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Neurologic and Psychiatric Manifestations of Celiac Disease and Gluten Sensitivity

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Abstract

Celiac Disease (CD) is an immune-mediated disease dependent on gluten (a protein present in wheat, rye or barley) that occurs in about 1% of the population and is generally characterized by gastrointestinal complaints. More recently the understanding and knowledge of gluten ${\cal V}$ sensitivity (GS), has emerged as an illness distinct from celiac disease with an estimated prevalence 6 times that of CD. Gluten sensitive people do not have villous atrophy or antibodies that are present in celiac disease, but rather they can test positive for antibodies to gliadin. Both CD and GS may present with a variety of neurologic and psychiatric co-morbidities, however, extraintestinal symptoms may be the prime presentation in those with GS. However, gluten sensitivity remains undertreated and underrecognized as a contributing factor to psychiatric and neurologic manifestiations. This review focuses on neurologic and psychiatric manifestations implicated with gluten sensitivity, reviews the emergence of gluten sensitivity distinct from celiac disease, and summarizes the potential mechanisms related to this immune reaction.

Keywords: Gluten, Schizophrenia, Neurologic, Immune, Celiac, Psychiatric

Introduction

Celiac disease (CD) is an illness which is currently well understood. This illness is caused by an immune reaction to gluten, a protein found in wheat, barley and rye, and is generally characterized by villous atrophy, crypt hyperplasia, and increased intraepithelial lymphocytes. Presenting symptoms typically include postprandial bloating, steatorrhea, and weight loss, and it is present in about one percent of the population [1]. The diagnosis is confirmed by testing for a number of different antibodies including anti-endomysial antibodies (EMA), antitissue transglutaminase autibodies (tTG), and anti-gliadin antibodies (AGA). In addition to understanding the cause of the disorder and the diagnostic tests to confirm it, we also understand the pathogenic mechanism related to intestinal damage [2] and the genetic basis of the disorder which includes haplotyes HLA-DQ2 or HLA DQ8. The increasing knowledge and understanding of this disorder has brought significant attention by physicians and health care workers in recent years as a disease with significant undesirable consequences. Yet, it is believed that many cases continue to go unidentified and untreated.

Only in recent years have we begun to understand gluten sensitivity, a gluten-mediated immune reaction that exists separate from CD and gluten allergic reactions (IgE mediated). Gluten sensitivity is estimated to occur at 6 times greater frequency than CD and is believed to be characterized by a different type of immune mediated reaction [3]. People with GS do not have villous atrophy or antibodies to tTG or EMA, but rather they can test positive to antibodies to gliadin [4]. Also, while the majority of people with CD test positive to HLA-DQ2 or DQ8, only 50% of people thought to be gluten sensitive will test positive for these haplotypes [2]. Another differentiating factor is the presence of interleukin 17A (IL-17A) gene expression in biopsy specimens of CD which is not present in gluten sensitive patients. In addition to laboratory evidence of this distinct difference [5], clinical data also provides evidence. Kaukinen et al. [6] had shown that in a population of slightly under 100 people who reported abdominal symptoms after consumption of gluten only 9% had CD, 8% had latent CD, 20% had a cereal allergy and 63% were not classified as having CD or an allergy. Of these 59 people, 10 (17%) presented with increases in CD3 T-helper cell receptor bearing intraepithelial lymphocytes but were negative for HLA DQ8. Forty percent also had anti-gliadin antibodies (either IgG or IgA). Lastly, Sapone and colleagues [5] recently reported that gluten sensitive patients in comparison to CD patients, showed normal intestinal permeability and activation of the innate but not the adaptive immune response. This suggests that in gluten sensitive patients there is a lack of adaptive immune response that prevents the autoimmune gastrointestinal insults that are seen in CD patients.

The relationship of celiac disease to neurologic and psychiatric complications has been observed for over 40 years [7, 8]. Gluten sensitive patients also have a host of neurologic and psychiatric complications. However it is notable, based on the lack of gut involvement, that neurologic and psychiatric complications seen in gluten sensitive patients may be the prime presentation in patients suffering from this disease. Therefore gluten sensitivity may easily go unrecognized and untreated. Data suggests that up to 22% of patients with CD develop neurologic or psychiatric dysfunction [9], and as many as 57% of people with neurological dysfunction of unknown origin test positive for anti-gliadin antibodies [10]. Neurologic and psychiatric complications observed with gluten-mediated immune responses include a variety of disorders. For example, a PubMed literature search (dates 1953-2011) located 162 original articles associating psychiatric and neurologic complications to celiac disease or gluten sensitivity. Thirty-six articles were located for seizure disorders, 20 articles for ataxia and cerebellar degeneration, 26

