International Journal of Celiac Disease, 2019, Vol. 7, No. 3, 66-68
Available online at http://pubs.sciepub.com/ijcd/7/3/8
Published by Science and Education Publishing
DOI:10.12691/ijcd-7-3-8

Celiac Disease Clinical, Pathophysiological, Epidemiological and Therapeutical Repertoire is Expanding

Aaron Lerner*, Torsten Matthias

AESKU.KIPP Institute, Wendelsheim, Germany
*Corresponding author: aaronlerner1948@gmail.com

Received October 15, 2019; Revised November 20, 2019; Accepted December 04, 2019

Abstract The present issue of the International Journal of Celiac Disease contains multiple aspects of celiac disease, spanning rare and new clinical presentations, associated conditions, reminds us on the lectin hypothesis of celiac disease, new epidemiological data on Africa, explore compliance with gluten elimination and describes the "waste bin diagnosis" of Sprue-like intestinal disease. Following is an overview of the issue content with a drop of a personal dip. Congratulations to the Editor and the Editorial Board of the International Journal of Celiac Disease (CD) for publishing volume 7, No 3 issue of the Journal.

Keywords: *celiac disease, clinical presentation, epidemiology, pathophysiology, gluten free diet, complications*

Cite This Article: Aaron Lerner, and Torsten Matthias, "Celiac Disease Clinical, Pathophysiological, Epidemiological and Therapeutical Repertoire is Expanding." *International Journal of Celiac Disease*, vol. 7, no. 3 (2019): 66-68. doi: 10.12691/ijcd-7-3-8.

It fulfills the journal's vision of deepening various aspects of the disease, imparting knowledge, and encouraging readers and scientists to "crack" the nut of the disease. In this regard, the current issue spans rare clinical presentations, renewed pathophysiological pathways, new epidemiological data and the college hard times in compliance with a gluten-free diet (GFD). Following is an overview of the issue content with a drop of a personal dip:

The review article brings up the lectin theory in CD induction [1]. After summarizing the luminal lectins' transit, their nutrients' and drugs' effects, and sugar specificity, the CD lectin hypothesis is reviewed. The lectin hypothesis of CD started in 1972 by Ament, Douglas, Weiser, et al. [2,3,4]. It is quite controversial between the pros [5,6,7,8], and cons [9,10]. The annual number of publications is decreasing in the last decade. However, most recently, the lectin theory was revived and even raised the question about dietary lectin exclusion as the next big food trend [11]. It seems that the jury is still out.

The first manuscript reported on the prevalence of CD autoimmunity in Ethiopian pregnant women to be quite low (0.05%). The authors describe the limitations of the study. However, several aspects should be zoomed out considering CD in Africa [12] even though a lot of local and international effort is allocated to fight infections and improve public health and quality of life. Allergies, cancer, and autoimmune diseases are increasing in Africa, as is in Western societies. In the Maghreb countries, CD incidence, phenotype, and epidemiology resemble the developed world. However, lack of awareness, resources, and qualified health care professionals, change of dietary habits, poverty, illiteracy, malnutrition, and infectious load are at the heart of the problematic situation. Gudeta AN et al. [13] are discussing reasons for the low incidence of autoimmune CD in Ethiopian pregnant women. Most probably, the trend of Westernization of the African continent and the adoption of gluten-containing processed foods will increase the CD burden in Ethiopia and other African countries.

The next manuscript switches to CD therapy with GFD. It explored the various individual and social influences on compliance with gluten withdrawal in college students [14]. GFD is a tough alley, and the adolescent and college periods are hard times [15,16]. In this sense, it should be remembered that gluten has several side effects, but on the other hand, the only approved therapy for CD, the GFD, is also not performed without risks. The clinical and dieticians communities should be aware of those drawbacks in the face of the gluten withdrawal boom "fashionista" [17].

The first case report expands on a new wheat-dependent entity entitled "wheat-dependent exercise-induced anaphylaxis" [18]. Until now, CD, non-CD/wheat sensitivity, wheat/gluten allergy, dermatitis herpetiformis, and gluten ataxia filled the list of wheat/gluten depended on conditions. The new entity is a particular allergy that occurs after wheat ingestion, followed by physical exercise. Dyspnea, wheezing, and respiratory allergy dominated the clinical picture. In this regard, we can show that there is an association between CD and lung disease [19]. In addition, asthma seems to be a high-risk factor for CD development [20]. The wheat-related conditions have a broad spectrum of 'evolving diseases' [21]. However, the

associative relations should be strengthened by a more causative relationship.

The presentation of CD in the elderly and its multisystem involvement is not new [22]. The authors describe the association between immune thrombocytopenic purpura (ITP), collagenous microscopic colitis, and mesangial-proliferative glomerulonephritis in elderly CD patients. The peculiar features of CD in the elderly were recently summarized [23]. Multiple environmental factors, luminal eco-events, gut-peripheral organ pathways, and axes exist, thus, connecting CD to extra-intestinal end organs [24-33].

