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Recurrent Psychosis in Non-celiac Gluten Sensitivity

Scarlet Louis-Jean
Department of Internal Medicine, Luminis Health Anne Arundel Medical Center, Annapolis, MD, USA, scarletl@auamed.net

Shire Chaudhry
Department of Internal Medicine, Luminis Health Anne Arundel Medical Center, Annapolis, MD, USA

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CASE REPORT

Recurrent Psychosis in Non-celiac Gluten Sensitivity

Scarlet Louis-Jean*, Shire Chaudhry

Department of Internal Medicine, Luminis Health Anne Arundel Medical Center, Annapolis, MD, USA

Abstract

Neuropsychological manifestations following food exposures in patients with food sensitivities are increasingly being identified in the literature, as understanding of the gut–brain axis is further improved. Non-celiac gluten sensitivity (NCGS) has been shown to occur in individuals without serological or biopsied evidence of celiac disease (CD), who manifest psychotic or mood disorders that resolve following elimination of gluten. In this case history, we discuss a similar manifestation in a 31-year-old woman without serological evidence of CD, whose psychiatric symptoms improve with gluten elimination.

Keywords: Non-celiac gluten sensitivity, Psychosis, Mood disorder

1. Introduction

The gut–brain axis has been established as a bidirectional pathway involving the neural, endocrine, immune, and humoral networks mediated by the gut microbiome, with increasing recognition of its associations with functional gastrointestinal and central nervous system disorders (e.g., anxiety-depressive disorders).[1] The phenomenon of psychosis due to non-celiac gluten sensitivity has been recently depicted in the literature with several case reports detailing neuro-psychiatric manifestations, such as schizophrenia, depression, and other mood disorders resolving after the removal of gluten from patients' diets.[2] The diagnosis is often made with a double-blind gluten challenge.[2]

2. Case report

A 31-year-old Caucasian woman with a history of severe gluten sensitivity, Hashimoto's disease, mosaic turner's syndrome, and presumed schizoaffective disorder, bipolar type with multiple inpatient psychiatric involuntary admissions presented to the ED on petition for aggressive behavior by law enforcement. The patient had a week-long breakdown at her parents' home, where she also resides. She reportedly broke several objects and threatened to slit her mother's throat. On the physical exam, she was malodorous, disheveled, and had long extremely matted hair. Psychiatric evaluation was notable for rambling speech, dysphoric mood, agitation, and irritability. Thought content was disorganized and consisted of grandiose and paranoid persecutory delusions. She was emergently given Haldol for worsening agitation, as she became physically threatening.

Per records, the patient was not on antipsychotic medications as she did not have any noticeable response to prior treatment. During previous admissions, she was managed on a gluten-free diet and dietary supplements, as she was presumed to have gluten sensitivity without serological evidence of celiac disease based on the association of her symptoms in the context of gluten exposure (Table 1). Per family, the patient regresses into psychosis following gluten ingestion, which often takes 2–3 weeks to completely resolve in the setting of a strict gluten-free diet. However, due to recurrent intentional exposures, her rate of resolution has been reportedly prolonging in course. The patient has a history of gluten binges prior to multiple psychiatric admissions (Fig. 1). Before this encounter she was stable for approximately four months, during which time she maintained a non-confrontational relationship with her parents and began working on a book series with her mother. The patient reportedly went on a gluten-binge that propelled her into another acute psychotic episode when her mother went away to visit another family member.

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* Corresponding author.
E-mail address: ScarletL@auamed.net (S. Louis-Jean).

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Table 1. Laboratory results detailing the patient's celiac disease panel, thyroid function and autoimmune panels, and infectious disease serologies.

Laboratories	Results	Reference Ranges
Celiac Disease Serologies		
Deamidated gliadin IgA	5 units	<20 units
Deamidated gliadin peptide IgG	3 units	<20 units
Endomysial IgA antibody	Negative	–
Tissue transglutaminase antibody	Negative	–
IgA (Total)	Normal	–
Thyroid Function Test and Autoimmune Serologies		
TSH	0.867 uIU/mL	0.380–4.700 uIU/mL
Free thyroxine	1.22 ng/dL	0.93–1.70 ng/dL
Thyroglobulin Ab	27 IU/mL	≤1 IU/mL
Thyroid peroxidase Ab	106 IU/mL	<9 IU/mL
Infectious Disease Serologies		
Syphilis IgG/IgM Ab	Non-reactive	–
HIV	Non-reactive	–

Ab: antibody; Ig: immunoglobulin; HIV: human immunodeficiency virus; TSH: thyroid-stimulating hormone.

During her current involuntary admission, the patient often appeared disengaged and uninterested in participating in conversation. She would wander aimlessly around the unit, often responding to internal stimuli. She was managed on low-dose risperidone as needed, which she received following bouts of agitation. She was maintained on a gluten-free diet and dietary supplements, and her psychiatric evaluation improved within 4 weeks. On discharge she was prescribed risperidone with anticipated tapering, which would be mediated by her outpatient psychiatrist. She was also recommended to follow up with a gluten challenge and further evaluation of gluten sensitivity at a university hospital, with which she did not proceed.

