

Assessment Outcomes of H Pylori in Iraqi Young Women and Children with Irritable Bowel Syndrome and Celiac Disease

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Abstract: Background: One of the uncommon infectious organisms, *Helicobacter pylori* (HP), causes several gastroduodenal illnesses, including chronic gastritis, duodenal ulcers, as well as adenocarcinoma. **Objective:** Our study aims to assess the outcomes of *H. pylori* in children with Celiac disease. **Patients and methods:** This paper was aimed to assess the outcomes of *H. pylori* in Iraqi children with Celiac disease for 80 cases, which have included two groups. Where the first group was the patients' group (patients with Celiac disease), with 40 cases, while the second group was the control group (patients without Celiac disease), with 40 cases, the databases of outcomes were conducted at the different hospitals in Iraq, which organized between 16th July 2021 to 25th August 2022. The methodology of databases was analysed and extracted by the SPSS program. **Discussion:** In line with prior research, we discovered small, non-significant variations in the blood levels of IgA-tTG in individuals who tested positive of *H. pylori* in comparison to those who did not. In contrast to children who tested negative for *H. pylori*, some of the latter we have had a slight rise in IgA-AGA levels. Regarding histological changes, we discovered that, in comparison to patients who were negative for *H. pylori* and displayed more severe intestinal damage, including complete villous atrophy, a greater proportion of CD patients had milder forms of enteropathy. **Conclusion:** In conclusion, the connection between stomach HP infection and celiac disease (CD) is inverse, proving that HP does not constitute a risk factor for CD. Additional research is needed to validate the conflicting evidence on the link between HP infection and celiac disease (CD).

Keywords: Celiac disease (CD); Endomysium antibody level (EMA); *Helicobacter pylori* (HP); and Doku-transglutaminase antibody level (DTG).

INTRODUCTION

One of the uncommon infectious organisms, *Helicobacter pylori* (HP), causes several gastroduodenal illnesses, including chronic gastritis, duodenal ulcers, as well as adenocarcinoma [Basyigit, S. *et al.*, 2017; Ivarsson, A. *et al.*, 2000]. It is also linked to the emergence of several autoimmune disorders. A prevalent autoimmune condition of the intestines called celiac disease (CD) is brought on by gluten [Stene, L.C. *et al.*, 2006 - Yazdani, R. *et al.*, 2017]. Despite the well-established pathophysiology of CD, its rising incidence has prompted researchers to investigate a variety of environmental risk factors which might cause autoimmunity to gluten to the small intestine. [McCarty, T.R. *et al.*, 2018 - Sergi, C. *et al.*, 2017] If a person is vulnerable, seroconversion to CD can happen at any stage of life. The fourth and fifth decades, as well as early infancy, are when the diagnosis is made most frequently. Under the age of 60, females are at a higher risk (2:1 – 3:1) than males [Ikeda, Y. *et al.*, 2005 - Caio, G. *et al.*, 2019]. Although many CD cases go untreated, roughly 2.2 million children globally had the CD as of 2010. An estimated 42,000 children worldwide every year pass away

from CD-related problems, and 4% of all diarrhea-related fatalities may be related to untreated celiac disease. Gluten may be present in oats. Additionally, gluten protein may be included in lip balm, Playdoh (Hasbro, Pawtucket, RI), toothpaste, mouthwash, envelope and stamp adhesive, mineral and vitamin supplements, as well as herbal and nutritional supplements. [Kalach, N. *et al.*, 2017 - Tosco, A. *et al.*, 2011]

PATIENTS AND METHODS

This paper was aimed to assess the outcomes of *H. pylori* in Iraqi children with Celiac disease for 80 cases, which have included two groups. Where the first group was the patients' group (patients with Celiac disease), with 40 cases, while the second group was the control group (patients without Celiac disease), with 40 cases. The databases of outcomes were conducted at the different hospitals in Iraq, which organized between 16th July 2021 to 25th August 2022. The methodology of databases was analysed and extracted by the SPSS program.

To follow-up of methodology, the databases were designed for the Distribution of in Iraqi children

with Celiac disease based on ages under 11 years for 80 cases under 11 years that can be seen in Table 1, sex into males and females were defined in Table 2, as well as BMI that include (16.5-18.5), (18.5 - 24.9), (25 - 30) which can be cleared in Table 3. Furthermore, the databases outcomes of clinical characteristics feature of H. pylori in Iraqi children with Celiac disease based on symptoms which contain Abdominal pain, Constipation, Diarrhea, Heartburn, and Vomiting where these outcomes can be determined in Table 4.