Volume 7 issue 3 of the Journal continues with a description of the celiac crisis, manifested by hypokalemic quadriparesis [34]. The acute presenting symptoms of CD were summarized recently [35], and the electrolyte deficiency mediated peripheral neuro-muscular abnormality should be added to the list [36].

The last paper is an exception in the present issue since it does not deal with CD, but instead with the association of Sprue-like intestinal disease with Crohn's disease [37]. Crohn's disease is associated with CD [38,39,40,41], and adding Sprue-like disease, all three cohabit in the human intestine. In the case presented, Sprue-like symptoms were not affected by GFD, and CD serology was negative, despite biopsy changes mimicking those of CD. Although a "waste bin diagnosis," Sprue-like diseases are a heterogeneous group that might present as a crisis [42], thus, mimicking the celiac crisis [43].

In summary, volume 7, issue 3 embraces several aspects of CD, including rare clinical presentations, renewed pathophysiological pathways, new epidemiological data and the college hard times in compliance with a gluten-free diet.

References

[1] Freeman HJ. Topographic Lectin Mapping of the Epithelial Cell Surface in Normal Intestine and Celiac Disease. Internat J of Celiac Dis. 2019; 7(3).

[2] Ament ME, Rubin CE. Soy protein--another cause of the flat intestinal lesion. Gastroenterology. 1972; 62: 227-34.

[3] Douglas AP. The binding of a glycopeptide component of wheat gluten to intestinal mucosa of normal and coeliac human subjects. Clin Chim Acta. 1976; 73: 357-61.

[4] Weiser MM, Douglas AP. An alternative mechanism for gluten toxicity in coeliac disease. Lancet. 1976; 1(7959): 567-9.

[5] Fälth-Magnusson K, Magnusson KE. Elevated levels of serum antibodies to the lectin wheat germ agglutinin in celiac children lend support to the gluten-lectin theory of celiac disease. Pediatr Allergy Immunol. 1995; 6: 98-102.

[6] Boniotto M, Braida L, Spanò A, Pirulli D, Baldas V, Trevisiol C, et al. Variant mannose-binding lectin alleles are associated with celiac disease.Immunogenetics. 2002; 54: 596-8.

[7] Iltanen S, Mäki M, Collin P, Mustalahti K, Kaukinen K, Partanen J, et al. The association between mannan-binding lectin gene alleles and celiac disease. Am J Gastroenterol. 2003; 98: 2808-9.

[8] Boniotto M, Braida L, Baldas V, Not T, Ventura A, Vatta S, et al. Evidence of a correlation between mannose binding lectin and celiac disease: a model for other autoimmune diseases. J Mol Med (Berl). 2005; 83: 308-15.

[9] de Carvalho EG, da Rosa Utiyama SR, da Silva Kotze LM, de Messias Reason IT. Serum mannan-binding lectin levels in patients with celiac disease: an analysis of clinical and autoimmune features.Dig Dis Sci. 2007; 52: 2145-51.

[10] da Silva Kotze LM, de Carvalho EG, da Rosa Utiyama SR, Nisihara RM, Messias-Reason I. Mannan-binding lectin levels related to spontaneous abortion in Brazilian patients with celiac disease. Dig Dis Sci. 2008; 53: 3152-7.

[11] Panacer K, Whorwell PJ. Dietary Lectin exclusion: The next big food trend? World J Gastroenterol. 2019; 25: 2973-2976.

[12] Lerner A, Lopez F, Schmiedl A, Matthias T. The Underdiagnosed Enemy: Africa Goes Celiac? Internat. J Celiac Dis. 2019; 7: 9-12.

[13] Gudeta AN, Brundin c, Feyissa DM, Balcha TT, Agardh D. A Cross Sectional Study from the Oromia Region. Internat J of Celiac Dis. 2019; 7.

[14] Sparks C, Zingg T, Cheney MK. Individual and Social Influences on College Student Compliance with a Gluten-Free Diet. Internat J of Celiac Dis. 2019; 7.

[15] Samasca G, Lerner A , Sur G, Lupan L, Makovicky P, Matthias T, Freeman HJ. Challenging in Gluten-free diet in celiac disease: Prague consensus. Eur J Clin Invest. 2017; 47: 394-397.

[16] Lerner A, Matthias T. Gluten free diet- tough alley in torrid time. Internat J of Celiac Disease 2017; 5: 50-55.

[17] Lerner A, O'Bryan T, Matthias T. Navigating the gluten-free diet boom: the dark side of gluten free diet. Frontiers in Pediatrics. 2019; 7: article 414.

[18] Rahmoune H, Boutrid N, Amrane M, Kherkhache T, Bioud B. Wheat-dependent Exercise-induced Anaphylaxis. Internat J of Celiac Dis. 2019; 7.

[19] Tarlo SM, Broder I, Prokipchuk EJ, Peress L, Mintz S. Association between celiac disease and lung disease. Chest. 1981; 80: 715-8.

