CASE REPORT

Association Between Headache and Sensitivities to Gluten and Dairy

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Abstract

Dietary factors are known triggers for migraine headaches. The most commonly implicated foods are wheat and dairy products. We present a case study of a patient with a 30-year history of debilitating migraine headaches who showed no benefit from various pharmaceutical interventions. Special panels for gluten and cross-reactive foods and a multiple autoimmune reactivity screen revealed significantly high levels of antibodies against wheat proteomes, transglutaminase, and dairy-related antigens. Not only did the implementation of a gluten-free and dairy-free diet result in an amelioration of the migraine headache symptomatology, the clinical improvements correlated with a significant decline in the levels of a majority of the previously elevated antibodies. This finding indicates that diet plays a significant role in a subgroup of patients with migraine headaches.

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Migraine headaches are a common neurological disorder that is induced by environmental triggers, including food. In fact, dietary factors that are known to activate the headache mechanism are called *migraine triggers*.[1] The foods most frequently implicated are wheat and dairy products.[2,3] In a multicenter, prospective study, increased prevalence of headache was noted in patients with celiac disease (CD) and inflammatory bowel disease.[4] Compared to 14% in controls, chronic headache was found in 30% of patients with CD; 56% of patients with nonceliac, gluten sensitivity; and 23% of patients with inflammatory bowel disease (IBD).[4] Similarly, cow's milk is one of the most commonly implicated foods, with sensitivity having been identified in 37% of migraine patients.[5,6] Furthermore, when either gluten or casein (a protein found in milk) was removed from the diet, a reduction in the number and severity of headaches to either a gluten-free or a casein-free diet was observed.[2,7]

This reported relationship between diet-triggered headaches or migraines and reactivity to wheat and gluten is difficult to prove since the reactivity is not consistently confirmed by the skin-prick test or IgE antibody titers. Therefore, an alternative to IgE testing, such as IgG and IgA antibody testing against food antigens, in combination with an elimination diet, would have clinical relevance.

For the present study, the authors will discuss the case of an individual with a 30-year history of debilitating headache that showed no benefit from various pharmaceutical interventions. Consequently, based on laboratory evaluations and the detection of IgG and IgA antibodies against various wheat and milk proteomes, a combination of a gluten- and dairy-free diet was implemented. The reduction in the number of and severity of the individual's headaches in response to this diet was demonstrated by a decline in the salient antibody levels.

Case Report

A 66-year-old Caucasian man with a 30-year history of debilitating headaches came to the Perlmutter Health Center for evaluation. Full neurological evaluations, including magnetic-resonance brain imaging, on multiple, prior occasions had revealed no abnormality. The patient had visited various headache-specialty clinics and had received no benefit from various prophylactic pharmaceutical interventions, including propranolol, amitryptiline, and topiramate. Medicative therapy was modestly effective and included oral sumatriptan—50 to 100 mg approximately twice weekly—as well as a hydrocodone/acetaminophen preparation—5/325 mg, 3 to 4 tablets approxi-

Table 1. IgG and IgA Antibodies Against Wheat/Gluten Proteomes Show Gluten Sensitivity

DETERMINATION	FIRST TEST Antibody/Index[a]	SECOND TEST Antibody/Index[b]	% Change[c]	REFERENCE RANGE Antibody/Index
WGA IgG	1.5	1.3	13.3%	0-1.5
WGA IgA	3.4	2.6	23.5%	0-1.9
α-Gliadin-33 IgG	1.4	1.3	7.1%	0-1.4
α-Gliadin-33 IgA	4.7	2.9	**38.3%**	0-1.8
α-Gliadin-17 IgG	1.5	1.2	20.0%	0-1.5
α-Gliadin-17 IgA	3.7	2.3	**37.8%**	0-2.0
γ-Gliadin IgG	4.5	3.1	**31.1%**	0-1.7
γ-Gliadin IgA	1.4	1.5	-7.1%	0-1.9
ω-Gliadin IgG	1.5	1.4	6.7%	0-1.6
ω-Gliadin IgA	3.3	2.6	21.2%	0-1.8
Glutenin IgG	1.5	1.3	13.3%	0-1.5
Glutenin IgA	4.0	2.7	**32.5%**	0-1.7
tTG-2 IgG	1.7	1.6	5.9%	0-1.4
tTG-2 IgA	2.0	1.9	5.0%	0-1.5
tTG-3 IgG	1.3	1.2	7.7%	0-1.6
tTG-3 IgA	1.1	1.0	9.1%	0-1.5
tTG-6 IgG	3.9	2.6	**33.3%**	0-1.5
tTG-6 IgA	3.5	2.0	**42.9%**	0-1.5

[a]Results for testing prior to any dietary intervention.
[b]Results for testing at 8 months after start of the gluten-free diet and 5 months after start of the combined dairy- and gluten-free diet.
[c]A percentage change of greater than 25% was considered significant.

