case of a toddler that was admitted to the emergency department with unenforceable vomiting and diarrhea with dehydration signs and increased abdominal sounds. The laboratory results showed metabolic acidosis, and the infectious parameters were negative. To our knowledge this is the first reported case of FPIES to quinoa.

Keywords: Diet therapy; Enterocolitis; Food hypersensitivity; FPIES

Introduction

Quinoa is a seed native of the Andean highlands, frequently used as part of complementary feeding in infants and toddlers for its exceptional nutritional value. There is limited evidence of allergic reactions to quinoa and in general it has been considered to be safe for infants and toddlers, especially those diagnosed with food induced enterocolitis (FPIES) to other foods [1]. Food protein induced enterocolitis is a non-IgE, cell mediated food allergy. Usually presenting in early life, the incidence of FPIES is estimated to be between 1.5 and 30 per 10,000 infants [9]. It is not a well-known condition, but it can be severe in some children [2].

Initially FPIES was described as a food hyper sensitivity mainly provoked by cow’s milk or soy, and rarely solid foods were considered as the cause. With further investigation some solid foods have been introduced as part of the evaluation of FPIES, between the foods that can potentially cause this condition are cereals, vegetables, and poultry meats. Other studies have added grains and eggs to the list [3, 4]. It should be taken into account that infants with FPIES are at risk for multiple dietary protein hypersensitivities during an apparent period of immunologic susceptibility [3].

Clinical manifestations depend on the frequency and dose of the allergenic food. The age of onset is early when it presents in children younger than 9 months of age and late when it occurs after this age. It is considered acute when it occurs with intermittent food exposures. Major criterions include vomiting usually onset between 1-4 hours after food...
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ingestion and absence of classic IgE-mediated allergic skin or respiratory symptoms. Minor criteria includes; lethargy, pallor, a second (or more) episode of repetitive vomiting after eating the same suspect food, repetitive vomiting episode 1-4 hours after eating a different food, need for emergency room visit with any suspected reaction, need for intravenous fluid support, diarrhea that can occur usually 5-10 hours later (up to 24 hours), hypotension, and hypothermia. The diagnosis requires that the patient meet the major criterion and 3 or more minor criteria. These children have normal weight gain and the symptoms resolve within 24 hours after eliminating the offending food from the diet [2]. Elevation of white blood cells count with neutrophil predominance can also be found in these patients [5].

Chronic FPIES occurs with daily ingestion of the food [2]. For chronic FPIES there are general criteria but due to lack of published reports of chronic FPIES, major and minor criteria have not been established [6]. Children usually present intermittent emesis, chronic diarrhea, poor weight gain or even failure to thrive [2]. Severe chronic FPIES may end up causing dehydration and hypovolemic shock.

The gold standard for the diagnosis is a physician-supervised oral food challenge, this is strongly suggested if only a single episode has occurred, but most patients do not need to undergo confirmatory oral food challenge when they have had a history of severe or repeated reactions to the same triggering food and their symptoms resolve after elimination of the offending food from the diet [6].

Management is mainly supportive therapy with intravenous fluids and elimination diet [2].

We report a case of a 1 year 10 months old boy that was previously diagnosed with cow’s milk protein allergy and presented acute FPIES after the ingestion of quinoa. It is the first report of FPIES to quinoa in children.

Case Report

A 1 year 10 months old boy presented to the emergency department with unenforceable vomiting that lead to dehydration, pallor, diaphoresis, hypotonia and hours later, diarrhea. Physical examination showed dehydration signs and increased abdominal sounds, patient was afebrile. The laboratory results showed metabolic acidosis, infectious parameters were negative. Serum IgE was normal (8.86mg/dl). We performed other laboratory tests to rule out other possible etiologies that could explain the clinical manifestations but they were negative. In the emergency department, intravenous fluid therapy was initiated, with 2 bolus of normal saline solution 20cc/kg and afterward maintenance fluid therapy. The symptoms resolved within 24 hours. After the patient was stable the parents were given instructions about the elimination diet and red flags for possible FPIES to other foods. The patient had a history of cow’s milk protein allergy and allergy to extensively hydrolyzed formulas. Additionally, a family history of a sibling that had cow’s milk protein allergy and atopic dermatitis. The patient had normal weight gain and growth. After a detailed history it was noted that the vomiting onset was 3 hours after consuming quinoa. Furthermore, the patient had been previously admitted at 15 and 21 months of age to the hospital for episodes of unenforceable vomiting that lead to dehydration, and afterwards presented diarrhea. Both of the previous admissions were categorized as acute viral gastroenteritis with moderate dehydration but retrospectively parents associated these episodes to quinoa consumption as well. Due to the severity of the case and the presence of 2 other episodes that could be attributed to quinoa consumption we believed that oral food challenge was not necessary and FPIES to quinoa diagnosis was established.

Discussion

Quinoa has a significant nutritional value. It has a high concentration of protein, being considered one of the best vegetal protein sources and having protein levels similar to milk and higher than other cereals. It contains unsaturated fatty acids and all essential amino acids, it has vitamins, minerals and it is gluten-free [7]. A study by Ruales et al. also showed that consumption of quinoa in children between the ages of 50-65 months resulted in an increase of plasma insulin-like growth factor (IGF-1) levels, when compared to the control group [8]. All these characteristics make quinoa a good option for complementary feeding in infants and toddlers.