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for neuropathy, 20 for schizophrenia, 14 for depression, 12 for migraine, and up to 10 articles each for anxiety disorders, attention deficit and hyperactivity disorder, autism, multiple sclerosis, myasthenia gravis, myopathy, and white matter lesions. Because the vast majority of research to date has not separated gluten sensitivity from celiac disease the true prevalence of neurologic and psychiatric complications associated with each is difficult to quantify. This review however brings focus to the fact that gluten sensitivity is distinct from CD and that gluten-mediated immune responses may be the cause of patients presenting with a host of psychiatric and neurologic complications. We review here the literature as it relates to psychiatric and neurologic complications known to be associated with any gluten-mediated disorder (GS or CD) and the potential pathophysiology associated with these complications as seen in gluten sensitivity in particular.

Evidence of Neurologic Complications

Gluten Ataxia

The best-characterized neurologic complication related to gluten sensitivity is ataxia, now termed "gluten ataxia". Gluten ataxia is characterized by positive anti-gliadin antibodies, changes in the cerebellum, and ataxic symptoms including upper or lower limb ataxia, gait ataxia, and dysarthria [11]. One study showed that 41% of 143 patients with sporadic idiopathic ataxia had anti-gliadin antibodies compared to only 12% of control subjects [10]. In addition to anti-gliadin antibodies, patients with gluten ataxia have oligoclonal bands in their cereberospinal fluid, inflammation at the cerebellum, and anti-Purkinje cell antibodies [4]. Changes in the cerebellum on post-mortem examination include Purkinje cell loss with cerebellar atrophy and Bergmann astrocytosis [12]. Some persons with gluten ataxia have antibodies that show reactivity with deep cerebellar nuclei brainstem and cortical neurons. These studies also suggest that persons with gluten ataxia may have additional antibodies that react with Purkinje cells and are not present in patients with anything other than gluten ataxia alone. It seems likely that the Purkinje cells of the cerebellum share epitopes with gliadin proteins [13].

A study by Hadjivassiliou et al. [14] measured the response of patients with gluten ataxia and neuropathy to administration of a gluten-free diet. After 1 year on a gluten-free diet, the patients experienced significant relief of their ataxic symptoms on all tests. Several studies have shown that screening for celiac disease and gluten sensitivity is beneficial in patients with ataxias and neuropathies of indefinite origin [14-16].

Epilepsy and Seizure Disorders

Epilepsy is another documented neurological manifestation of GS or CD. The prevalence of celiac cases in people with epilepsy ranges from approximately 0.8-6% [17, 18]. The clinical picture generally includes a specific triad of symptoms- occipital calcifications, seizures originating from a number of brain locations; and GS or CD. A study by Pfaender et al. [19] describes patients with visual manifestations due to seizure activity, which included blurred vision and seeing colored dots. In this study all the patients had bilateral cortical calcification and celiac disease. Researchers in Argentina identified 32 patients at their clinic with this triad of symptoms (seizures, CD, bilateral cortical calcification). Of the patients with hypodense areas in the white matter around their calcifications, three patients had a reduction of these areas on a gluten-free diet. As expected, seizure activity was better managed in the patients who received the earliest gluten-free diets [20]. A smaller study of four patients with this triad of symptoms reported that three of the four patients had significant reduction of their seizure activity after going on a gluten-free diet [21]. A number of studies have reported similar improvement in patients with this triad of symptoms encompassing seizures, GS or CD, and cortical calcifications [22-24].

Epilepsy related to GS and CD may not always manifest in the occipital lobe. For example, Peltola et al. [25] compared patients with temporal lobe epilepsy and hippo-campal sclerosis, to those with temporal lobe epilepsy and no hippocampal sclerosis, and to those with extra-temporal epilepsy alone for prevalence of gluten sensitivity or CD. Seven of the 16 patients with temporal lobe epilepsy and hippocampal sclerosis were positive for GS while none of the patients from the other two groups had CD or GS. Overall, review of these epilepsy articles supports screening patients with idiopathic epilepsies for gluten sensitivity and celiac disease.

Other Neurological Manifestations

Other neurological manifestations of gluten sensitivity and celiac disease include peripheral neuropathy [26], inflammatory myopathies [27], myelopathies [3], headache [28], and gluten encephalopathy [29]. White matter abnormalities associated with gluten sensitivity have also been reported [30].