In addition, our results were defined biochemical and serological parameters of H. pylori in Iraqi children with Celiac disease, where the parameters outcomes include Haemoglobin, Iron, AST, ALT, IgA-tTG, and IgA-AGA, which can be defined in Figure 1. Our study was compared between the patients' group and control group throughout the Histology of H. pylori in Iraqi children with Celiac disease based on Marsh I-II and Marsh IIIa-c,

where these outcomes have been shown in Figure 2. To further of outcomes, the study was conducted with serological and histopathological data of autoantibodies for H. pylori in Iraqi children with Celiac disease DTG U/mL and EMA U/mL that it can be expressed in Figure 3. Our study was extended results parameters with HP+ and HP- of H. pylori in Iraqi children with Celiac disease, which include Haemoglobin, TTG > 100, Scalloping, and Gastritis, that can be defined in Table 5. Moreover, this study was evaluated of outcomes of Iraqi children with Celiac disease based on histopathological scoring. The parameters were determined into acute inflammation, chronic inflammation, mucosal atrophy, intestinal metaplasia, Pseudopyloral metaplasia, and Infections, where the results have been seen in Table 6.

RESULTS

Table 1: Distribution of H. pylori in Iraqi children with Celiac disease based on ages under 11 years for 80 cases

N	V	11
	Mi	0
M		6.0000
SEM		1.00000
Me		6.0000
Mo		1.00 ^a
SD		3.31662
Var		11.000
Min		1.00
Max		11.00
S		66.00

Table 2: Distribution of H. pylori in Iraqi children with Celiac disease based on sex

		F, 40	P (%)	VP (%)	CP (%)
V	Female	47	58.8	58.8	58.8
	Male	33	41.3	41.3	100.0
	T	80	100.0	100.0	

Table 3: Distribution of H. pylori in Iraqi children with Celiac disease based on BMI

		F, 40	P (%)	VP (%)	CP (%)
V	16.5-18.5	34	42.5	42.5	42.5
	18.5 - 24.9	18	22.5	22.5	65.0
	25 - 30	28	35.0	35.0	100.0
	T	80	100.0	100.0	

Table 4: Clinical characteristics features of *H. pylori* in Iraqi children with Celiac disease based on symptoms

		F, 40	P (%)	VP (%)	CP (%)
V	Abdominal pain	22	27.5	27.5	27.5
	Constipation	16	20.0	20.0	47.5
	Diarrhea	13	16.3	16.3	63.7
	Heartburn	14	17.5	17.5	81.3
	Vomiting	15	18.8	18.8	100.0
T	80	100.0	100.0		

