
**Abstract**

Celiac disease (CD) is a lifelong immune-mediated disorder caused by the ingestion of wheat gluten in genetically susceptible persons. Most cases of CD are atypical and remain undiagnosed, which exposes the individuals to the risk of life-threatening complications. Serologic endomysial and tissue transglutaminase antibody tests are used to screen at-risk individuals, although a firm diagnosis requires demonstration of characteristic histopathologic findings in the small-intestinal mucosa. A gluten challenge, with a repeat biopsy to demonstrate recurrence of histopathologic changes in the intestinal mucosa after the re-introduction of gluten, is considered for those persons in whom diagnosis remains in doubt. In this paper, we review studies that evaluated: (1) the possibility of using oral mucosa for the initial diagnosis of CD or for local gluten challenge; and (2) the possibility of using salivary CD-associated antibodies as screening tests. Our review shows that orally based diagnosis of CD is attractive and promising, although additional evaluations with standardized collection and analysis methods are needed. There is some evidence of a dissociation between systemic and oral mucosal immune responses in CD. The hypothesis that gluten could stimulate naive lymphocytes directly in the oral cavity would have important implications for the understanding, diagnosis, and management of CD.


**Abstract**

Gluten sensitivity appears to be emerging as a separate condition from celiac disease, yet no clear definition or diagnosis exists. As a result, patients with gluten sensitivity experience delayed diagnosis and continuing symptoms if they consume gluten. This emerging medical problem may involve human genetics, plant genetic modifications, gluten as a food additive, environmental toxins, hormonal influences, intestinal infections and autoimmune diseases. The treatment is similar to that for celiac disease - a gluten-free diet. The use of a gluten-free diet or an elimination diet is encouraged in assisting people to determine whether or not they are gluten sensitive. It is time to not only recognize, but to treat and further research gluten sensitivity, as unconfirmed environmental factors continue to spread this problem further into the genera population.

Abstract

Until a few years ago, celiac disease (CD) was thought to be a rare food intolerance that was confined to childhood and characterized by severe malabsorption and flat intestinal mucosa. Currently, CD is regarded as an autoimmune disorder that is common in the general population (affecting 1 in 100 individuals), with possible onset at any age and with many possible presentations. The identification of CD is challenging because it can begin not only with diarrhea and weight loss but also with atypical gastrointestinal (constipation and recurrent abdominal pain) and extra-intestinal symptoms (anemia, raised transaminases, osteoporosis, recurrent miscarriages, aphthous stomatitis and associated autoimmune disorders), or it could be completely symptomless. Over the last 20 years, the diagnostic accuracy of serology for CD has progressively increased with the development of highly reliable tests, such as the detection of IgA tissue transglutaminase and antiendomysial and IgG antideamidated gliadin peptide antibodies. The routine use of antibody markers has allowed researchers to discover a very high number of 'borderline' cases, characterized by positive serology and mild intestinal lesions or normal small intestine architecture, which can be classified as potential CD. Therefore, it is evident that the 'old celiac disease' with flat mucosa is only a part of the spectrum of CD. It is possible that serology could identify CD in its early stages, before the appearance of severe intestinal damage. In cases with a positive serology but with mild or absent intestinal lesions, the detection of HLA-DQ2 and HLA-DQ8 can help reinforce or exclude the diagnosis of gluten sensitivity.


Abstract

OBJECTIVES:

Exposure to gliadin and related prolamins and appropriate HLA-DQ haplotype are necessary but not sufficient for contracting celiac disease (CD). Aberrant innate immune reactions could be contributing risk factors. Therefore, jejunal biopsies were screened for bacteria and the innate immune status of the epithelium investigated.

METHODS:

Children with untreated, treated, challenged CD, and controls were analyzed. Bacteria were identified by scanning electron microscopy. Glycocalyx composition and mucin and antimicrobial peptide production were studied by quantitative RT-PCR, antibody and lectin immunohistochemistry.