Evidence of Psych atric Complications

A wide range of psychiatric symptoms and disorders have been associated with CD and GS. Those occurring mostly commonly, as reported here, include anxiety disorders [31] depressive and mood disorders [32, 33], attention deficit hyperactivity disorder (ADHD) [34], autism spectrum disorders [35], and schizophrenia [2, 36-38]. While there is limited research on the relationship of most psychiatric disorders to GS and CD, accumulating evidence suggests a variety of connections.

Anxiety Disorders

Various types of anxiety are associated with gluten intolerance. One study found that CD patients were significantly more likely to have state anxiety when compared to controls, and that after 1 year on a gluten-free diet, there was a significant improvement in state anxiety symptoms [31]. Other anxiety disorders such as social phobia and panic disorder have been linked to gluten response. Addolorato and colleagues [39] reported that a significantly higher proportion of CD patients had social phobia compared to normal controls. Additionally, a higher lifetime prevalence of panic disorder has been found in CD patients [33] and new studies have confirmed the increased association between CD and anxiety [40].

Depression and Mood Disorders

Overview of celiac disease.



What is celiac disease? Celiac disease is an inherited autoimmune disorder that affects the digestive process of the small intestine. The small intestine is connected to the stomach; the first parts of the small intestine—
the duodenum and the jejunum—are where celiac disease is commonly found.

When a person who has celiac disease consumes gluten—a protein found in wheat, rye, and barley—the individual's immune system responds by attacking the small intestine, inhibiting the absorption of important nutrients into the body. Specifically, the tiny fingerlike protrusions called villi on the lining of the small intestine are lost. Normally, nutrients from food are absorbed into the bloodstream through these villi. Celiac disease can be associated with other autoimmune disorders and, if undiagnosed and untreated, can lead to osteoporosis, infertility, neurological conditions, and, in rare cases, cancer.

What is dermatitis herpetiformis (DH)?

Dermatitis herpetiformis (DH) is an itchy, blistering skin condition that is a form of celiac disease. The rash usually occurs on the elbows, knees, and buttocks, and is characterized by its bilateral nature, which means that both knees and/or both arms are affected, seldom just one. Many people with DH have no digestive symptoms and only about 40% of them have positive blood tests (serology) for celiac disease; however, they almost always have

the same gluten-dependent intestinal damage as people with celiac disease.

Unless otherwise specified, the information pertaining to celiac disease also pertains to people with dermatitis herpetiformis. In addition to the required, strict gluten-free diet, DH is also commonly treated with a medication called dapsone.

Is celiac disease a rare condition?

No. Celiac disease affects at least 1% of Americans, or nearly 3 million people in the United States. By comparison, Alzheimer's disease affects approximately 2 million people. It is possible to be diagnosed with celiac disease at any age.

Is it possible to have celiac disease but have NO symptoms?

Yes. Research has demonstrated that a significant percentage of children and adults with positive celiac blood tests had no, or minimal, symptoms when they were tested. Further, there are a few patients who carry the gene for celiac disease and have no or minimal symptoms and negative blood tests, yet a positive biopsy shows that the disease is active.

Why is it difficult to find a doctor who knows about celiac disease?

Most physicians learned during medical school that celiac disease is so rare they would likely never see a patient with symptoms in their entire medical career. Lectures on celiac disease in medical schools, even today, are few and far between. When your doctor was in medical school, he or she may have heard a 20 to 30 minute celiac disease lecture during 4 years of classes. Medical textbooks still contain outdated information.

Additionally, <u>celiac disease often</u>
<u>presents with seemingly unrelated</u>
<u>symptoms, such as fatigue, joint</u>
<u>pain, anemia, and infertility, making,</u>
<u>diagnosis that much more difficult.</u>

The University of Chicago Celiac Disease Center is working hard to properly educate doctors about celiac disease so that those at risk for the disease are screened immediately.

For more information, contact The University of Chicago Celiac Disease Center at www.cureceliacdisease.org.

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ILLINOIS DEPARTMENT OF CORRECTIONS

Auxiliary Aids & Services Assessment / Communication Plan

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If yes, list changes:	
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A. Deaf:	Left ear Right ear Both
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DeafBlind or Visually Impaired:	Yes No (X)
3. Offender uses sign language?	Yes No No
If yes, is sign language the Offender's primary language?	Yes No
Offender's proficiency?	Fluent Conversational Beginner
C. Type of interpreter needed:	ASL (American Sign Language Signed English Signed English Sign Language from other Country Cother: DONL NUCLEO
D. Any Secondary Disabilities which If yes, please list:	could limit communication? Yes No
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Assessment of Reading / Wr For example: Is the personal able to r	ead and write? Does the person have the ability to engage in basic communications
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Distribution: Offender, Offender's Medical File, Facility ADA Coordinator, Agency ADA Coordinator

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