Abbreviations: WGA, wheat germ agglutinin.

mately 6 days weekly. The patient described his headaches as pancranial and without antecedent aura. They typically lasted all day, and the patient rated them maximally at 8 to 9 on a 1 to 10 scale, with 10 being the worst. The patient had no associated nausea, but photophobia occasionally occurred in association with headache.

The patient's family history was remarkable in that his sister also suffered from intractable headaches, had significant food intolerances, and was suffering from a 20-year history of muscle stiffness, which was related to an antibody to gluten according to the patient, although she did not maintain a gluten-free diet.

The patient's own past medical history was unremarkable, with the exception of long-standing muscle stiffness. He had no ongoing symptoms or history of gastrointestinal distress. Physical and neurological examinations were normal.

Testing for special IgG and IgA antibodies against wheat/gluten proteomes and against gluten-associated and cross-reactive foods and a multiple, autoimmune reactivity screen were ordered through Cyrex Laboratories (Phoenix, Arizona, USA). In this testing, reference ranges were established based on the assessment of levels of these antibodies in 120 blood samples of healthy individuals age 18 to 70. Results summarized in Table 1 reveal that the patient had a significant elevation in antibodies for the following tests: (1) wheat germ agglutinin (WGA) IgA—3.4, (2) α-gliadin-33 IgA (alpha-gliadin-33)—4.7, (3) α-gliadin-17 IgA (alpha-gliadin-17)—3.7, (4) γ-gliadin IgG (gamma gliadin)—4.5, (5) ω-gliadin IgA (omega gliadin)—3.3, and (6) glutenin IgA. Antibody testing was also conducted against tTG-2, tTG-3, and tTG-6. While tTG-2 antibodies were slightly higher than normal ranges, both IgG and IgA against tTG-6 were significantly higher. In

Table 2. IgG And IgA Antibodies Against Gluten-associated and Cross-reactive Foods Show Dairy Sensitivity

DETERMINATION	FIRST TEST Antibody/Index[a]	SECOND TEST Antibody/Index[b]	% Change[c]	REFERENCE RANGE Antibody/Index
Cow's milk IgG + IgA	3.5	2.1	40.0%	0-2.0
α+β casein IgG + IgA	4.0	2.3	42.5%	0-2.3
Casomorphin IgG + IgA	2.7	1.6	40.7%	0-1.9
Milk butyrophilin IgG + IgA	1.9	1.1	42.1%	0-1.3
American cheese IgG + IgA	3.1	1.8	41.9%	0-2.2
Milk chocolate IgG + IgA	3.0	1.9	36.7%	0-2.4

[a]Results for testing prior to any dietary intervention.
[b]Results for testing at 8 months after start of the gluten-free diet and 5 months after start of the combined dairy- and gluten-free diet.
[c]A percentage change of greater than 25% was considered significant.

addition to antibodies against wheat proteomes, antibodies in the patient's blood were also highly reactive against milk, α+β casein, casomorphin, milk butyrophilin, American cheese, and milk chocolate (Table 2).

In light of the patient's laboratory results, particularly the significant elevation in antibodies against various wheat proteomes, he was instructed to maintain a gluten-free diet. He was seen at 8 weeks after the dietary intervention, and he reported that his worst headaches were now rated at 5 (8-9 before implementation of diet) and only lasted at most 3 to 4 hours as opposed to all day. His muscle pain completely resolved. Twelve weeks after this partial symptomatic response and amelioration of his headache suffering, or 20 weeks since the beginning of the dietary intervention, the patient was put on a dairy-free diet based on the elevation in antibodies against α+β casein (Table 2). At 6 months after the implementation of this combination of a gluten- and dairy-free diet, the patient's headaches were rated at 3 and did not last more than an hour.

To evaluate the correlation between the IgG and IgA antibody levels and improvement in symptoms, the same lab tests were repeated 5 months after the start of the combined dairy- and gluten-free diet (8 months after the start of the initial gluten-free diet). The results, depicted in the second columns of Tables 1 and 2, show that the elimination diets resulted in a significant decline in antibodies against various wheat proteomes, tTG-6, and milk proteins. This decline in antibody levels against dietary proteins together with improvement in symptoms indicate that the patient's migraine headaches were indeed associated with gliadin and dairy products in his diet.