RESULTS:

Rod-shaped bacteria were frequently associated with the mucosa of CD patients, with both active and inactive disease, but not with controls. The lectin Ulex europaeus agglutinin I (UEAI) stained goblet cells in the mucosa of all CD patients but not of controls. The lectin peanut agglutinin (PNA) stained glycocalyx of controls but not of CD patients. mRNA levels of mucin-2 (MUC2), alpha-defensins HD-5 and HD-6, and lysozyme were significantly increased in active CD and
returned to normal in treated CD. Their expression levels correlated to the interferon-gamma mRNA levels in intraepithelial lymphocytes. MUC2, HD-5, and lysozyme proteins were seen in absorptive epithelial cells. beta-defensins hBD-1 and hBD-2, carcinoembryonic antigen (CEA), CEA cell adhesion molecule-1a (CEACAM1a), and MUC3 were not affected.

CONCLUSIONS:
Unique carbohydrate structures of the glycocalyx/mucous layer are likely discriminating features of CD patients. These glycosylation differences could facilitate bacterial adhesion. Ectopic production of MUC2, HD-5, and lysozyme in active CD is compatible with goblet and Paneth cell metaplasia induced by high interferon-gamma production by intraepithelial lymphocytes.


SEVEN YEARS AGO, I sat down for an in-depth interview with Alessio Fasano, MD, director of the Center for Celiac Research, shortly after his landmark study that determined one in 133 people has celiac disease. There was great excitement at that time about all the things likely to happen now that it was clear celiac disease was much more common than anyone thought. We met that time in his office at the University of Maryland School of Medicine in downtown Baltimore. It seemed fitting to be talking about the study and other cutting edge research not far from the celiac center clinic lab where much of the work was going on. This time we talked over lunch in a noisy pizza parlor that has gluten-free pizza on its menu—something we could only have dreamed about in 2003. Fasano, a prolific celiac researcher, writer, ambassador and advocate, had just returned from an ambitious trip to Europe where he traveled between Finland, Germany and his native Italy. In Finland, he donned a topcoat with tails and a very tall hat to participate in formal proceedings where a fellow researcher was defending his thesis on the role the biopsy plays in the diagnosis of celiac disease. Read More…


**Abstract**

The 14th International Celiac Disease Symposium (ICDS) took place in Oslo, Norway, on 20-22 June 2011, with 530 registered attendees, including clinicians, researchers, sponsors and patients. The 3-day symposium included an international scientific section, and a concurrent clinical forum. It provided a comprehensive overview of the basic, clinical and translational aspects of celiac disease in two keynote lectures, 11 introductory presentations, ten lectures from experts, 227 poster presentations and 38 oral presentations. It highlighted the importance of integrating the knowledge from clinics, genetic studies and immunological studies to better understand celiac disease.

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Abstract

BACKGROUND:
There is an increased prevalence of osteoporosis among patients with celiac disease. However, the relative prevalence of celiac disease among osteoporotic and nonosteoporotic populations is not known, and the benefit of screening the osteoporotic population for celiac disease remains controversial.

METHODS:
We evaluated 840 individuals, 266 with and 574 without osteoporosis, from the Washington University Bone Clinic by serologic screening for celiac disease. Individuals with positive serologic test results for antitissue transglutaminase or antiendomysial antibody were offered endoscopic intestinal biopsy to confirm the diagnosis of celiac disease. Individuals with biopsy-proven celiac disease were treated with a gluten-free diet and followed up for improvement in bone mineral density.

RESULTS:
Twelve (4.5%) of 266 patients with osteoporosis and 6 (1.0%) of 574 patients without osteoporosis tested positive by serologic screening for celiac disease. All but 2 serologically positive individuals underwent intestinal biopsy. Nine osteoporotic patients and 1 nonosteoporotic patient had positive biopsy results. The prevalence of biopsy-proven celiac disease was 3.4% among the osteoporotic population and 0.2% among the nonosteoporotic population. All biopsy-positive individuals tested positive by antitissue transglutaminase and antiendomysial antibody. The antitissue transglutaminase levels correlated with the severity of osteoporosis as measured by T score, demonstrating that the more severe the celiac disease the more severe the resulting osteoporosis. Treatment of the patients with celiac disease with a gluten-free diet resulted in marked improvement in T scores.

