## LETTERS

patients (but we did observe lesions on the necks of some patients).

Our data coincided with those of the French study and reinforce the specificity of this dermatologic sign. However, this was not the only coincidence; cases also occurred among the investigators after contact with the infected material in each of the outbreaks. Perhaps both signs may characteristic this dermatitis: the comet sign and "the sign of the infected investigators" of the outbreaks.

## Juan B. Bellido-Blasco, Alberto Arnedo-Pena, and Francisca Valcuende

Author affiliations: Centro de Salud Pública—Epidemiología, Castellón, Spain (J.B. Bellido-Blasco, A. Arnedo-Pena); and Hospital de la Plana (Castellon)—Dermatología, Castellón (F. Valcuende)

#### DOI: 10.3201/eid1503.081461

### References

- Del Giudice P, Blanc-Amrane V, Bahadoran P, Caumes E, Marty P, Lazar M, et al. *Pyemotes ventricosus* dermatitis, southeastern France. Emerg Infect Dis. 2008;14:1759–61. DOI: 10.3201/ eid1411.080288
- Bellido-Blasco JB, Arnedo-Pena A, González-Morán F, Ripollés-Molés JL, Pac-Sa MR, Chiva-Nebot F. Dermatitis outbreaks caused by *Pyemotes*. Med Clin (Barc). 2000;114:294–6.

Address for correspondence: Juan B. Bellido-Blasco, Centro de Salud Pública— Epidemiología, Castellón, Spain; email: bellido\_jua@gva.es

The opinions expressed by authors contributing to this journal do not necessarily reflect the opinions of the Centers for Disease Control and Prevention or the institutions with which the authors are affiliated.

# Helicobacter pylori Infection in Patients Undergoing Upper Endoscopy, Republic of Georgia

To the Editor: Helicobacter py*lori* infection is the principal cause of chronic active gastritis and peptic ulcer disease and a major contributor for gastric adenocarcinoma and mucosa-associated lymphoid tissue lymphoma (1). Approximately 50% of the world's population is infected (2), but only 10%-20% of infected persons become symptomatic (3). The annual incidence rate of H. pylori infection is  $\approx 4\% - 15\%$  in developing countries, compared with  $\approx 0.5\%$  in industrialized countries (4). Studies in the Republic of Georgia (ROG), a developing country with an economy in transition, suggested that >70% of adults are infected with H. pylori (5,6) and that the prevalence rate of gastric cancer is 18 cases per 100,000 population, ≈6- to 9-fold higher than in the United States (National Center for Disease Control, Tbilisi, ROG, unpub. data, 2003). We investigated the prevalence of infection in patients in ROG in whom gastritis, peptic ulcer, and gastric cancer had been diagnosed.

We performed a cross-sectional study of patients referred for upper endoscopy from all regions of ROG to 23 tertiary-care medical centers in the capital, Tbilisi, during 2003–2005. Patients whose medical records and gastric biopsy slides were available were eligible for inclusion. Two pathologists reviewed hematoxylin and eosin-stained slides prepared from formalin-fixed. paraffin-embedded gastric biopsy specimens. Pathologists graded the amounts of H. pylori, acute and chronic inflammation, intestinal metaplasia, and atrophy according to the visual analogue scale of the Updated Sydney Classification System for Gastritis (7). Histologic characteristics were dichotomized as presence (grades  $\geq 1$ ) or absence (grade = 0) of a feature.

We conducted statistical analyses in SAS version 9.0 (SAS Institute, Inc., Cary, NC, USA). The human subjects committees at the National Center for Disease Control and Medical Statistics of ROG and the Centers for Disease Control and Prevention (Atlanta, GA, USA) approved the study.

We identified 90 eligible persons. Their median age was 62 years (range 6–81 years); 48 (54%) were male. Biopsy specimens were taken from the antrum in 89 (99%) persons and from the corpus in 1 person. *H. pylori* infection was diagnosed in 59 (72%) persons, acute inflammation in 81 (90%), chronic inflammation in 77 (87%), metaplasia in 29 (35%), and atrophy in 11 (16%). *H. pylori* was detected in 78% of patients who had gastritis, in 58% of patients who had peptic ulcer, and in 58% of patients who had dysplasia or gastric cancer (Table).

In a multivariable Poisson regression model, *H. pylori* positivity was strongly associated with acute inflammation (adjusted prevalence ratio [aPR] 1.4, 95% confidence interval [CI] 1.2–1.8) and chronic inflammation (aPR 1.5, 95% CI 1.2–1.9). Age  $\geq$ 50 years (aPR 0.9, 95% CI 0.8–1.2) and male sex (aPR 1.0, 95% CI 0.9– 1.2) did not confer increased risk for *H. pylori* infection.

*H. pylori* requires gastric mucus for growth, and mucus produced by the metaplastic and neoplastic cells is postulated to lack characteristics that sustain growth of *H. pylori*. When *H. pylori* has been observed in patients with ulcers, intestinal metaplasia, and adenocarcinoma, the bacteria usually are present in areas of the stomach that do not have these lesions.