3. Discussion

NCGS is a syndrome characterized by gastrointestinal symptoms of abdominal pain, bloating, and alterations in bowel habit, along with systemic or neuropsychiatric manifestations such as weight loss, anemia, fogginess, fatigue, and depression.[2,3] It is estimated that NCGS accounts for a general prevalence of 0.6–13% and has been reported to occur frequently in women in their fourth decade who are domiciled in urban areas.[4] The association of celiac disease induced psychosis or mood disorders, among several other autoimmune diseases, have been discussed in the literature; however, the epidemiology of NCGS and psychiatric disorders remain to be studied.[5]

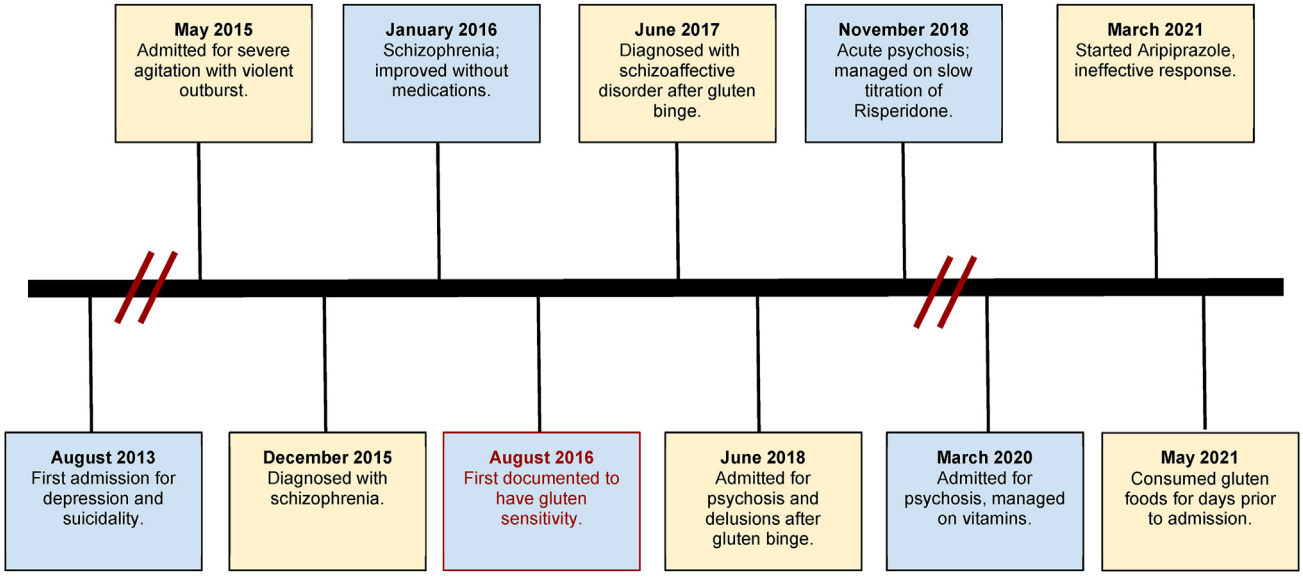

Fig. 1. This is a timeline of the patient's psychiatric hospitalizations, beginning with her first documented episode of mood disorder. Each hospitalization is denoted by salient points that were documented in her admission records. The patient was noted to have consumed gluten with all admissions following her August 2016 admission. The slanted red bars depict one to several year lapses between each admission.

Currently, the pathophysiology of NCGS is largely unknown; however, intestinal inflammation and the activation of the innate immune system and its components are being explored as contributing pathophysiologic mechanisms.[6] Triggering food products include gluten, amylase-trypsin inhibitors, and fermentable oligo-, di-, and monosaccharides, and polyols (FODMAPs).[6] The diagnosis is established following removal of gluten, often after the exclusion of wheat allergy or CD, and is confirmed in the absence of classic celiac disease small bowel histopathology demonstrating villous atrophy, increased lamina propria density, and crypt hyperplasia.[2] Diagnostic certainty can be enhanced by adhering to a gluten free diet for 6 weeks followed by completing a double blinded placebo-controlled gluten challenge, as recommended by the Salerno Experts' Criteria; however, this approach is cumbersome and can be unfeasible in clinical practice.[6,7]

Extraintestinal manifestations of NCGS, such as neuropsychiatric symptoms as evidenced by our patient have been reported in the literature with a spectrum from depression, ataxia, encephalopathy, and psychosis.[8] Management of NCGS is largely limited to dietary elimination of gluten and other triggering foods which can attenuate symptoms; however, long term maintenance of a gluten free diet has not shown to elicit a complete amelioration of symptoms.[6,9,10] Our patient exhibited a potential dose-dependent gluten insensitivity response, with prolonged rates of resolution following recurrent exposure. We believe that further investigation is warranted to explore the existence of a dose-dependent association as there remains a paucity of data of this aspect.

4. Conclusion

NCGS is an evolving entity with multi-systemic organ involvement and its extraintestinal symptoms involving the neuropsychiatric axis can challenge clinicians' differential diagnosis, disease identification, work-up, and diagnosis. Increased awareness of NCGS through case report contributions and additional research is warranted, as it will elucidate the underlying pathophysiology, further define clinical presentation and spectrum, and develop a sustainable management and treatment plan.

Conflict of interest

No conflicts of interest statement.

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