The authors’ aim in this study was to explore the prevalence, symptomatology, and risk factors for peptic ulcer in a general adult population. Between December 1998 and June 2001, the authors surveyed a random sample (n = 3,000) of the adult population (n = 21,610) in two communities in northern Sweden using a validated questionnaire, the Abdominal Symptom Questionnaire (response rate = 74%). A subsample (n = 1,001) of the responders was randomly invited to undergo esophagogastroduodenoscopy and symptom assessment (response rate = 73%). The prevalence of peptic ulcer was 4.1% (20 gastric ulcers and 21 duodenal ulcers). Nausea and gastro-esophageal reflux were significant predictors of peptic ulcer disease, but epigastric pain/discomfort was not. Six persons with gastric ulcer and two persons with duodenal ulcer were asymptomatic. Eight subjects with duodenal ulcer (38%) lacked evidence of current *Helicobacter pylori* infection. Five (25%) of the gastric ulcers and four (19%) of the duodenal ulcers were idiopathic (no use of aspirin or nonsteroidal antiinflammatory drugs, no *H. pylori* infection). Smoking, aspirin use, and obesity were risk factors for gastric ulcer; smoking, low-dose (<160 mg) aspirin use, and *H. pylori* infection were risk factors for duodenal ulcer. Peptic ulcer disease often coexists with atypical symptoms or no symptoms at all, and idiopathic duodenal ulcer may be more common than anticipated.

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**Abbreviations:** CI, confidence interval; NSAID(s), nonsteroidal antiinflammatory drug(s); OR, odds ratio; PUD, peptic ulcer disease.

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In cross-sectional, population-based studies, up to every third adult in the Western world reports dyspepsia (1–4). The prevalence of peptic ulcer among patients with upper gastrointestinal symptoms is reported to be 10–20 percent (5), but the prevalence in the general population is unknown. The probability of finding peptic ulcer disease (PUD) or any organic cause as an
explanation for these symptoms is higher in secondary-care patients, who are usually referred for treatment, than in primary-care patients (6). In two Scandinavian surveys of primary-care patients with dyspepsia (7, 8), 13 percent were found to have PUD, while 43 percent and 64 percent, respectively, had no obvious explanation and were considered to have functional dyspepsia. In an Italian study of secondary-care patients, the prevalence of PUD was 31 percent, and only 21 percent of the patients were considered to have functional dyspepsia (9); the corresponding figures in a US survey were 12 percent and 20 percent, respectively (10). The prevalence of PUD in the general population cannot be reliably estimated from studies of patients, because health-care-seeking behavior is driven by factors other than symptoms (2, 11–15).

The development of idiopathic ulcers (i.e., ulcers not caused by Helicobacter pylori infection, nonsteroidal anti-inflammatory drugs (NSAIDs), or aspirin) is an increasing problem (16). However, their prevalence in the general population is unknown.

Our aim in this study was to investigate the prevalence of PUD, including idiopathic ulcers, and concomitant symptoms and risk factors in a randomly selected adult population.

MATERIALS AND METHODS

Setting, sampling, and study design

The study setting consisted of two communities in northern Sweden with a total of 28,988 inhabitants (17). The

FIGURE 1. Design of a randomized, population-based endoscopic study of peptic ulcer disease (the Kalixanda Study), Kalix and Haparanda, Sweden, December 1998–June 2001. ID, identification number; ASQ, Abdominal Symptom Questionnaire; EGD, esophagogastroduodenoscopy.
esophagogastroduodenoscopy visit, and blood samples for *H. pylori* serologic analysis and measurement of gastrin-17 and pepsinogen-1 levels were taken (17).

The study protocol was approved by the Umeå University ethics committee, and the study was conducted in accordance with the Declaration of Helsinki.

**Endoscopy**

Endoscopy was performed by three experienced endoscopists who participated in regular quality assessment programs. Internal validity was assessed by means of consensus sessions (17, 20). The three endoscopists were unaware of the subjects’ symptoms before and during endoscopy. The endoscopy findings were recorded, and biopsies were taken from the cardia, the corpus, the angulus (except for the first 200 subjects), and the antrum for histologic analysis. In addition, biopsies were taken from the antrum and the corpus for *H. pylori* culture. Any visible lesions were also biopsied (17).