CONCLUSIONS:
The prevalence of celiac disease among osteoporotic individuals (3.4%) is much higher than that among nonosteoporotic individuals (0.2%). The prevalence of celiac disease in osteoporosis is high enough to justify a recommendation for serologic screening of all patients with osteoporosis for celiac disease.


Celiac disease is an immune-mediated enteropathy triggered by the ingestion of gluten-containing grains (including wheat, rye, and barley) in genetically susceptible persons. The disease is associated with HLA-DQ2 in 90 to 95 percent of cases and with HLA-DQ8 in 5 to 10 percent of cases and is self-perpetuating in the continued presence of gluten. It is the interplay between genes (both HLA and other types) and environment (i.e., gluten) that leads to the intestinal damage that is typical of the disease. Under physiologic circumstances, this interplay is prevented by competent intercellular tight junctions, structures that limit the passage of macromolecules . . .

**Abstract**

**OBJECTIVE:**
The goal was to evaluate the impact of immunoglobulin A endomysial antibody testing on the incidence and clinical presentation of childhood celiac disease.

**METHODS:**
The incidence and clinical presentation of celiac disease in patients <18 years of age in 1990-1996 (pretesting group) versus 2000-2006 (testing group) were compared.

**RESULTS:**
The median age at diagnosis was 2 years (95% confidence interval: 2-4 years) in the pretesting group (N = 36), compared with 9 years (95% confidence interval: 8-10 years) in the testing group (N = 199; P < .001); the female/male ratios (1.6:1) were similar (P = .982). The incidence of celiac disease increased from 2.0 cases per 100000 children (pretesting group) to 7.3 cases per 100000 children (testing group; P = .0256). The frequency of classic celiac disease presentations decreased from 67% (pretesting group) to 19% (testing group; P < .001), but the incidence of classic celiac disease did not differ (0.8 vs 1.6 cases per 100000; P = .154). In the testing group, 13 previously unrecognized clinical presentations were observed in 98 children, including 35 with family history, 18 with abdominal pain, and 14 with type 1 diabetes mellitus. The frequency of Marsh IIIc lesions decreased from 64% (pretesting group) to 44% (testing group; P = .0403). In the testing group, classic celiac disease remained predominant (67%) in young children (<3 years), whereas atypical gastrointestinal and silent presentations predominated in older children.

**CONCLUSIONS:**
Antibody testing for celiac disease tripled the incidence of celiac disease and quadrupled the median age at diagnosis.


**Abstract**

**INTRODUCTION:**
To explore whether the excess mortality in celiac disease is related directly to the disease and duration of gluten exposure before diagnosis we have examined the long-term mortality experience of people with celiac disease diagnosed as children and as adults.

**METHODS:**
Two hundred eighty-five children and 340 adults diagnosed with celiac disease were followed until death, loss to follow-up, or December 31, 2004. We calculated standardized mortality ratios (SMRs).
RESULTS:

All-cause mortality more than 5 yr after diagnosis was increased threefold in children (SMR 3.32, 95% CI 2.05-5.07) compared with only a 38% increase in adults (SMR 1.38, 95% CI 1.16-1.63). This excess mortality in children was primarily because of an increased risk of death from accidents, suicide, and violence (seven deaths, SMR 3.22, 95% CI 1.29-6.63), cancer (five deaths, SMR 3.72, 95% CI 1.21-8.67), and cerebrovascular disease (two deaths, SMR 10.03, 95% CI 1.21-36.00).

CONCLUSIONS:

Children diagnosed with celiac disease had a threefold increased risk of long-term mortality. This is in marked contrast to the experience of adult celiac disease where the long-term increase of mortality was modest. The increased mortality in children from external causes may reflect behavioral change associated with coping with a chronic disease and its treatment.


BACKGROUND AND AIM:

Individuals with coeliac disease have increased risk of depression and death from external causes, but conclusive studies on death from suicide are missing. We examined the risk of suicide in coeliac disease and amongst individuals where the small intestinal biopsy showed no villous atrophy.

