Celiac disease: Recent observations and some reflections on older ones

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Selected Bibliography

1. Gee S. On The Coeliac Affliction. *St. Bartholomew's Hospital Report* 1888;24:17-20, Quoted in Cooke WT, Holmes GTK, Livingstone C. Coeliac Disease 1984 Churchill & Livingston, Longman Group LTD NYC

The classical clinical description of the "celiac affection" by the great British clinician Samuel Gee. Proposed that "errors in the diet may perhaps be a cause of the disease and that cow's milk is the least suitable kind of food for these children." (a prediction of later recognition of lactase deficiency in celiac disease). Cites a child that "wonderfully throve when fed upon a quart of the best Dutch mussels daily". He concludes that "if a patient be cured at all, it may be by means of diet".)

2. Dicke WK. Doctoral Thesis, University of Utrecht , Netherlands

The origin of the gluten-free diet.

3. Van de Kamer JH, Weijers HA, Dicke WK. Coeliac disease. IV – An investigation into the injurious constituents of wheat in connection with their action on patients with coeliac disease. *Acta Paediatr* 1953; 42:223-231.

2 & 3.These two of a classic series of studies indicate that wheat, barley, and rye as well as oats induce steatorrhea in celiac disease.

4. Paulley JW. Observations on the aetiology of idiopathic steatorrhoea. Br Med J 1954;2:1318-1321.

The first histologic observation of the flat lesion in celiac disease at laporotomy by JW Paully – a British physician.

5. Shiner M, Doniach I. Histopathologic studies in steatorrhea. *Gastroenterology* 1960;38:419-440.

The first demonstration of the flat intestinal lesion by suction biopsy.

6. Rubin CE, Brandborg LL, Phelps PC, Taylor HC. Studies of Celiac Disease. I. The Apparent Identical and Specific Nature of the Duodenal and Proximal Jejunal Lesion in Celiac Disease and Idiopathic Sprue. *Gastroenterology* 1960;38(1):28-49. (Citation Classic)

More extensive observations on the flat lesion in celiac disease in children and idiopathic sprue in adults. This is the origin of the name celiac sprue.

7. Rubin CE, Dobbins WO. Progress in Gastroenterology, peroral biopsy of the small intestine, a review of its diagnostic usefulness. *Gastroenterology* 1965;49:676-697.

Recognizes the characteristic, but non-diagnostic histology of the flat intestinal lesion of untreated celiac sprue. Therefore proposed "a second criteria for a firm diagnosis of celiac sprue – a well documented and dramatic clinical response after institution of a strict gluten-free diet." These two criteria remain the cornerstones for the diagnosis of celiac sprue to this day.

8. Kaukinen K, Turjanmass K, Mäki M, Partanen J, Venäläinen R, Reunala T, & Collin P. Intolerance to Cereals is Not Specific for Coeliac Disease. *Scand J Gastroenterol* 2000; 9:942-946.

Intolerance to gluten in non-celiacs is common and thus the use of a clinical response to a gluten free diet alone is diagnostically unreliable.

9. Dieterich W, Ehnis T, Bauer M, Donner P, Volta U & et al. Identification of tissue transglutaminase as the autoantigen of celiac disease. *Nature Medicine* 1997;3(7):797-801.

10. Dieterich W, Laag E, Schöpper H, Volta U, Ferguson A & et al. Autoantibodies to tissue transglutaminase as predictors of celiac disease. *Gastroenterology* 1998;115:1317-1321.

This is the first work describing the specificity and sensitivity of serum TTG for the diagnostic screening for celiac sprue.

11. Tesei N, Sugai E, Vazquez H, Smecuol E, Niveloni S & et al. Antibodies to human recombinant tissue transglutaminase may detect coeliac disease patients undiagnosed by endomysial antibodies. *Aliment Pharmacol Ther* 2003; 17(11):1415-1423.

12. Tonutti E, Visentini D, Bizzaro N, Caradonna M, Cerni L & et al. The role of antitissue transglutaminase assay for the diagnosis and monitoring of coeliac disease: a French-Italian multicentre study. *J Clin Pathol* 2003;56(5):389-393.

11 &12 These two papers provide further proof of the superiority of serum TTG in screening for celiac disease.

HISTOLOGIC PROOF OF INJURIOUS PROPERTIES OF WHEAT, BARLEY & RYE IN CELIAC SPRUE

13. Quinton WE, Flick AL, Rubin CE. The design of a hydraulic suction tube for peroral biopsy of the human gastrointestinal tract. *Gastroenterology* 1962;42:281-284.

An instrument making it possible to biopsy the whole length of the bowel repeatedly while it remains in situ.

14. Rubin CE, Brandborg LL, Flick AL, Phelps P, Parmentier C & Van Niel S. Studies of celiac sprue – III. The effect of repeated wheat instillation inot the proximal ileum of patients on a gluten free diet. *Gastroenterology* 1962;43(6):621-641.

First real time histologic demonstration of small bowel injury by wheat in celiac sprue.

15. Rubin CE, Brandborg LL, Flick AL, MacDonald WC, Parkins AR & et al. Biopsy studies on the pathogenesis of coeliac sprue. In: Ciba Foundation Study Group No. 14 Intestinal Biopsy. Little, Brown and Company, Boston MA, 1962.

Only wheat, barley, and rye destroy small bowel villi in celiac sprue. Oats do not destroy the villi but only non-specifically cause active inflammation in both celiac sprue and normal controls.