To determine whether or not years of immune reactivity to gliadin and casein may have resulted in autoimmune reactivity, another set of repeat analyses were performed. Antibodies were measured against 24 different tissue-specific antigens by Cyrex Labs (Table 3). In the first test, the indices show that the patient had significant elevations in antibodies against 10 out of 24 antigens, including cytochrome P450, glutamic acid decarboxylase 65 (GAD-65), collagen, myelin basic protein (MBP), arthritic peptide, tropomyosin, synapsin, adrenal 21-hydroxylase, cerebellar, and antisaccharomyces cerevisiae antibodies (ASCA)+antineutrophil cytoplasmic antibody (ANCA). In the follow-up test after the gluten- and casein-free diet, a significant decline of more than 25% was observed in antibodies against synapsin, 53.85%; cerebellar, 47.83%; arthritic peptide, 41.94%; intrinsic factor, 36.36%; ASCA+ANCA, 30.43%; adrenal 21-hydroxylase, 29.17%; tropomyosin, 28.57%; and collagen, 25.53% (Table 3).

Elevations in autoantibodies targeted against a variety of self-antigens are detected years before symptoms of autoimmune disease occur.[8] If detected early enough, dietary and medical interventions can be initiated to minimize long-term illness and promote a better quality of life.[8,9]

Discussion

Chronic headache in adults represents a pandemic, affecting 21.8% of females and 10% of males. Treatment generally involves pharmaceutical interventions using prophylactic medications to reduce headache frequency and severity and symptomatic medication to reduce pain once a headache is present. Specific dietary triggers have been identified and include chocolate, cheese, caffeine, monosodium glutamate, and alcohol. Less commonly recognized is the role of gluten sensitivity in relation to headaches despite research demonstrating headaches in 56% of gluten-sensitive and 37% of dairy-sensitive individuals.

Table 3. Multiple Autoimmune Reactivity Screen

TESTED PARAMETERS IgG + IgA Antibody	FIRST TEST Antibody/Index[a]	SECOND TEST Antibody/Index[b]	% Change[c]	REFERENCE RANGE Antibody/Index
Parietal cell	1.0	0.9	10.00%	0-1.4
Intrinsic factor	1.1	0.7	**36.36%**	0-1.2
α+β tubulin	1.1	1.3	-18.18%	0-1.4
α-myosin	1.1	1.2	-9.09%	0-1.5
Adrenal 21-hydroxylase	2.4	1.7	**29.17%**	0-1.2
Arthritic peptide	3.1	1.8	**41.94%**	0-1.3
ASCA+ANCA	2.3	1.6	**30.43%**	0-1.4
Cerebellar	2.3	1.2	**47.83%**	0-1.4
Collagen complex	4.7	3.5	**25.53%**	0-1.6
Cytochrome P450	5.7	5.3	7.02%	0-1.6
Fibulin	1.1	1.3	-18.18%	0-1.6
GAD-65	5.3	5.0	5.66%	0-1.4
Ganglioside	1.2	1.3	-8.33%	0-1.4
Insulin islet cell	1.9	2.1	-10.53%	0-1.7
MBP	3.6	2.8	22.22%	0-1.4
Myocardial peptide	1.3	1.4	-7.69%	0-1.5
Osteocyte	1.1	1.2	-9.09%	0-1.4
Ovary/testis	1.2	1.4	-16.67%	0-1.2
Phospholipid	1.4	1.3	7.14%	0-1.3
Platelet glycoprotein	1.0	0.8	20.00%	0-1.3
Synapsin	2.6	1.2	**53.85%**	0-1.2
Tropomyosin	2.8	2.0	**28.57%**	0-1.1
Thyroglobulin	0.8	1.0	-25.00%	0-1.3
Thyroid peroxidase	1.0	1.1	-10.00%	0-1.3

[a]Results for testing prior to any dietary intervention.
[b]Results for testing at 8 months after start of the gluten-free diet and 5 months after start of the combined dairy- and gluten-free diet.
[c]A percentage change of greater than 25% was considered significant.

Abbreviations: ASCA, antisaccharomyces cerevisiae antibodies; ANCA , antineutrophil cytoplasmic antibody; GAD-65, glutamic acid decarboxylase 65; MBP, myelin basic protein.

The specific underlying cause of migraines is unknown.[10] However, experts believe that a number of conditions can trigger an abnormal electrical discharge in the brain that leads to the release of a variety of chemicals that can cause migraines. These conditions include psychological factors, especially stress; hormonal factors; genetic factors[11]; medications; physical events; and food antigens. Experts believe that eating certain foods may cause changes in brain concentrations of chemicals that set off migraine headaches.[12,13] However, the relationship between food and migraines is difficult to prove, and the concept remains controversial. In many cases, food is considered a migraine trigger if the consumed food triggers a headache within 24 hours, and it must do so more than half of the time that the food is eaten. Although food-sensitive individuals produce significant levels of antibodies against various food antigens, the susceptibility to the triggering of a headache through dietary intake is not consistently confirmed by measurements of antibody titers. Furthermore, food-elimination diets are discouraged in clinical practice.[14] Therefore, a need exists for objective laboratory evaluation that documents a patient's sensitivity to certain foods, the correlation of those reactivities to the patient's symptoms, and the improvement and amelioration of the patient's migraine headaches and associated symptoms after the implementation of an elimination diet, as demonstrated in this case.

