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Letters to the Editor

Relationship between *H. pylori* and hepatitis A virus infection

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Dear Editor,

Helicobacter pylori (*H. pylori*) infection is a very common disease, especially in developing countries, affecting 80-90% of the adult population and 30% in the younger population (1-4).

The most accepted mechanism of disease transmission is fecal-oral route. It is also believed that the main way of transmission in countries with low health is the contamination in drinking water and food (5).

In countries or communities with a good social and health level, we must support different transmissions mechanisms, not yet well clarified, from person to person at various levels: domestic, in nurseries or in closed institutions, by oral-oral line (saliva), gastro-oral way (vomits) or fecal-oral. This transmission, from person to person, probably has a very important role in communities or countries with low socio-health level through overcrowding, or sharing bedrooms and bathrooms, etc. (5) The fecal-oral route is the main mechanism of transmission of infection of hepatitis A virus (HAV) (6). The prevalence of this disease has fallen down because of the improvement in sanitary conditions of the population. The prevalence of VHA infection has been considered by some researchers as an indirect marker for risk of infection by fecal-oral contamination of water or food or health level of a population. The impact of fecal-oral transmission in *H. pylori* infection has been valued by some researchers, comparing in certain populations the prevalence of both infections. Fecal-oral transmission would have little value in *H. pylori* infection when the prevalence of this infection is relatively important respect to prevalence of HAV infection, and when any relationship is not detected , in a certain population and in different age groups, between the prevalence of both infections (7-9). In recent reviews of studies comparing the seroprevalence of *H. pylori* and HAV infections, the meaning of these researches is being questioned. The conclusion of this systematic review is that the comparison of seroprevalence of both infections is not a conclusive method of evaluation fecal-oral transmission in the infection by *H. pylori* (10). This review does not include any studies completed in Spain, which motivates this posting to share our experience in this area.

During 2004-2007 we investigated the presence of IgG antibodies against VHA and *H. pylori* in a potentially homogeneous population: young adult, naturals and residents in Western Andalusia (Seville, Huelva and Cádiz) and with a socio-sanitary level allegedly medium-high. We included 220 undergraduates, 163 women and 57 men, with ages between 18 and 23 years (21.3 average age), who join our hospital, to start or continue studies of medicine or nursering. We excluded students from different geographical areas. We investigated simultaneously the presence of antibodies to *H. pylori* (Bioelisa Helicobacter IgG. Biokit) and to HAV (Havab 2.0 Axsym. Abbott). No students had suffered from gastro-duodenal ulcer or HAV infection or had been vaccinated against this virus. It were detected antibodies to *H. pylori* and against HAV in 72 (32.7%) and in 7 (3.2%) students severally. Serology was negative for both infections in 144 cases (65.4%) and positive in 3 cases (1.3%). Most students with positive serology for *H. pylori* (95.8%) showed negative serology for VHA. The seroprevalence of HAV infection was low in both the population infected with *H. pylori* (4.1%) and without *H. pylori* (2.7%) (Table I). We compared prevalence of both infections by McNemar's test, with significant results ($p < 0.0005$), showing that they were very different. The low overall seroprevalence of HAV infection (3.2%) may suggest an appropriate sanitary level in population studied and, and according to some researchers, a low risk of infection by fecal-oral route, including infection by *H. pylori*. In our population, despite an admissible health level, we detected a significant seroprevalence of infection by *H. pylori* (32.7%). We can consider that the improvement in health status of population has been enough to reduce dramatically HAV infection but insufficient to control infection with *H. pylori*.

The existence of a human reservoir of infection, the possibility of reinfection and transmission person to person, especially in domestics areas, should condition the adoption of preventive mea-

Table I. Seroprevalence about infection by *H. pylori* and by hepatitis A virus (HAV) (n = 220)

	VHA (+) (n = 7)	*VHA (-) (n = 213)*
H. pylori (+) n = 72	3 (1.3%)	69 (31.3%)
H .pylori (-) n =148	4 (1.8%)	144 (65.4%)

sures to reduce the prevalence of *H. pylori* infection and the possibility of suffer from peptic ulcer disease, and specially gastric neoplasm. Any country has adopted public health actions related to treatment of infected population or to prevent the infection, at least in population with more risk. While we have not effective vaccines we can consider, as it had been recommended by experts, being more generous in the research or treatment of *H. pylori* infection. In patients with dyspepsia not investigated, who needs aspirin treatments, anti-inflammatory or gastric antisecretory during a long time, as close relatives from infected patients, is recommended to research the infection by *H. pylori* and it must to be treated with evidence of active infection.

Manuel Castro-Fernández, Julio Vargas-Romero, Elena Hoyas, Eloisa Lamas, Raquel Millán and Manuel Romero-Gómez

Unit of Digestives Diseases and CIBERehd. Unit of Microbiology and Diseases. CIBERehd. Hospital Universitario de Valme. Sevilla, Spain

References

1. Martín-de-Argila C, Boixeda D, Cantón R, Mir N, Rafael L, Gisbert J, et al. Helicobacter pylori infection in a healthy population in Spain. Eur J Gastroenterol Hepatol 1996;8:1165-8.
2. Rodrigo Sáez L, Riestra Menéndez S, Fernández Rodríguez E, Fernández Velázquez MR, García Alonso S, Lauret Braña ME. Estudio epidemiológico de la prevalencia de la infección por Helicobacter pylori en población general en Asturias. Rev Esp Enf Digest 1997; 89:511-6.
3. Baena Díez, García Lareo M, Martí Fernández J, León Marín I, Muñiz Llama D, Teruel Gila J, et al. Prevalencia de la infección por Helicobacter pylori en atención primaria: estudio epidemiológico. Atención Primaria 2002;29:553-7.
4. Sanz JC, Fernández M, Sagues MJ, Ramírez R, García-Comas L, López-Brea M. Seroprevalencia dependiente de la edad frente a Helicobacter pylori en niños y adolescentes de la Comunidad de Madrid. Enferm Infecc Microbiol Clin 2000;18:147-8.
5. Vilaichone RK, Mahachai V, Graham DY. Helicobacter pylori Diagnosis and Management. Gastroenterol Clin North America 2006;35:229-47.
6. Bruguera M, Vidal J, Rodés J. Factores de riesgo en la hepatitis A de los adultos. Gastroenterol Hepatol 1992;15:129-33.
7. Webb Pm, Knight T, Newell DG, Elder JB, Forman D. Helicobacter pylori transmission: evidence from a comparison with hepatitis A virus. Eur J Gastroenterol Hepatol 1996;8:439-41.
8. Luzza F, Imeneo M, Maletta M, Paluccio G, Giancotti A, Perticone F, et al. Seroepidemioloy of Helicobacter pylori infection and hepatitis A in a rural area:evidence against a common mode of transmission. Gut 1997;41:164-8.
9. Fujisawa T, Kumagai T, Akamatsu T, Kiyosawa K, Matsunaga Y. Changes in seroepidemiological pattern of Helicobacter pylori and hepatitis A virus over the last 20 years in Japan. Am J Gastroenterol 1999;94: 2094-9.
10. BinSaeed AA. Is There a link between seropositivity to Helicobacter pylori and hepatitis A virus? A systematic revision. Intern J Infect Dis 2010;14: e567-e571.