METHODS:

We collected biopsy data from all 28 clinical pathology departments in Sweden for individuals diagnosed during 1969-2007 with coeliac disease (Marsh 3: villous atrophy; n=29,083 unique individuals), inflammation without villous atrophy (Marsh 1-2; n=13,263) or positive coeliac disease serology but normal mucosa (Marsh 0, n=3719). Through Cox regression we calculated Hazard ratios for suicide as recorded in the Swedish Cause of Death Register.

RESULTS:

The risk for suicide was higher in patients with coeliac disease compared to general population controls (HR=1.55; 95%CI=1.15-2.10; based on 54 completed suicides). Whilst suicide was also more common amongst individuals with inflammation (HR=1.96; 95%CI=1.39-2.77), no such increase was seen amongst individuals with a normal mucosa but positive coeliac disease serology (HR=1.06; 95%CI=0.37-3.02).

CONCLUSIONS:

We found a moderately increased risk of suicide amongst patients with coeliac disease. This merits increased attention amongst physicians treating these patients.
Abstract

CONTEXT:
Studies of mortality in celiac disease have not taken small-intestinal pathology into account.

OBJECTIVE:
To examine mortality in celiac disease according to small-intestinal histopathology.

DESIGN, SETTING, AND PATIENTS:
Retrospective cohort study. We collected data from duodenal/jejunal biopsies taken between July 1969 and February 2008 on celiac disease (Marsh stage 3: villous atrophy; n = 29,096 individuals) and inflammation (Marsh stage 1-2; n = 13,306) from all 28 pathology departments in Sweden. A third cohort consisted of individuals with latent celiac disease from 8 university hospitals (n = 3719). Latent celiac disease was defined as positive celiac disease serology in individuals with normal mucosa (Marsh stage 0). Through linkage with the Swedish Total Population Register, we estimated the risk of death through August 31, 2008, compared with age- and sex-matched controls from the general population.

MAIN OUTCOME MEASURE:
All-cause mortality.

RESULTS:
There were 3049 deaths among patients with celiac disease, 2967 with inflammation, and 183 with latent celiac disease. We found an increased hazard ratio (HR) for death in celiac disease (HR, 1.39; 95% confidence interval [CI], 1.33-1.45; median follow-up, 8.8 years), inflammation (HR, 1.72; 95% CI, 1.64-1.79; median follow-up, 7.2 years), and latent celiac disease (HR, 1.35; 95% CI, 1.14-1.58; median follow-up, 6.7 years). The absolute mortality rate was 10.4 (95% CI, 10.0-10.8) per 1000 person-years in celiac disease, 25.9 (95% CI, 25.0-26.8) in inflammation, and 6.7 (95% CI, 5.7-7.6) in latent celiac disease. Excess mortality was 2.9 per 1000 person-years in celiac disease, 10.8 in inflammation, and 1.7 in latent celiac disease. This risk increase was also seen in children. Excluding the first year of follow-up, HRs decreased somewhat.

CONCLUSION:
Risk of death among patients with celiac disease, inflammation, or latent celiac disease is modestly increased.

**Abstract**

Public anxiety over gluten has fuelled widespread demand for gluten-free food, yet coeliac disease remains significantly underdiagnosed and some confusion remains regarding optimal diagnostic practices. Small bowel histology is the gold standard for diagnosis. High-quality commercial enzyme-linked immunosorbent assays for transglutaminase immunoglobulin A and deamidated gliadin immunoglobulin A and G are sensitive tools for screening, but almost 10% of coeliac disease is seronegative and serological testing is unreliable in the very young, in people already following a gluten-reduced diet, and those using immunosuppressive medications. HLA DQA and DQB genotyping to show that alleles encoding HLA DQ2 and DQ8 are absent virtually excludes coeliac disease. Confirming histological remission reduces the risks of later complications, such as osteoporosis and cancer. Monitoring remission by serology is unreliable. Because gluten is an exogenous antigen and the small intestine is readily accessible, the immunopathogenesis of coeliac disease is better understood than other strongly major histocompatibility complex class II-associated diseases, such as type 1 diabetes mellitus. Therapeutic targets have been identified and drugs are under development to supplement or even replace gluten-free diet. With greater awareness and non-dietary therapeutics, diagnosis and treatment of coeliac disease will be increasingly prominent in medical practice.


