

Comorbidities in focus

Diagnosing celiac disease and improving quality of life

Celiac disease – a life-long systemic autoimmune disease triggered by gluten ingestion that affects primarily the small intestine but can impact the whole body¹ – afflicts 1 in 100 individuals in the general population.² Once diagnosed, celiac disease management builds on regular laboratory tests for antibody status (IgA tTG), nutritional anemia profile (hemoglobin, hematocrit, folate, ferritin, vitamin B12), complete blood count, thyroid stimulating hormone, and anomalies in vitamin, mineral, lipid, electrolyte, and renal profiles. Adults are advised to undergo a bone mineral density scan within the first year after diagnosis.

One way to identify more celiac disease patients is to recognize that the disorder is often accompanied by extra-digestive manifestations³ and is a known comorbidity in a number of autoimmune conditions, including type 1 diabetes mellitus, Sjögren’s syndrome,⁴ autoimmune thyroid disease,⁴ and autoimmune hepatitis.⁵ Given the increased risk of celiac disease in patients diagnosed with other autoimmune diseases (Figure 1), expanding lab diagnostics to assess patients whose symptoms may be attributable to celiac disease is almost certainly cost-effective and can improve their quality of life.

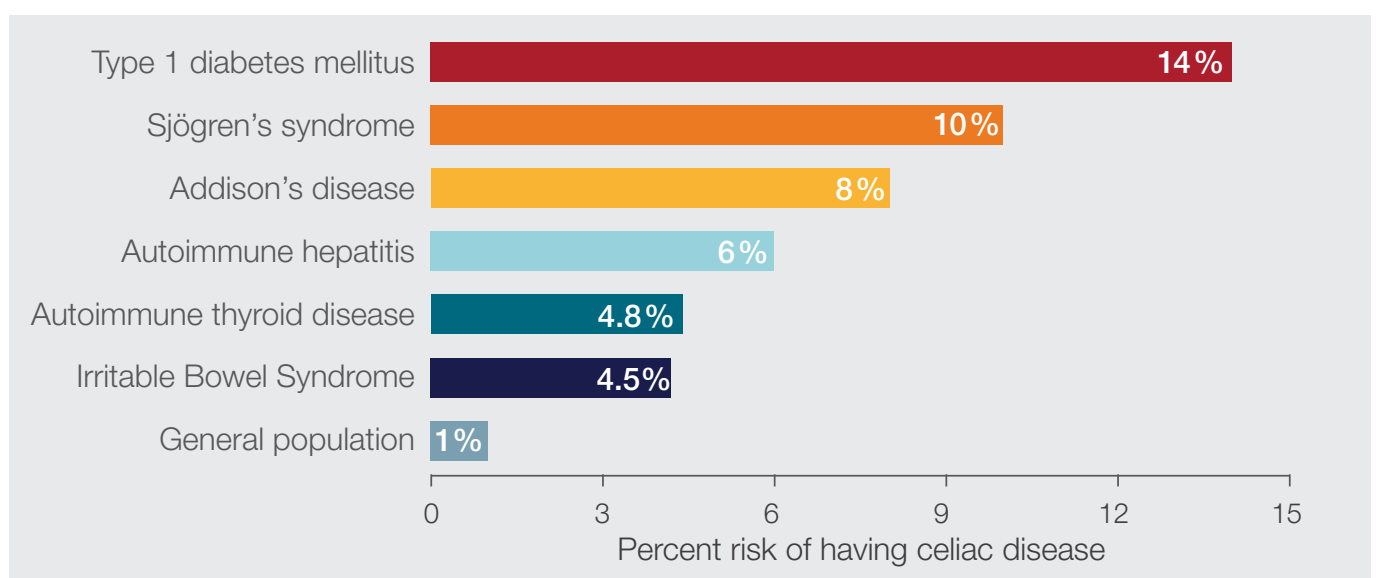


Figure 1: Prevalence of celiac disease in patient populations with common autoimmune conditions compared to the general population.^{4, 5, 7}