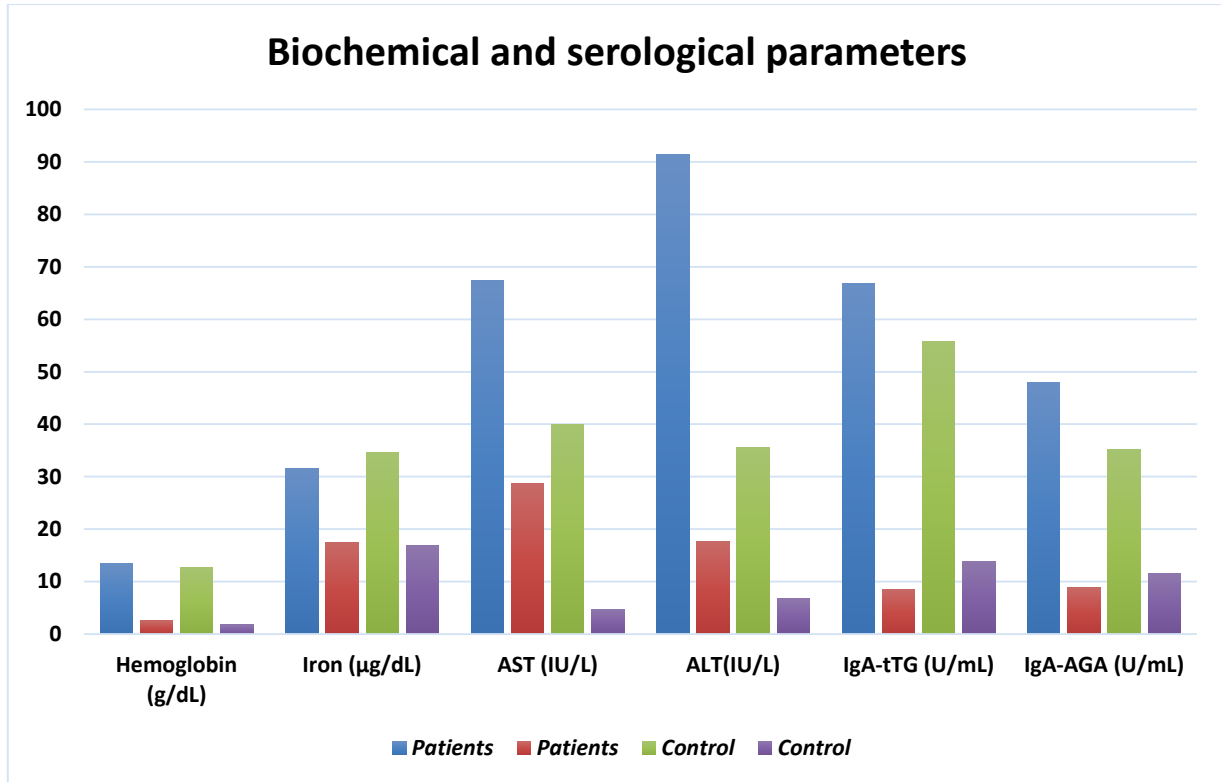


Figure 1: Biochemical and serological parameters of *H. pylori* in Iraqi children with Celiac disease

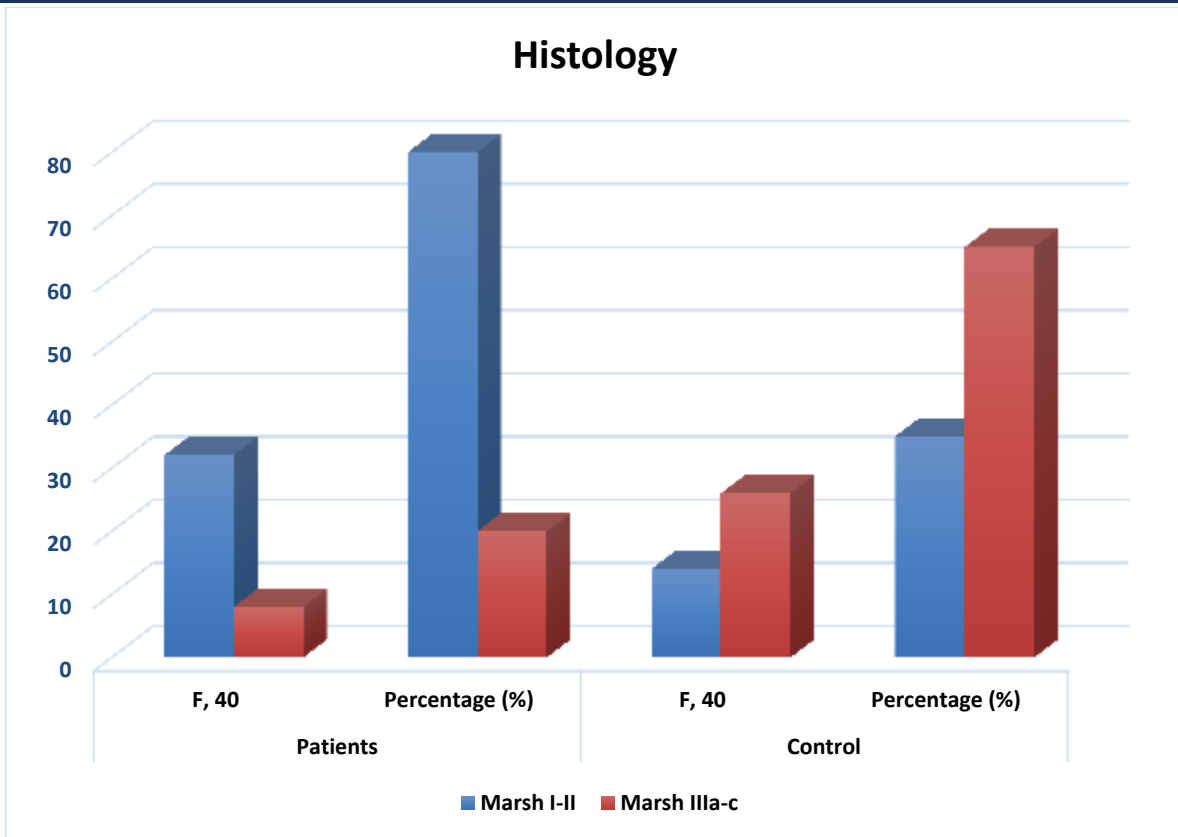


Figure 2: Comparison between patients' group and control group throughout Histology of H. pylori in Iraqi children with Celiac disease

