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Comet Sign (and Other) in *Pyemotes* Dermatitis

To the Editor: Recently, Pascal Del Giudice et al. published an interesting article (*1*) about dermatitis in France caused by *Pyemotes ventricosus* in which they highlight the presence of the comet sign in a number of their patients. It is, they assert, a sign that because of its peculiarity could be useful for diagnosing this type of dermatitis in outbreaks and sporadic cases.

Some years ago, we studied 3 outbreaks (with >100 cases) of dermatitis caused by *P. ventricosus* parasitic mites in Castellón, Spain, produced by different infected materials (*2*). When we published the results, we concentrated on the epidemiologic characteristics and the discovery of the mite; perhaps we paid too little attention to the appearance of the lesions, of which we did not provide images. Nevertheless, we also observed the descriptions by Del Giudice et al., which we now show in the Figure. In 2 patients (Figure, panels A and B), the comet sign can be clearly assessed; the patients were 2 women who had had direct contact with the infected material against their legs. The other patient (Figure, panel C) displayed 56 macules with 1 pruritic central vesicle. We did not observe facial lesions on any of the case-

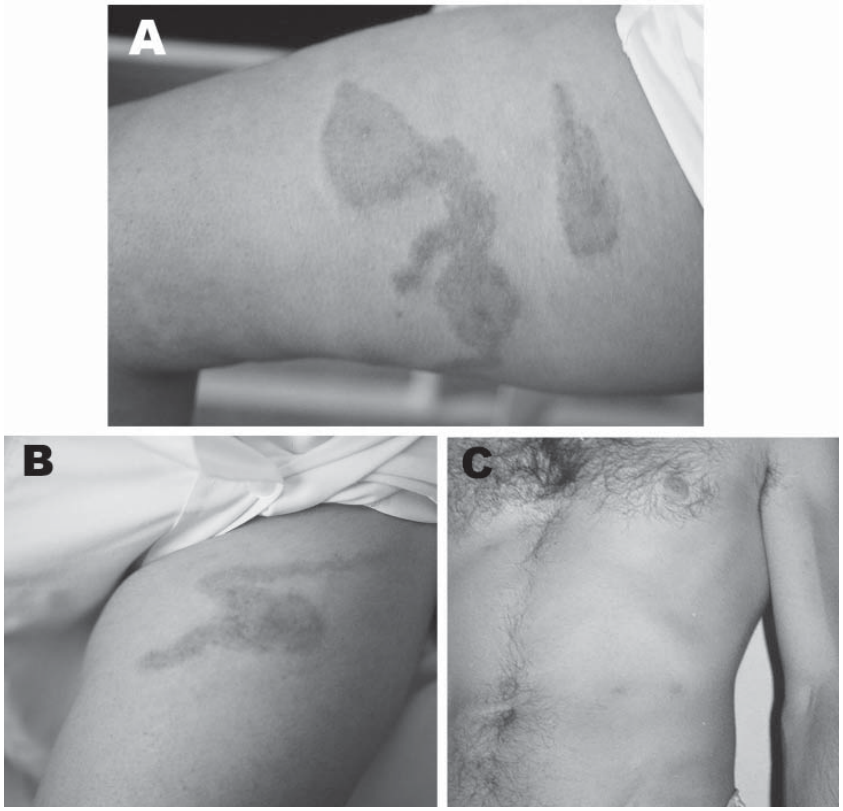

Figure. Photographs of 3 persons with skin lesions of *Pyemotes* dermatitis during the same outbreak in Castellón, Spain, showing the comet sign in 2 affected women (panels A, B), and macular form of the lesions in 1 of the affected investigators (panel C).

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.

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

To the Editor: *Helicobacter pylori* 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 ≈4%–15% in developing countries, compared with ≈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 ≥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 ≥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-