**Abstract**

**BACKGROUND & AIMS:**

Patients with non-celiac gluten sensitivity (NCGS) do not have celiac disease but their symptoms improve when they are placed on gluten-free diets. We investigated the specific effects of gluten after dietary reduction of fermentable, poorly absorbed, short-chain carbohydrates (fermentable, oligo-, di-, monosaccharides, and polyols [FODMAPs]) in subjects believed to have NCGS.

**METHODS:**

We performed a double-blind cross-over trial of 37 subjects (aged 24-61 y, 6 men) with NCGS and irritable bowel syndrome (based on Rome III criteria), but not celiac disease. Participants were randomly assigned to groups given a 2-week diet of reduced FODMAPs, and were then placed on high-gluten (16 g gluten/d), low-gluten (2 g gluten/d and 14 g whey protein/d), or control (16 g whey protein/d) diets for 1 week, followed by a washout period of at least 2 weeks. We assessed serum and fecal markers of intestinal inflammation/injury and immune activation, and indices of fatigue. Twenty-two participants then crossed over to groups given gluten (16 g/d), whey (16 g/d), or control (no additional protein) diets for 3 days. Symptoms were evaluated by visual analogue scales.

**RESULTS:**

In all participants, gastrointestinal symptoms consistently and significantly improved during
reduced FODMAP intake, but significantly worsened to a similar degree when their diets included gluten or whey protein. Gluten-specific effects were observed in only 8% of participants. There were no diet-specific changes in any biomarker. During the 3-day rechallenge, participants’ symptoms increased by similar levels among groups. Gluten-specific gastrointestinal effects were not reproduced. An order effect was observed.

CONCLUSIONS:
In a placebo-controlled, cross-over rechallenge study, we found no evidence of specific or dose-dependent effects of gluten in patients with NCGS placed diets low in FODMAPs.


Abstract

BACKGROUND:
Current evidence suggests that many patients with self-reported non-coeliac gluten sensitivity (NCGS) retain gastrointestinal symptoms on a gluten-free diet (GFD) but continue to restrict gluten as they report ‘feeling better’.

AIM:
To investigate the notion that a major effect of gluten in those with NCGS is on mental state and not necessarily on gastrointestinal symptoms.

METHODS:
Twenty-two subjects (24-62 years, five male) with irritable bowel syndrome who had coeliac disease excluded but were symptomatically controlled on a GFD, undertook a double-blind cross-over study. Participants randomly received one of three dietary challenges for 3 days, followed by a minimum 3-day washout before crossing over to the next diet. Challenge gluten-free food was supplemented with gluten (16 g/day), whey (16 g/day) or not supplemented (placebo). End-points included mental state as assessed by the Spielberger State Trait Personality Inventory (STPI), cortisol secretion and gastrointestinal symptoms.

RESULTS:
Gluten ingestion was associated with higher overall STPI state depression scores compared to placebo [M = 2.03, 95% CI (0.55-3.51), P = 0.010] but not whey [M = 1.48, 95% CI (-0.14 to 3.10), P = 0.07]. No differences were found for other STPI state indices or for any STPI trait measures. No difference in cortisol secretion was identified between challenges. Gastrointestinal symptoms were induced similarly across all dietary challenges.

CONCLUSIONS:
Short-term exposure to gluten specifically induced current feelings of depression with no effect on other indices or on emotional disposition. Gluten-specific induction of gastrointestinal symptoms was not identified. Such findings might explain why patients with non-coeliac gluten sensitivity feel better on a gluten-free diet despite the continuation of gastrointestinal symptoms.
Abstract

BACKGROUND:
Non-celiac gluten sensitivity (NCGS) is still an undefined syndrome with several unsettled issues despite the increasing awareness of its existence. We carried out a prospective survey on NCGS in Italian centers for the diagnosis of gluten-related disorders, with the aim of defining the clinical picture of this new syndrome and to establish roughly its prevalence compared with celiac disease.

METHODS:
From November 2012 to October 2013, 38 Italian centers (27 adult gastroenterology, 5 internal medicine, 4 pediatrics, and 2 allergy) participated in this prospective survey. A questionnaire was used in order to allow uniform and accurate collection of clinical, biochemical, and instrumental data.