In this cohort of patients, 14 (16%) had dysplasia or adenocarcinoma. Dysplasia and eventually cancer occur in a small group of susceptible persons with atrophy and intestinal metaplasia (8,9). Thus, *H. pylori* is now considered a type-1 carcinogen, and the ab-

Table. Histopathologic characteristics assessed in the biopsy specimens of study participants, by final pathologic diagnosis, Republic of Georgia, 2003–2005

	No. Heliobacter pylori positive/no.	No. with acute inflammation/no.	No. with chronic inflammation/no.	No. with intestinal metaplasia/no.	No. with glandular atrophy/no. tested
Final pathologic diagnosis	tested (%), n = 82	tested (%), n = 90	tested (%), n = 89	tested (%), n = 84	(%), n = 68
Gastritis* (n = 62)	45/58 (78)	55/62 (89)	55/62 (89)	19/60 (32)	10/50 (20)
Peptic ulcer (n = 14)	7/12 (58)	14/14 (100)	12/13 (92)	5/13 (38)	1/10 (10)
Dysplasia or cancer (n = 14)	7/12 (58)	12/14 (86)	10/14 (71)	5/11 (45)	0/8 (0)
Total (n = 90)	59/82 (72)	81/90 (90)	77/89 (87)	29/84 (35)	11/68 (16)
*According to the Updated Sydney Classification System for Gastritis (7).					

sence of infection is predicted to result in at least 60% fewer cases of gastric cancer worldwide (10).

Our study has several limitations. First, because of the study's retrospective nature, pathologists reviewed archived hematoxylin and eosin-stained sections of biopsy specimens. The inability to use additional techniques (e.g., special staining, immunohistochemistry) commonly used to visualize H. pylori might have resulted in underdetection of the true rate of infection in the study population. Second, in 99% of cases, only 1 biopsy specimen was obtained from the antrum instead of the 5 antrum and corpus biopsy specimens recommended for appropriate diagnosis by the Updated Sydney Classification System (7). Third, the relatively small sample size precluded exploration of additional relationships between demographic characteristics and histologic grades.

Our results indicate a high prevalence of *H. pylori* in patients with gastritis, peptic ulcer, and gastric cancer and suggest that *H. pylori* infection represents a serious public health problem in ROG. Studies are needed to explore demographic, socioeconomic, and behavioral risk factors that contribute to the high prevalence of *H. pylori* infection in symptomatic and asymptomatic persons living in ROG so that preventive measures can be identified.

This study was supported by the US Department of Health and Human Services' Biotechnology Engagement Program no. G-1195. The funding source played no role in designing the study; collecting, analyzing, or interpreting the data; writing the report; or deciding to submit it for publication.

Nato Tarkhashvili, Rusudan Beriashvili, Neli Chakvetadze, Maia Moistsrapishvili, Maia Chokheli, Merab Sikharulidze, Lile Malania, Nato Abazashvili, Ekaterine Jhorjholiani, Marina Chubinidze, Nanuli Ninashvili, Tamar Zardiashvili, Ucha Gabunia, Dimitri Kordzaia, Paata Imnadze, Jeremy Sobel, and Jeannette Guarner

Author affiliations: Centers for Disease Control and Prevention, Atlanta, Georgia, USA (N. Tarkhashvili, J. Sobel); National Center for Disease Control and Medical Statistics, Tbilisi, Republic of Georgia (R. Beriashvili, N. Chakvetadze, M. Moistsrapishvili, M. Chokheli, M. Sikharulidze, L. Malania, N. Abazashvili, E. Jhorjholiani, M. Chubinidze, N. Ninashvili, T. Zardiashvili, U. Gabunia, D. Kordzaia, P. Imnadze); Emory University School of Medicine, Atlanta (J. Guarner); and Children's Healthcare of Atlanta, Atlanta (J. Guarner)

DOI: 10.3201/eid1503.080850

#### References

- Suerbaum S, Michetti P. Helicobacter pylori infections. N Engl J Med. 2002;347:1175–86. DOI: 10.1056/NEJMra020542
- Lacy BE, Rosemore J. *Helicobacter py-lori*: ulcers and more: the beginning of an era. J Nutr. 2001;131:2789S–93S.

- Makola D, Peura D, Crowe S. *Helicobacter pylori* infection and related gastrointestinal diseases. J Clin Gastroenterol. 2007;41:548–58. DOI: 10.1097/ MCG.0b013e318030e3c3
- Duck WM, Sobel J, Pruckler JM, Song Q, Swerdlow D, Friedman C, et al. Antimicrobial resistance incidence and risk factors among *Helicobacter pylori*–infected persons, United States. Emerg Infect Dis. 2004;10:1088–94.
- Kretsinger K, Sobel J, Tarkhashvili N, Chakvetadze N, Moistrafishvili M, Sikharulidze M, et al. *Helicobacter pylori*, Republic of Georgia. Emerg Infect Dis. 2005;11:780–1.
- Olivares A, Buadze M, Kutubidze T, Lobjanidze M, Labauri L, Kutubidze R, et al. Prevalence of *Helicobacter pylori* in Georgian patients with dyspepsia. Helicobacter. 2006;11:81–5. DOI: 10.1111/ j.1523-5378.2006.00367.x
- Dixon MF, Genta RM, Yardley JH, Correa P. Classification and grading of gastritis: the updated Sydney system. Am J Surg Pathol. 1996;20:1161–81. DOI: 10.1097/00000478-199610000-00001
- Muñoz N, Kato I, Peraza S, Lopez G, Carrillo E, Ramirez H, et al. Prevalence of precancerous lesions of the stomach in Venezuela. Cancer Epidemiol Biomarkers Prev. 1996;5:41–6.
- Correa P, Haenszel W, Cuello C, Zavala D, Fontham E, Zarama G, et al. Gastric precancerous process in a high risk population: cross-sectional studies. Cancer Res. 1990;50:4731–6.
- International Agency for Research on Cancer. *Helicobacter pylori*. In: IARC monograph on the evaluation of carcinogenic risks to humans. Vol. 61. Schistosomes, liver flukes and *Helicobacter pylori*. Lyon (France): International Agency for Research on Cancer; 1994. p. 177–240.

Address for correspondence: Nato Tarkhashvili, Centers for Disease Control and Prevention, 615 E 4th St, Pierre, SD 57501, USA; email: bwi2@cdc.gov