

Volume: 1 | Issue: 1 | SEP - OCT 2021 | Available Online: www.irespub.com

Helicobacter infections: status update

Ashaolu, J.O.¹ and Ashaolu, T.J.^{2*} ¹International Health Programme, School of Medicine, National Yang-Ming University, Taipei, Taiwan. ²Duy Tan University, Danang, Vietnam

Short communication

ABSTRACT

The prevalent rate of Helicobacter infections among the human population is reducing in the developed world, but constant migration, general globalization, and other environmental factors have kept every population at risk. This puts a question on the seemingly progress made so far in the treatment and eradication of these bacteria and their infections. This communication is aimed at clinical and epidemiological gains and the challenges so far in the treatment and eradication of helicobacter spp. infections.

KEYWORDS

helicobacter spp.; infections; human population; bacteria; eradication

CORRESPONDING AUTHOR

Dr. Tolulope Joshua Ashaolu

INTRODUCTION

Presently, there are about 35 species in the helicobacter genus, of which Helicobacter pylori is the most significant species and an important aetiological factor in a variety of human gastric disorders, such as duodenal ulcer, gastric ulcers, and gastric cancer [1,2]. More than half the world's human population is infected with H. pylori across varied regions. For instance, it was reported that the regions with the highest H. Pylori prevalence were Africa (70.1%), South America (69.4%), and Western Asia (66.6%) while regions with the lowest reported H. Pylori prevalence were Oceania (24.4%), Western Europe (34.3%), and Northern America (37.1%), respectively [3].

At the beginning of this 21st century, H. pylori infection prevalence started to decline in highly industrialized countries of the Western world, whereas prevalence perpetually remains at a high level in developing and newly industrialized countries. The widening differential gap in prevalence has important implications on the future worldwide prevalence of sequelae associated with H. pylori, including peptic ulcer disease and gastric cancer [3]. Furthermore, even though helicobacter infections are often acquired in childhood, a recent meta-analysis reveals that the prevalence of the infection in adults (\geq 18 years) was significantly higher than in children (48.6% vs 32.6%, respectively) [1]. Hitherto, is there actual progress being made in the eradication of the bacteria and its associated outcome?

GASTRIC AND DUODENAL ULCERS AND CANCERS

H. pylori contribute to duodenal ulceration mainly through high levels of gastric acid secretion in the antrum, which leads to the replacement of intestinal-type tissue in the duodenum with gastric tissue. The overall effect culminates in the allowance of H. pylori in colonizing the areas of gastric metaplasia and together with elevated acid levels, ultimately promote ulceration in this locale [1,4].

Clinical data has shown that 95% of duodenal and 70% of gastric ulcers are associated with H. pylori. Incidentally, peptic ulcer disease is the cause of dyspepsia in about 10% of people [5]. There is currently no evidence that H. pylori eradication therapy is an effective treatment in people with gastric ulcers or that it is effective in preventing recurrence of duodenal ulcers compared to ulcer-healing drugs [5]. There is a mix in the multifactorial causes, and therefore, the need for long-term cohort studies and/or studies with enough statistical power to ascertain the role of helicobacter eradication in the etiology of gastric ulcer reoccurrence.

H. pylori are also involved in the initiation and progression of gastric cancer through several mechanisms. For instance, chronic gastric inflammation, progressing to the precancerous changes of atrophic gastritis and intestinal metaplasia has been an identified pathway. The extent and/or severity of these precancerous changes have a direct relationship with the risk of developing gastric cancer [6]. Another mechanism reported through which Chronic H. pylori contributes to gastric mucosal genetic instability is by reducing gastric acid secretion (hypochlorhydria).

This reduction has the potential of promoting the growth of the gastric microbiome that processes dietary components into carcinogens [7]. Gastric cancer is a major global health threat and is the third leading cause of cancer deaths worldwide causing an estimated >720,000 deaths per year globally [8].

Even though it has been suggested that the eradication of H. pylori, using some antibiotics regimen, can result in resolution of gastric inflammation, halt the progression of gastric mucosal damage, prevent further H. pylori-induced DNA damage, improve gastric acid secretion, and restore the microbiome toward normal [7], the result has been conflicting with uncertainty in its generalization across the globe, especially across the high-risk groups including the low-incidence populations [9]. Moreover, the best of such antibiotic therapeutic applications, from global clinical data, has resulted in only a significant reduction (54%) in the incidence of gastric cancer. It has been speculated that without effective preventive measures, the current high incidence of gastric cancer will remain stable or even increase by 2030 [8]. Consequently, the International Agency for Research on Cancer (IARC) has indicated the urgent need for effective preventive measures and for a critical assessment of H. pylori eradication as a preventive strategy [10].