[20] Patel B, Wi CI, Hasassri ME, Divekar R, Absah I, Almallouhi E, et al. Heterogeneity of asthma and the risk of celiac disease in children. Allergy Asthma Proc. 2018; 39: 51-58.

[21] Gasbarrini G, Mangiola F. Wheat-related disorders: A broad spectrum of 'evolving' diseases. United European Gastroenterol J. 2014; 2: 254-62.

[22] Chakrabarti U, Thakur MB, Khanna G. A Rare Case of Celiac Presenting at an Atypical Age with Multisystem Involvement. Internat J of Celiac Dis. 2019; 7.

[23] Lerner A, Matthias T. Increased knowledge and awareness of celiac disease will benefit the elderly. Intern. J of Celiac Dis. 2015; 3: 112-114.

[24] Lerner A, Matthias T. GUT-the Trojan horse in remote organs' autoimmunity. Journal of Clinical & Cellular Immunology, 2016; 7: 401.

[25] Samasca G, Ramesh A, Sur D, Cornel A, Sur L, Flocaa E, SurG, Lupand L, Matthias T, Lerner A. Polyautoimmunity - The missing ingredient. Autoimmun Rev. 2018:17: 840-841.

[26] Lerner A, Matthias T, Wusterhausen P. Autoimmunity in celiac disease: extra-intestinal manifestations. Autoimm. Rev. 2019; 18: 241-246.

[27] Lerner A, Aminov R, Matthias T. Dysbiosis may trigger autoimmune diseases via inappropriate posttranslational modification of host proteins. Frontiers in Microbiology. 2016; 7: Article 84.

[28] Lerner A, Aminov R, Matthias T. Intestinal dysbiotic transglutaminases are potential environmental drivers of systemic autoimmunogenesis. Frontiers in Microbiology, 2017; 8; article 66.

[29] Lerner A, Arleevskaya M, Schmiedl A, Matthias T. Microbes and viruses are bugging the gut in celiac disease. Are they friends or foes? Frontiers in Microbiol. 2017; 8: 1392.

[30] Lerner A, Aminov R, Matthias T. Potential effects of horizontal gene exchange in the human gut. Front Immunol. 2017; 8: 1630.

[31] Lerner A, Ramesh A, Matthias T. David and Goliath war revival in the enteric viruses and microbiota struggle. Potential implication for celiac disease, Microorganisms, 2019; 7: 173.

[32] Lerner A, Berthelot L, Jeremias P, Abbad L, Matthias T, Monteiro RC. Gut-kidney axis: gluten, transglutaminase, celiac disease and IgA nephropathy J Clin Cell Immunol, 2017; 8: 499-503.

[33] Lerner A, Neidhöfer S, Matthias T. The gut microbiome feelings of the brain: perspective for Non-Microbiologists. Microorganisms, 2017; 5(4), 66.

[34] Chakrabarti U, Thakur MB, Goel A. Celiac Disease Presenting as Hypokalemic Quadriparesis. Internat J of Celiac Dis. 2019; 7.

[35] Lerner A, Matthias T. A Silent or Hypo-symptomatic Disease Can Erupt: Acute Presentations of Celiac Disease. Internat J Celiac Dis 2017; 5: 129-132.

[36] Lerner A, Makhoul BF, Eliakim R. Neurological manifestations of celiac disease in children and adults. Europ Neurolog J. 2012; 4: 15-20.

[37] Freeman HJ. Sprue-Like Intestinal Disease Following Crohn's Disease. Internat J of Celiac Dis. 2019; 7.

[38] Lerner A, Neidhöfer S, Matthias T. The gut-gut axis: Cohabitation of celiac, Crohn's disease and IgA deficiency. Internat J Celiac Dis. 2016; 4: 68-70.

[39] Casella G, Di Bella C, Salemme M, Villanacci V, Antonelli E, Baldini V, Bassotti G. Celiac disease, non-celiac gluten sensitivity and inflammatory bowel disease. Minerva Gastroenterol Dietol. 2015; 61: 267-71.

[40] Tse CS, Deepak P, De La Fuente J, Bledsoe AC, Larson JJ, Murray JA, Papadakis KA. Phenotype and Clinical Course of Inflammatory Bowel Disease With Co-existent Celiac Disease. J Crohns Colitis. 2018; 12: 973-980.

[41] Pascual V, Dieli-Crimi R, López-Palacios N, Bodas A, Medrano LM, Núñez C. Inflammatory bowel disease and celiac disease: overlaps and differences. World J Gastroenterol. 2014; 20: 4846-56.

[42] Cellier C, Delabesse E, Helmer C, Patey N, Matuchansky C, Jabri B, et al. Refractory sprue, coeliac disease, and enteropathy-associated T-cell lymphoma. French Coeliac Disease Study Group. Lancet. 2000; 356(9225): 203-8.

[43] Freeman HJ. Refractory celiac disease and sprue-like intestinal disease. World J Gastroenterol. 2008; 14: 828-30.