16.MacDonald WC, Flick AL, Brandborg LL, Trier JS, Rubin CE. Studies of celiac sprue. IV. The response of the whole length of the small bowel to a gluten-free diet. *Gastroenterology* 1964;47:573-589.

Severity of malabsorption is proportional to length of injured small bowel.

SEEKING COMPLICATIONS OF CELIAC SPRUE BY SCREENING?

17. Cellier C, Delabesse E, Helmer C, Patey N, Matuchansky C & et al. Refractory sprue, coeliac disease, and enteropathy-associated T-cell lymphoma. *The Lancet* 2000; 356:203-208.

75% of cases of celiac sprue not responding to a gluten free diet have an aberrant T-cell population similar to that seen in T-cell lymphoma complicating celiac disease.

18. Robert ME, Ament ME, Weinstein WM. The Histologic Spectrum and Clinical Outcome of Refractory and Unclassified Sprue. *Am J of Surg Path* 2000;24(5):676-687.

There were histologic patterns predictive of poor outcome: 1. development of collagenous sprue, 2. marked mucosal thinning, 3. gastric metaplasia, 4. gastric inflammation and lymphoma.

19. Catassi C, Fabiani E, Corrao G, et al. Risk of Non-Hodgkin Lymphoma (NHL) in Celiac Disease. *JAMA* 2002;1413-1419.

This is one of the best studies of NHL in celiac disease in an otherwise confusing literature. This paper indicates that early diagnosis of celiac disease by mass screening does not significantly reduce its impact on the general population; therefore it comes out against mass screening to detect lymphoma in celiac disease.

20.Sategna GC, Solerio E, Scaglione N, et al. Duration of gluten exposure in adult coeliac disease does not correlate with the risk for autoimmune disorders. *Gut* 2001;49:502-505.

Early treatment of celiac disease with a GFD does not decrease risk for autoimmune disease.

21. Bagdi E, Diss TC, Munson P, Isaacson PG. Mucosal Intra-epithelial Lymphocytes in Enteropathy-Associated T-Cell Lymphoma (EATL), Ulcerative Jejunitis, and Refractory Celiac Disease Constitute a Neoplastic Population. *Blood* 1999;94(1):260-264.

"Loss of response to a gluten free diet (refractory sprue) and ulcerative jejunitis are complications of celiac disease that may progress to enteropathy-associated T-cell lymphoma (EATL). Both conditions are characterized by the presence of nonlymphomatous monoclonal populations that show clonal identity with the lymphoma itself is also present in the enteropathic mucosa."

DERMATITIS HERPETIFORMIS (DH)

22. Marks J, Shuster S, Watson AJ. Small bowel changes in dermatitis herpetiformis. *Lancet* 1966; 2:1280-1282.

The first description of the flat small intestinal lesion in Dermatitis Herpetiformis (DH).

23. Brow JR, Parker F, Weinstein WM, Rubin CE. The small intestinal mucosa in dermatitis herpetiformis – I. Severity and distribution of the small intestinal lesion and associated malabsorption. *Gastroenterology* 1971; 60(3): 355-361.

Description of the varied small intestinal lesion in a series of DH patients despite almost uniform lack of malabsorption.

24. Weinstein WM, Brow JR, Parker F, Rubin CE. The small intestinal mucosa in dermatitis herpetiformis – II. Relationship of the small intestinal lesion to gluten. *Gastroenterology* 1971;60(3)362-369.

Demonstration that the intestinal lesion in DH is related to gluten.

25. Fry L, Seah PP, Riches DJ, Hoffbrand AV. Clearance of skin lesions in dermatitis herpetiformis after gluten withdrawal. *The Lancet* 1973; Feb 10;1(7798):288-91.

Demonstration that a strict gluten free diet of long duration cures the skin lesion in dermatitis herpetiformis.

26. Weinstein WM. Latent celiac sprue. *Gastroenterology* 1974; 66(4):489-493.

Demonstration that rare patients with a normal proximal bowel in DH also carry the gene for celiac sprue in a latent form that can be made to penetrate by a high gluten diet.

GENERAL REFERENCE

27. National Institutes of Health Consensus Development Conference Statement Celiac Disease June 28-30, 2004. http://consensus.nih.gov/cons/118/118cdc_intro.htm

A generally excellent statement but it does not describe the genetic identity of DH and celiac sprue nor does it recognize that serum TTG is the method of choice for screening for celiac sprue because it is reproducible easily and objectively and has a high sensitivity and specificity.

28. Diamond Jared. Guns, Germs, and Steel, the Fate of Human Societies, WW Norton Inc. NYC, NY 1999 (paperback)

A fabulous book on the origin of human societies and the domestication of plants being the initial step in progression from a primitive hunter-gatherer society to a more complex, sedentary agricultural society. The initial sedentary agricultural society first occurred 8000 BCE in the Fertile Crescent of southwestern Asia, where wild cereals grew prolifically after the end of the Pleistocene era, at the end of the ice age. Emmer wheat and barley were the first plants in the world to be domesticated from the wild cereals in the Fertile Crescent. They remain a major source of nutrition worldwide even though they are two of the three are the toxic grains in celiac disease!

29. MacDonald WC, Dobbins WO, Rubin CE. Studies of the familial nature of celiac sprue using small intestinal biopsy. *New Engl J of Med* 1965;272:448-456.

Family study revealing the celiac gene to be a dominant with incomplete penetrance.

30.Van de Kamer JH. Coeliac Disease: A Historical Review. *Journal of the Irish Medical Association* 1974;67(15):405-406