IS ERADICATION POSSIBLE?

Challenges associated with helicobacter infections include antibiotic resistance [11], reoccurrence of infection [12], behavioral and lifestyle of individuals such as observance of standard hygiene and sanitary practices [12], careful interaction with pet (for pet owners) and other animals in order to avoid zoonotic transmission of the bacteria [13]. All these factors have to be appropriately dealt with in order to ensure adequate protection in the human population.

It seems the eradication of helicobacter infections will take longer than anticipated. Special attention however needs to be given to developing and underdeveloped nations as they may serve as the rate-determining step in the eradication of these long-term human infectious agents.

REFERENCES

- [1] Ashaolu, J.O (2020). Is the human race winning the war against Helicobacter Infections? International Journal of Scientific Advances (IJSCIA), Volume 1| Issue 1: Jul-Aug 2020, Pages 7-9
- [2] Sanders, M. K., & Peura, D. A. (2002). Helicobacter pylori-associated diseases. Current gastroenterology reports, 4(6), 448-454.
- [3] Hooi, J. K., Lai, W. Y., Ng, W. K., Suen, M. M., Underwood, F. E., Tanyingoh, D., ... & Chan, F. K. (2017). Global prevalence of Helicobacter pylori infection: systematic review and metaanalysis. Gastroenterology, 153(2), 420-429. doi: 10.1053/j.gastro.2017.04.022
- [4] Varga, M. G., & Epplein, M. (2019). Helicobacter Pylori-Mediated Carcinogenesis. 87-197. doi.org/10.1016/B978-0-12-801238-3.65172-3
- [5] Ford, A. C., Delaney, B., Forman, D., & Moayyedi, P. (2006). Eradication therapy for peptic ulcer disease in Helicobacter pylori positive patients. Cochrane Database of Systematic Reviews, 4(4):CD003840.
- [6] Correa, P. (2004). The biological model of gastric carcinogenesis. IARC scientific publications, (157), 301-310.
- [7] Machado, A. M. D., Figueiredo, C., Touati, E., Maximo, V., Sousa, S., Michel, V., ... & Rasmussen, L. J. (2009). Helicobacter pylori infection induces genetic instability of nuclear and mitochondrial DNA in gastric cells. Clinical Cancer Research, 15(9), 2995-3002.
- [8] IARC Helicobacter pylori Working Group. Helicobacter pylori Eradication as a Strategy for Gastric Cancer Prevention. Lyon, France: International Agency for Research on Cancer (IARC Working Group Reports, No. 8). Available at: http://www.iarc.fr/en/publications/pdfs-online/ wrk/wrk8/index.php. Accessed on November 21, 2015
- [9] Lee, Y. C., Chiang, T. H., Chou, C. K., Tu, Y. K., Liao, W. C., Wu, M. S., & Graham, D. Y. (2016). Association between Helicobacter pylori eradication and gastric cancer incidence: a systematic review and metaanalysis. Gastroenterology, 150(5), 1113-1124. doi: 10.1053/j.gastro.2016.01.028
- [10] Herrero, R., Parsonnet, J., & Greenberg, E. R. (2014). Prevention of gastric cancer. JAMA, 312:1197–119

- [11] Drossman, D. A., & Hasler, W. L. (2016). Rome IV— functional GI disorders: disorders of gut-brain interaction. Gastroenterology, 150(6), 1257-1261.
- [12] Hu, Y., Wan, J. H., Li, X. Y., Zhu, Y., Graham, D. Y., & Lu, N. H. (2017). Systematic review with meta-analysis: the global recurrence rate of Helicobacter pylori. Alimentary pharmacology & therapeutics, 46(9), 773-779. doi:10.1111/apt.14319
- [13] [Mladenova-Hristova, I., Grekova, O., & Patel, A. (2017). Zoonotic potential of Helicobacter spp. Journal of Microbiology, Immunology and Infection, 50(3), 265-269.