**Questionnaire**

The Abdominal Symptom Questionnaire has been validated (11, 21, 22). The participants indicated (yes/no) whether they had been troubled by any of the listed gastrointestinal symptoms (n = 27) or by any of 11 listed descriptors of abdominal pain or discomfort. Symptom frequency (daily, weekly, or the past 3 months) was also recorded, as was the participants’ medication use in the previous 3 months (17).

**Definitions**

**Symptom groups.** “Gastroesophageal reflux symptoms” were defined as troublesome heartburn and/or acid regurgitation over the past 3 months (23).

“Dyspepsia” was defined as troublesome pain or discomfort, expressed as one or more of the 11 listed types of pain or discomfort, in the epigastric part of the abdomen, or reporting of one or more of the symptoms “uncomfortable feeling of fullness,” “early satiety,” or “nausea,” in accordance with the Rome II definition of dyspepsia (24). “Upper abdominal bloating” was not recorded. A simple definition of dyspepsia labeled “epigastric pain or discomfort,” based on the Rome I definition of dyspepsia, was also used (25).

“Abdominal pain” was defined as troublesome pain or discomfort in the abdomen. It was expressed as one or more of the 11 listed types of pain or discomfort anywhere in the abdomen.

“Irritable bowel syndrome” was defined as one or more of the 11 listed types of abdominal pain or discomfort at any site, combined with reported disturbances in bowel habits. This definition has been shown to have good diagnostic agreement with both the Manning criteria and the Rome I criteria (26).

“Atypical PUD symptoms” were defined as other gastrointestinal symptoms, except dyspepsia or “epigastric pain or discomfort” concomitant with PUD.

“No symptoms or minor symptoms” were defined as individual symptoms not fulfilling any of the above symptom classifications, or an absence of symptoms.

The above definitions allowed concomitant reporting of symptoms of gastroesophageal reflux, dyspepsia, and irritable bowel syndrome.

**Gastric and duodenal ulcers.** Ulcer was defined as a mucosal break at least 3 mm in diameter, with or without a necrotic base in the middle of the lesion, in either the stomach (gastric) or the duodenum (duodenal). In the case of several ulcers/erosions, at least one had to fulfill this definition.

**Histology and *H. pylori* infection**

Biopsy samples were stained with hematoxylin and eosin. *H. pylori* infection was histologically detected by means of Warthin-Starry silver staining (27). The histologic parameters of the gastric mucosa were assessed using the updated Sydney System score definitions (28). Gastritis, including features of former *H. pylori* (minimal chronic inactive or ex- *H. pylori*) gastritis, was diagnosed according to the method of Oberhuber et al. (29). Chemical-reactive gastritis proposed to be caused by aspirin, NSAIDs, or bile reflux was defined according to the updated Sydney System definitions (28, 30, 31).

Two experienced pathologists (M. V. and M. S.) evaluated the biopsies and gave a common report, and then a third experienced pathologist (Dr. M. Walker, Imperial College London, London, United Kingdom) reevaluated the biopsies from 100 randomly chosen subjects. The kappa value for agreement between observers in the evaluation of *H. pylori* infection was 0.76 (95 percent confidence interval (CI): 0.56, 0.96) for the corpus and 0.78 (95 percent CI: 0.59, 0.98) for the antrum. The corresponding figures for granulocyte infiltration were 0.57 (95 percent CI: 0.37, 0.76) and 0.73 (95 percent CI: 0.53, 0.93), respectively.

Samples taken from the antrum and corpus were cultured and analyzed as described previously (27, 32).

Current *H. pylori* infection was defined as a positive culture or histologic finding. There was overall agreement of 99.3 percent, with a kappa value of 0.96 (95 percent CI: 0.94, 0.98) for agreement between the tests (27).