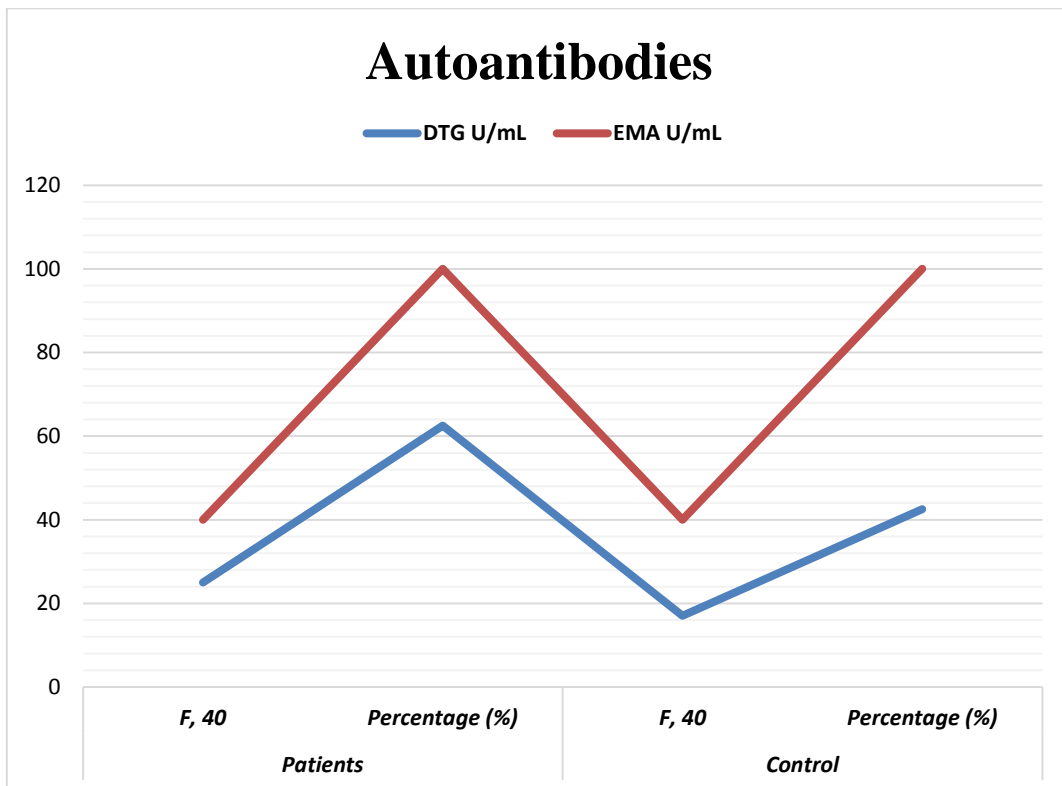


Figure 3: Distribution of serological and histopathological data of autoantibodies for H. pylori in Iraqi children with Celiac disease

Table 5: Results parameters with HP+ and HP- of H. pylori in Iraqi children with Celiac disease

Variables	Patients, 40	Control, 40	OR (95% CI)	P-value
Haemoglobin, g/dL, (SD)	8.1 ± 3.61	9.83 ± 3.783	0.72 (0.55-0.89)	0.00368
TTG > 100, No. (%)	20 (50%)	18 (45%)	3.22 (2.81-6.36)	0.00355
Scalloping, No. (%)	8 (20%)	9 (22.5%)	2.37 (1.58-7.91)	0.00364
Gastritis, No. (%)	12 (30%)	15 (37.5%)	7.66 (3.85-16.44)	0.00428

Table 6: Evaluations of outcomes of Iraqi children with Celiac disease based on Histopathological scoring

Variables	Patients (40), +ve	Patients (40) -ve	P-value
Acute inflammation	2.493±0.25	1.61±0.42	0.03321
Chronic inflammation	1.373±0.2253	0.885±0.153	0.0451
Mucosal atrophy	1.279±0.637	1.034±0.54	0.0462
Intestinal metaplasia	4.15±0.465	3.58±0.994	0.0474
Pseudopyloral metaplasia	1.348±0.053	2.445±0.175	0.0415
Infections	4.36±0.4336	4.75±0.075	0.04898