RESULTS:
In total, 486 patients with suspected NCGS were identified in this 1-year period. The female/male ratio was 5.4 to 1, and the mean age was 38 years (range 3-81). The clinical picture was characterized by combined gastrointestinal (abdominal pain, bloating, diarrhea and/or constipation, nausea, epigastric pain, gastroesophageal reflux, aphthous stomatitis) and systemic manifestations (tiredness, headache, fibromyalgia-like joint/muscle pain, leg or arm numbness, 'foggy mind,' dermatitis or skin rash, depression, anxiety, and anemia). In the large majority of patients, the time lapse between gluten ingestion and the appearance of symptoms varied from a few hours to 1 day. The most frequent associated disorders were irritable bowel syndrome (47%), food intolerance (35%) and IgE-mediated allergy (22%). An associated autoimmune disease was detected in 14% of cases. Regarding family history, 18% of our patients had a relative with celiac disease, but no correlation was found between NCGS and positivity for HLA-DQ2/-DQ8. IgG anti-gliadin antibodies were detected in 25% of the patients tested. Only a proportion of patients underwent duodenal biopsy; for those that did, the biopsies showed normal intestinal mucosa (69%) or mild increase in intraepithelial lymphocytes (31%). The ratio between suspected NCGS and new CD diagnoses, assessed in 28 of the participating centers, was 1.15 to 1.

CONCLUSIONS:
This prospective survey shows that NCGS has a strong correlation with female gender and adult age. Based on our results, the prevalence of NCGS seems to be only slightly higher than that of celiac disease.
INTRODUCTION:
Crohn's disease (CD) and ulcerative colitis (UC), the two main forms of inflammatory bowel disease, are characterised by increased mucosal activation of pro-inflammatory signalling molecules. The anti-tumour necrosis (TNF)-alpha monoclonal antibody infliximab is more effective in the treatment of CD than UC but its mechanism of action is still unknown. We therefore evaluated the effect of infliximab on the expression of a panel of phospho-proteins by inflamed CD and UC colonic biopsies cultured ex vivo.

METHODS:
Colonic biopsies were obtained from macroscopically inflamed areas of 5 patients with CD and 2 patients with UC, and were then cultured for 24 h in 300ul of serum-free HL-1 medium with infliximab (5ug/ml), or control IgG1 (5ug/ml). The biopsies were then snap frozen and later lysed to extract the protein. A Path Scan RTK signalling array kit from New England Biolabs was used to measure the expression of a panel of 39 phosphorylated proteins in the biopsy homogenates.

RESULTS:
Infliximab significantly reduced the expression of phosphorylated ALK, FLT3, EphB3,p44/42 MAPK, S6 Ribosomal Protein and Stat1 by over 40 fold compared to IgG1 in Crohn's disease biopsies cultured ex vivo. In UC biopsies infliximab did not induce any significant change in phosphoprotein expression compared to IgG1 control except for a 10 fold reduction in phospho-VEGFR2.

CONCLUSION:
Infliximab reduces the expression phospho-proteins Stat 1, ALK and p44/42 MAPK, which have a central role in sustaining the pro-inflammatory immune response. Differences in the effect of infliximab on the phosphorylation status of mucosal proteins may account for its different efficacy profile in CD and UC.


Coeliac disease is uncommon in childhood and diagnosed in fewer than 1 in 2500 children in the United Kingdom. Subclinical disease is, however, common in adults, and can be detected by testing for serum IgA antiendomysial antibodies (IgA-EMA). We aimed to establish the prevalence of undiagnosed coeliac disease in the general population at age seven, and to look for associated clinical features.
Dr. Tom O'Bryan is an internationally recognized speaker and workshop leader specializing in the complications of Non-Celiac Gluten Sensitivity and Celiac Disease as they occur inside and outside of the intestines. He is the founder of www.theDr.com. He recently hosted the paradigm-shifting ‘The Gluten Summit - A Grain of Truth’, bringing together 29 of the world’s experts on Celiac Disease and Non-Celiac Gluten Sensitivity at www.theglutensummit.com.