Quinoa allergenicity is low, after a review of the literature we found that there are no cases of FPIES to quinoa or allergy to quinoa reported in pediatric patients. Although we did find a case of a 52-year-old man who developed anaphylaxis to quinoa [9]. Also, another case of a 29-year-old female that presented a rash on the arm and chest, urticaria, itching on the palms and soles of feet and angioedema in the lips 5 minutes after ingesting quinoa salad, in this case skin prick test was positive for quinoa. This was the first reported case of quinoa allergy in the USA [10].

Food protein induced enterocolitis (FPIES) is not a well-known entity, even when it’s the most studied non-IgE food allergy [11]. It can be severe and lead to hypothermia, methemoglobinemia, acidosis and shock [2]. There are no sufficient epidemiological data and we can only estimate its prevalence based on a limited number of studies [11]. Thus, a large prospective birth cohort study from Israel conducted over 2 years reports a 0.34% prevalence of FPIES to cow’s milk [12]. Another more current prospective study from Australia showed an incidence of 15, 4/100,000 cases per year, this study included all different food triggers [13]. The pathophysiology is not well understood, although current
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knowledge suggests FPIES may result from defects in barrier and immunological function of the digestive tract. The ingestion of the offending food causes local inflammation that result in an increased intestinal permeability and fluid shift. T cells are thought to be involved in local inflammation but its role is yet to be confirmed. The inflammatory response is caused by cytokine release. Other factors to be considered are a deficit in TGF-β1 response and excessive TNF-α response. Cuabet et al. also consider an increase in specific IgA and decrease in specific IgG4 antibodies to be involved in the pathophysiology of FPIES [14].

There are major and minor clinical criteria in order to diagnose FPIES, but the main manifestation is recurrent and abundant vomiting that can be accompanied by diarrhea, and variable degree of dehydration that can even cause hypotensive shock [2]. Diagnosis of FPIES is based on clinical manifestations, response to elimination diets with resolution of symptoms, oral food challenges with the subsequent reappearance of symptoms after the ingestion of the offending food, endoscopy and biopsy findings and the exclusion of causes such as infections, inflammatory bowel disease, necrotizing enterocolitis, sepsis, severe lactose intolerance, neurologic disorders (cyclic vomiting) gastro esophageal reflux disease, gastrointestinal obstruction and metabolic disorders [15, 16].

Our patient presented an acute severe FPIES to quinoa. Generally, quinoa is considered a safe food for infants with other solid food FPIES [1]. The most common allergens in infants under 6 months of age are cow’s milk protein and soy, compared to FPIES to solid foods that occur between 6-12 months of age or later, the main triggers reported are rice, oats and, in some regions, grains. The allergenic food may vary according to genetical and environmental factors of each region [2].

The major clinical criterion that our patient presented was vomiting that started 1-4 hours after quinoa ingestion and 6 minor criteria (lethargy, pallor, previous events with same food, required emergency department visit, need for intravenous fluid support and diarrhea) [2].

Management in FPIES is mainly supportive therapy with intravenous fluids, after the patient has been stabilized, diet therapy should be started by avoidance of the food that caused the symptoms, nutritional counseling in order to avoid nutritional deficits, counseling for solid food introduction and reevaluations for resolution with oral food challenge [2,11]. In our patient intravenous fluid therapy and maintenance fluid therapy was given. Afterwards the parents were given instructions about food elimination diet and red flags for possible FPIES to other foods. Tolerance to quinoa should be attempted in this patient after 6-12 months of the last FPIES episode. But there are especial considerations; for example, it has been observed that older age at initial FPIES episode is associated with a longer period for tolerance acquisition. Our patient was diagnosed at 22 months so it would be recommended to wait 12 months after the last episode. Another recommendation would be to perform a skin prick test before the oral food challenge, but since quinoa is rarely allergenic the test does not count as a parameter. Severity has not been associated with a slower acquisition of tolerance, and the fact that our patient presents severe FPIES might not influence in the time for tolerance [4].

Quinoa is more frequently introduced as part of the complimentary feeding in Europe and North America, and possible food allergies should be noted. After reviewing the literature, we found that there is no evidence of previous reports of FPIES to quinoa. The lack of biomarkers that are specific for this pathology and a lack of published reports of FPIES in children may make the diagnosis challenging [11]. FPIES is commonly presented after milk and soy consumption, solid foods are rarely considered potential triggers of FPIES and generally there is a delay in the diagnosis of FPIES solid foods. Nowak et al. identified that patients with solid food FPIES were being diagnosed after a median of 2 reactions (range: 2-5) despite a high rate of severe reactions [3]. This was also the case in our patient which makes us think that the diagnosis in these cases is being frequently delayed. Patients presenting acute FPIES are commonly being misdiagnosed as acute viral gastroenteritis or sepsis, resulting in patients being hospitalized and going through extensive work-up and unnecessary treatments [16]. Therefore, it is important to consider FPIES as a possible diagnosis even when the suspected offending food is not a common trigger in other children; bearing in mind that even apparently safe foods such as quinoa can cause FPIES.

References


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