DISCUSSION

A dangerous multi-genetic autoimmune condition with an increasing number of diagnoses is celiac disease (CD). Patients with this chronic condition, their families, and healthcare professionals face challenges and possibilities. Currently, there is no cure other than a strict gluten-free diet. Our results found the incidence for H. pylori infection, along with risk factors among children, that diagnosis of celiac disease (CD) appears to be within the range of information observed in children without CD patients. When compared to children who were H. pylori-negative, children with CD and H. pylori infection had milder types of enteropathies. Additional research is required to define the relevance among H. pylori infection and CD in children and the protective or detrimental effects of H. pylori strains that may account for the inconsistent results too far. The relationship between CD and H. pylori remains controversial; however, some studies have reported that H. pylori infection appears to confer protection against CD.

performed a cross-sectional study among patients undergoing upper endoscopy and found an inverse relationship between these two entities after adjusting for age and socioeconomic factors. Patients with CD had lower rates of H. pylori infection than those with normal duodenal mucosa. In research with 80 adult CD patients who were tested before and after the gluten-free diet, who had CD and H. pylori in comparison with non-infected people, they reported similar findings.

In this study, H. pylori infection prevalence and risk variables in children with a confirmed diagnosis of CD were examined. We discovered that neither gender nor place of residence has an impact on H. pylori colonization in CD children [Volta, U. et al., 2018]. No appreciable differences

were discovered between individuals with CD and H. pylori infection compared to those with CD without H. pylori infection in terms of clinical characteristics, laboratory results, or a family history of CD. In line with prior research, we discovered small, non-significant variations in the blood levels of IgA-tTG in individuals who tested positive of H. pylori in comparison to those who did not.

In contrast to children who tested negative for H. pylori, some of the latter we have had a slight rise of IgA-AGA levels. Regarding histological changes, we discovered that, in comparison to patients who were negative for H. pylori and displayed more severe intestinal damage, including complete villous atrophy, a greater proportion of CD patients had milder forms of enteropathy. These findings were also reported by Villanacci, et al., and Aydogdu, et al. The association between H. pylori prevalence with the severity of histological duodenal characteristics in CD patients was a unique finding of our investigation. Our findings point to a minor tendency among H. pylori-infected individuals for milder duodenal ulcers.

CONCLUSION

In conclusion, the connection between stomach HP infection and celiac disease (CD) is inverse, proving that HP does not constitute a risk factor for CD. Additional research is needed to validate the conflicting evidence on the link between HP infection and celiac disease (CD).

REFERENCES

1. Basyigit, S., Unsal, O., Uzman, M., Sapmaz, F., Dogan, O.C., Kefeli, A., Asilturk, Z., Yeniova, A.O. and Nazligul, Y. "Relationship between Helicobacter pylori infection and