Awareness brings improved quality of life



Factors connecting celiac disease and other autoimmune disorders are diverse. Some comorbidities involve a shared genetic base,^{8,9} while overlapping pathogenic mechanisms¹⁰ and compounding metabolic changes or nutrient insufficiencies are implicated in others. Interestingly, celiac disease patients observing a gluten-free diet (GFD) can experience improved clinical course and slower evolution of associated diseases, from resolved iron-deficiency anemia to improved disease management.¹ In most cases (>75%), however, the autoimmune disease is diagnosed before celiac disease¹¹ and the possibility of celiac disease is often overlooked, partly because symptoms are misinterpreted in the context of the associated disease.^{12,13}

An opportunity emerges in laboratories

On average, patients wait 10–13 years from onset of celiac disease symptoms before receiving a correct diagnosis^{14,15,16} and roughly 75% of cases are likely undiagnosed.^{14,17} Clinical laboratories are in the unique position to spearhead more timely and accurate diagnosis of celiac disease. On the one hand, requests to test for associated autoimmune diseases can be expanded to include celiac disease.⁶ On the other hand, untreated celiac disease can lead to deficiencies in iron, vitamins B12, D and E,¹⁸ and other parameters measured in standard blood tests. Long-term monitoring of autoimmunity cases can reveal chemistry patterns that point to concurrent celiac disease. Labs can then highlight those results and suggest testing for celiac disease.

Table 1. Baseline blood parameters measured in 78 adult celiac disease patients (from Lee & Clarke 2017).¹⁸

Parameter	Normal range	Average in celiac disease patients
IgA tTG (U/mL)	<4	12 (1–100)
Iron (mcg/dL)	35–150	79±34
Ferritin (ng/mL)	10–250	48±66
Vitamin B12 (pg/dL)	200–900	557±297
Vitamin D (ng/mL)	30–100	35±14
Vitamin E (mg/L)	4.6–17.8	12.1±4.4

Technical and ordering information

Product	Article No.	Package size	Cut-off		
			Negative	Equivocal	Positive
EliA™ Celikey IgA	14-5517-01	4 x 12	< 7 EliA U/mL	7–10 EliA U/mL	> 10 EliA U/mL
EliA™ Celikey IgG	14-5518-01	2 x 12	< 7 EliA U/mL	7–10 EliA U/mL	> 10 EliA U/mL
EliA™ Gliadin ^{DP} IgA	14-5538-01	4 x 12	< 7 EliA U/mL	7–10 EliA U/mL	> 10 EliA U/mL
EliA™ Gliadin ^{DP} IgG	14-5539-01	4 x 12	< 7 EliA U/mL	7–10 EliA U/mL	> 10 EliA U/mL

References

- Di Sabatino A, et al. Coeliac disease. *Lancet* 2009; 373(9673):1480–93.
- Al-Toma A, et al. *UEG Journal* 2019; 7(5):583–6139.
- Lauret E, et al. *Biomed Res Int*, 2013;127589.
- Green PHR, et al. *Annu Rev Med*, 2006; 57:2007–21.
- Iqbal U, et al. *J Investig Med High Impact Case Rep*, 2017; 5(2):2324709617705679.
- Mohseninejad L, et al. *Eur J Health Econ*, 2013; 14:947–57.
- Verdu EF, et al. *Am J Gastroenterol*, 2009; 104:1587–94.
- Assa A, et al. *Acta Paediatr*, 2017; 106:967–72.
- Ch'ng CL, et al. *Clin Med Res*, 2007; 5:184–92.
- Naiyer AJ, et al. *Thyroid*, 2008; 18:1171–8.
- Slate J, et al. *Dig Dis Sci*, 2005; 50:1705–07.
- Hansen D, et al. *Diabetes Care*, 2006; 29:2452–6.
- Simmons JH, et al. *J Pediatr*, 2011; 158:276–81.e1
- Gujral N, et al. *World J Gastroenterol* 2012;18(42):6036–6059
- Norström F, et al. *BMC Gastroenterology* 2011;11(1):118
- Gray A M, et al. *BMC Health Serv Res* 2010;10:105
- West J, et al. *Am J Gastroenterol* 2014;109(5):757–768
- Lee J, et al. *Scandinavian J Gastroenterol*, 2017; 52:1235–9.

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