Based on the patient's family history, the research team decided first to measure IgG and IgA antibodies against various wheat proteomes and different transglutaminases involved in nonceliac gluten sensitivity and CD.[15] Results summarized in Table 1 show that the antibody levels were 2- to 4-fold higher against wheat proteomes as compared to reference ranges established based on the assessment of levels of these antibodies in 120 blood samples of healthy individuals age 18 to 70. The IgG and IgA antibodies against tTG-6 were much higher than antibodies against tTG-2 and tTG-3. This elevation in tTG-6 IgG and IgA further indicates a neurological manifestation of gluten sensitivity in this patient, since antibodies against tTG-6 are found in patients with gluten ataxia.16 Deposits of tTG-6 IgA seem to accumulate in the periphery of vessels, which can enhance vascular leaking.[17] Additionally, tTG isozymes and immune complexes are found in patients with gluten neuropathy or myopathy.[18]

It has been shown that a high degree of homology or cross-reactivity exists between a-gliadin-33-mer peptide and bovine α+β casein.[19] This homology between milk protein and gliadin has been demonstrated not only by both polyclonal and monoclonal, gliadin-antibody immune reactivity with milk proteins[20] but also in the in-vivo system.[21] This in vivo study was conducted to investigate why many celiac patients did not improve significantly even when on a gluten-free diet. Suspecting the possible involvement of cow's milk protein, researchers used rectal protein challenge to investigate the inflammatory reaction to gluten and milk proteins in 20 adult CD patients and 15 healthy controls. A mucosal inflammatory response similar to that elicited by gluten was observed with cow's milk protein in approximately 50% of the patients but not in healthy controls. This finding was determined by the measurement of the release of neutrophil myeloperoxidase and nitric oxide production. The researchers concluded that casein was involved in the induction of CD-like symptoms.[21]

Knowing that gluten cross-reacts with casein,[21] the current research team decided also to measure antibodies against milk-associated antigens. As was the case with gliadin antibodies, the patient demonstrated very high levels of antibodies against dairy products (Table 3). To find if any association existed between the wheat and dairy proteins and his migraine headaches, the patient was first put on a gluten-free diet; the headaches scaled down from 8 or 9 to a much lower 5 and from a duration of all day to just 3 or 4 hours a day. Moreover, the addition of the elimination of dairy products in combination with the gluten restriction resulted in further improvements; the patient's headaches scaled down to a 3, and their duration was reduced to no more than an hour a day. Therefore, significant improvement in clinical symptomatology may indicate that both gluten and casein or their additive effect was indeed involved.

Since it is very well documented that gluten cross-reacts with various tissue antigens, such as joint, bone, heart, brain, neuronal synapsin, thyroid, skin, and endocrine organs,[22-33] the current research team tested the patient's blood against a panel of 24 different tissue antigens to determine whether any relationship existed between the muscle stiffness and autoimmune reactivity. These tests can detect reactivity associated with various body tissues at a very early stage. The patient demonstrated abnormally elevated levels of antibodies against 10 different antigens, including cytochrome P450, GAD-65, collagen, MBP, arthritic peptide, tropomyosin, synapsin, adrenal 21-hydroxylase, cerebellar and ASCA+ANCA (Table 3). After implementation of a gluten- and dairy-free diet, the levels of antibodies against a majority of these antigens declined significantly, except for cytochrome P450 and GAD-65. These results demonstrate an association between the levels of antibodies against gluten and casein and the autoimmune reactivity in this patient suffering from migraine headaches.

Conclusion

In summary, it has been demonstrated that individuals may develop autoimmune injury to the gut, skin, brain, joints, liver, thyroid, bone, reproductive organs, and other parts of the body if gluten and dairy sensitivity are left untreated.[34] The recognition that headaches are among the many extraintestinal manifestations of gluten sensitivity may provide meaningful nonpharmaceutical therapeutic options. The current research team has presented the case

of a patient whose migraine headaches were associated with gluten and dairy consumption. This association was supported by testing for gluten and other dietary-protein sensitivities and the implementation of an elimination diet. Because gluten and casein have been associated with multiple autoimmune reactivities, we had also tested for antibodies against tissue antigens. Not only did the gluten-free and dairy-free diet result in an amelioration of the migraine headache symptomatology, the clinical improvements correlated with a significant decline in the levels of a majority of the previously elevated antibodies. This finding indicates that diet plays a significant role in a subgroup of patients with migraine headaches. The current case study is indeed an exploration of only one case, and further study is needed, but the outcome may not be limited merely to headaches but may extend to the development of autoimmunities if the roles of diet and food sensitivity in disease are real.

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