- celiac disease: a cross-sectional study and a brief review of the literature." *Gastroenterology Review/Przegląd Gastroenterologiczny* 12.1 (2017): 49-54.
2. Ivarsson, A., Persson, L.Å., Nyström, L., Ascher, H., Cavell, B., Danielsson, L., Dannaeus, A., Lindberg, T., Lindquist, B., Stenhammar, L. and Hernell, O. "Epidemic of coeliac disease in Swedish children." *Acta paediatrica* 89.2 (2000): 165-171.
 3. Stene, L.C., Honeyman, M.C., Hoffenberg, E.J., Haas, J.E., Sokol, R.J., Emery, L., Taki, I., Norris, J.M., Erlich, H.A., Eisenbarth, G.S. and Rewers, M. "Rotavirus infection frequency and risk of celiac disease autoimmunity in early childhood: a longitudinal study." *Official journal of the American College of Gastroenterology| ACG* 101.10 (2006): 2333-2340.
 4. Mårild, K., Stephansson, O., Montgomery, S., Murray, J.A. and Ludvigsson, J.F. "Pregnancy outcome and risk of celiac disease in offspring: a nationwide case-control study." *Gastroenterology* 142.1 (2012): 39-45.
 5. Lucero, Y., Oyarzún, A. and O'Ryan, M, et al. "Helicobacter pylori cagA+ is associated with milder duodenal histological changes in Chilean celiac patients." *Front Cell Infect Microbiol.* 2017 (2017):427.
 6. Mirbagheri, S.A., Khajavirad, N., Rakhshani, N., Ostovaneh, M.R., Hoseini, S.M.E. and Hoseini, V. "Impact of Helicobacter pylori infection and microscopic duodenal histopathological changes on clinical symptoms of patients with functional dyspepsia." *Digestive diseases and sciences* 57 (2012): 967-972.
 7. Yazdani, R., Azizi, G., Abolhassani, H. and Aghamohammadi, A. "Selective IgA deficiency: epidemiology, pathogenesis, clinical phenotype, diagnosis, prognosis and management." *Scandinavian journal of immunology* 85.1 (2017): 3-12.
 8. McCarty, T.R., O'Brien, C.R., Gremida, A., Ling, C. and Rustagi, T. "Efficacy of duodenal bulb biopsy for diagnosis of celiac disease: a systematic review and meta-analysis." *Endoscopy international open* 6.11 (2018): E1369-E1378.
 9. Suzana, M.K., Skender, T., Emine, D.S., Halil, A., Vjollca, S.M., Agron, K., Sadushe, L., Labinot, S., Goneta, G. and Arijeta, P. "Helicobacter pylori gastritis updated Sydney classification applied in our material." *Sec Biol Med Sci* 30.1 (2009): 45-60.
 10. Sergi, C., Shen, F. and Bouma, G. "Intraepithelial lymphocytes, scores, mimickers and challenges in diagnosing gluten-sensitive enteropathy (celiac disease)." *World Journal of Gastroenterology* 23.4 (2017): 573-589.
 11. Ikeda, Y., Nishikura, K., Watanabe, H., Watanabe, G., Ajioka, Y. and Hatakeyama, K. "Histopathological differences in the development of small intestinal metaplasia between antrum and body of stomach." *Pathology-Research and Practice* 201.7 (2005): 487-496.
 12. Al-Toma, A., Volta, U., Auricchio, R., Castillejo, G., Sanders, D.S., Cellier, C., Mulder, C.J. and Lundin, K.E. "European Society for the Study of Coeliac Disease (ESsCD) guideline for coeliac disease and other gluten-related disorders." *United European gastroenterology journal* 7.5 (2019): 583-613.
 13. Zevit, N. and Shamir, R. "Diagnosis of celiac disease: where are we heading after the ESPGHAN 2012 guidelines?." *Journal of Pediatric Gastroenterology and Nutrition* 59 (2014): S13-S15.
 14. Caio, G., Volta, U., Sapone, A., Leffler, D.A., De Giorgio, R., Catassi, C. and Fasano, A. "Celiac disease: a comprehensive current review." *BMC medicine* 17 (2019): 142.
 15. Kalach, N., Bontems, P. and Raymond, J. "Helicobacter pylori infection in children." *Helicobacter* 22 (2017): e12414.
 16. Aydogdu, S., Cakir, M., Ali Yuksekkaya, H., Tumgor, G., Baran, M., Arikan, C. and Yagci, R.V. "Helicobacter pylori infection in children with celiac disease." *Scandinavian journal of gastroenterology* 43.9 (2008): 1088-1093.
 17. Tosco, A., Salvati, V.M., Auricchio, R., Maglio, M., Borrelli, M., Coruzzo, A., Paparo, F., Boffardi, M., Esposito, A., D'Adamo, G. and Malamisura, B. "Natural history of potential celiac disease in children." *Clinical Gastroenterology and Hepatology* 9.4 (2011): 320-325.
 18. DeGaetani, M., Tennyson, C.A., Lebwohl, B., Lewis, S.K., Daya, H.A., Arguelles-Grande, C., Bhagat, G. and Green, P.H. "Villous atrophy and negative celiac serology: a diagnostic and therapeutic dilemma." *Official journal of the American College of Gastroenterology| ACG* 108.5 (2013): 647-653.
 19. Volta, U., Caio, G., Boschetti, E., Giancola, F., Rhoden, K.J., Ruggeri, E., Paterini, P. and De

-
- Giorgio, R. "Seronegative celiac disease: shedding light on an obscure clinical entity." *Digestive and Liver Disease* 48.9 (2016): 1018-1022.
20. Aziz, I., Peerally, M.F., Barnes, J.H., Kandasamy, V., Whiteley, J.C., Partridge, D., Vergani, P., Cross, S.S., Green, P.H. and Sanders, D.S. "The clinical and phenotypical assessment of seronegative villous atrophy; a prospective UK centre experience evaluating 200 adult cases over a 15-year period (2000–2015)." *Gut* 66.9 (2017): 1563-1572.

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