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Coeliac disease and dermatitis herpetiformis

We read with interest the Seminar about coeliac disease by Benjamin Lebwohl and colleagues (Jan 6, p 70).[1] However, dermatitis herpetiformis was not listed among the indications that should prompt testing for coeliac disease. Dermatitis herpetiformis is an itchy, blistering skin condition that appears on elbows, knees, and buttocks, with granular IgA deposits in the papillary dermis of perilesional skin.[2] More than 90% of patients with dermatitis herpetiformis have an associated gluten-sensitive enteropathy upon endoscopic examination.[2] The skin symptoms heal with a gluten-free diet (allowing patients to discontinue treatment with dapsone) and relapse on gluten challenge.[2]

Dermatitis herpetiformis and coeliac disease share the same genetic background, with a high frequency of *HLA-DQ2* and *HLA-DQ8* haplotypes.[3] Monozygotic twins born to people with dermatitis herpetiformis have high incidences of both conditions.[4] Autoimmune disorders associated with dermatitis herpetiformis are the same as those associated with coeliac disease, including hypothyroidism, type 1 diabetes, and pernicious anaemia.[2]

IgA antibodies against gut tissue transglutaminase are markers of coeliac disease. In dermatitis herpetiformis, they are thought to cross-react with the highly homologous epidermal transglutaminase.[2] Deposition of IgA and epidermal transglutaminase complexes in the papillary dermis cause dermatitis herpetiformis lesions through complement activation and neutrophil recruitment.[2] Exposure to gluten in the gut is thought to initiate the immune response in dermatitis herpetiformis. Serum samples from patients with gluten-sensitive enteropathy, with or without skin disease, contain IgA antibodies to both transglutaminases,[2] and IgA and epidermal transglutaminase slowly disappear from the papillary dermis of patients with dermatitis herpetiformis under a gluten-free diet.[5]

These factors suggest that dermatitis herpetiformis is a cutaneous manifestation of coeliac disease, and its presence should prompt testing for coeliac disease.

We declare no competing interests.

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Potential coeliac disease is characterised by the presence of serological and genetic markers of coeliac di-sease with little or no damage to the mucosa of the small intestine.[1] Potential coeliac disease is a growing clinical entity, accounting for 10–20% of the total number of coeliac disease cases.[2,3] In their excellent Seminar, Benjamin Lebwohl and colleagues[4] do not give adequate attention to this increasingly observed disorder. Patients with potential coeliac disease can manifest with gastrointestinal or extraintestinal symptoms, or both, or be completely asymptomatic. Because villous atrophy is lacking in potential coeliac disease, whether a gluten-free diet should be recommended to patients is still a matter of debate. The scientific community suggests a gluten-free diet for patients with symptomatic potential coeliac disease, whereas asymptomatic patients are left on a gluten-containing diet and periodically followed up.[2,3,5,6]

With the aim of improving knowledge about potential coeliac disease, our research group has designed a (still ongoing) prospective cohort study[3] and found that about 80% of adult patients with potential coeliac disease are symptomatic and benefit from gluten-free diet.[3] Only 5% of the patients with asymptomatic potential coeliac disease left on a gluten-containing diet progress to active coeliac disease (ie, new onset of villous atrophy) in a median follow-up period of 7 years (mean 6·52 years, SD 3·54). On the basis of these results, we suggest a gluten-free diet for patients with symptomatic potential coeliac disease, whereas asymptomatic patients should be left on a gluten-containing diet. We would be delighted to learn what Lebwohl and colleagues think about the management of patients with potential coeliac disease.

We declare no competing interests.

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Authors' reply

We appreciate the comments by Giacomo Caio and colleagues and François Rodrigues and colleagues. The topics of potential coeliac disease and dermatitis herpetiformis were omitted from our Seminar[1] for reasons related to length and scope. We agree with the principles regarding the management of potential coeliac disease, as outlined by Caio and colleagues, but would like to add two points. First, when assessing a patient who appears to have potential coeliac disease, distinguishing this condition from coeliac disease, in which the diagnosis was missed because of inadequate sampling of the duodenum in relation to the number of specimens submitted,[2] exclusion of the duodenal bulb[3] or misinterpretation of subtle signs of villus atrophy by a pathologist is important.[4] Second, after the diagnosis of potential coeliac disease is confirmed, we suggest that a gluten-free diet be offered in symptomatic patients with potential coeliac disease, but that the diet be considered provisional based on the patient's response. Given the poor correlation between symptoms and coeliac disease,[5] the assumption that gluten is causing symptoms in a given patient is not always correct; if a patient with potential coeliac disease does not improve after a trial of the gluten-free diet, considering alternative causes of symptoms and liberalising the diet is reasonable.

Dermatitis herpetiformis is closely related to coeliac disease with regard to pathogenesis, as outlined by Rodrigues and colleagues, with most patients with dermatitis herpetiformis exhibiting enteropathy. However, one important distinction is that a non-dietary therapy, dapsone, is effective at treating dermatitis herpetiformis, and can be used in conjunction with a gluten-free diet.[6] We should learn from patients' experiences with dapsone now that we are in an era in which non-dietary therapies for coeliac disease are on the horizon.

We declare no competing interests.

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Health of Hungarians: worsens or improves?

In an Editorial (April 21, p 1549),[1] Viktor Orbán, currently third-time re-elected prime minister of Hungary, was suggested as not delivering health for his nation. Since this opinion is based on certain health-care indicators, I should like to supplement with additional data. The Editors state that "Under Orbán's leadership, the number of new cases of HIV/AIDS has more than doubled in a decade, rising from 1·0 per 100 000 in 2005 to 2·7 per 100 000 in 2015." Since the population of Hungary is about 10 million, these figures represent 107 new cases in 2005, and 271 new cases in 2015.[2] However, a major problem with this statement is that Prime Minister Orbán has been in government since 2010 rather than 2005. The period from 2002 to 2010 falls under three previous socialist governments, a fact that is relevant to the interpretation of the data. In 2005, the number of recognised new cases of HIV infection was 107, whereas 182 new cases were registered in 2010, and 271 new cases were registered in 2015, showing that the increase during the pre-Orbán period was greater (70%) than between 2010 and 2015 (49%). However, to look only at the yearly number of newly recognised HIV cases does not reveal much about the quality of health care because this figure also reflects the rising number of people who voluntarily get tested for HIV annually. A more detailed analysis of the available data shows that, although the cumulative number of people with HIV increased gradually from 1638 cases in 2010 to 2747 cases in 2015, the annual number of patients dying from AIDS remained the same (ten deaths in 2010 and 11 deaths in 2015),[2] which indicates improved care of patients with AIDS after 2010.

	2010	2016	Decrease or increase in the number of deaths (%)
Cause of death			
Acute myocardial infarction	7·481	5·744	–23%
Ischaemic heart disease	26·361	25·540	–5%
Stroke	14·001	10·701	–24%
Hepatic diseases	4·622	3·306	–31%
Malignancies	32·460	32·978	1·5%
Neonatal death	481	365	–24%
Suicide	2·492	1·763	–29%
Motor vehicle accidents	793	692	–13%
Life expectancy at birth	74·7 years	76·2 years	2% (1·5 years)

Population of Hungary: 10 014 324 in 2010 and 9 830 485 in 2016 (–2%).

Table: **Number of deaths and life expectancy at birth in Hungary in 2010 and 2016**