**Serology**

The presence of *H. pylori* immunoglobulin G antibodies was determined by enzyme immunoassay (Pyloriset EIA-G; Orion Diagnostica, Espoo, Finland) (33). A positive test in the absence of *H. pylori* detection by culture or histology was considered indicative of past infection.

Levels of gastrin-17 (cutoff, <10 pmol/liter) and pepsinogen-1 (cutoff, <25 μg/liter) were analyzed using specific enzyme immunoassays (Biohit Plc, Helsinki, Finland).

**Covariates**

**Use of aspirin and NSAIDs.** All participants were thoroughly interviewed face to face regarding their medication use. Reported use of aspirin or NSAIDs for all subjects with idiopathic ulcers was rechecked by means of a telephone interview and a review of the subjects’ medical records.

**Body mass index.** Body mass index (weight (kg)/height (m)²) was calculated and categorized according to World Health Organization recommendations (34).
Statistical analysis

The significance of age and gender in the prevalence of both individual symptoms and combined symptoms (gastroesophageal reflux symptoms, “epigastric pain or discomfort,” dyspepsia, abdominal pain, and irritable bowel syndrome) was tested by applying a logistic regression model. The significance of individual symptoms and combined symptoms, *H. pylori*, use of acid-reducing drugs (antacids, histamine-2 receptor antagonists, and proton pump inhibitors), obesity, use of NSAIDs, use of aspirin, and smoking in the risk of PUD was analyzed by applying a multivariate logistic regression model adjusting for age and gender. The results are presented as odds ratios with 95 percent confidence intervals. The goodness of fit of the models was judged from the Pearson χ² test. The fit of the model was considered acceptable if the p value was ≥0.05. Fisher’s exact test was applied in appropriate cases. A two-sided p value less than 0.05 was regarded as statistically significant, and 95 percent confidence intervals were computed using a logistic regression model. The Intercooled Stata 8 program was used for the analyses (35).

RESULTS

Original study population

The mean age in the original study population was 50.4 years, and 1,560 participants (52.0 percent) were men (as compared with 50.0 years and 49.7 percent in the corresponding Swedish population).

Esophagogastroduodenoscopy

Altogether, 1,001 subjects had an esophagogastroduodenoscopy performed. These subjects were slightly older than the original study population (age 54.1 years vs. 50.4 years; p < 0.001); 488 (48.8 percent) were men.

Peptic ulcer disease

Twenty subjects (2.0 percent, 95 percent CI: 1.1, 2.9) had gastric ulcer. A single ulcer was found in 12 subjects (60.0 percent); two ulcers were found in two subjects (10.0 percent); and one subject (5.0 percent) had three ulcers, one subject (5.0 percent) had four ulcers, and one subject (5.0 percent) had five ulcers. Three subjects (15.0 percent) had more than five ulcers. Fifteen of the subjects (75.0 percent) had their ulcers located in the prepyloric/antral area, while four (20.0 percent) had ulcers in the middle of the stomach at either the angulus (n = 1) or the curvatura major (n = 3). One subject (5.0 percent) had ulcers in both the fundus and the antrum. The mean age of the subjects with gastric ulcer was 58.1 years.

Duodenal ulcer. Twenty-one subjects (2.1 percent, 95 percent CI: 1.2, 3.0) were found to have duodenal ulcer. Fourteen (66.7 percent) had a single ulcer, five (23.8 percent) had two ulcers, and two (9.5 percent) had three ulcers. The mean age of the subjects with duodenal ulcer was 53.3 years.

No one had both gastric ulcer and duodenal ulcer. Thus, there were 41 subjects (4.1 percent, 95 percent CI: 2.9, 5.3) with PUD. The age and gender distributions of subjects with gastric and duodenal ulcers are shown in figure 2.

Gastric cancer

One 78-year-old woman who did not report alarm symptoms (i.e., difficulties in swallowing, weight loss, early satiety, or blood in the stool) was found to have an adenocarcinoma upon histologic analysis in a benign-appearing gastric ulcer.

Symptoms at endoscopy and their relation to PUD

The 3-month prevalences of the 27 individual symptoms are shown in table 1, and the 3-month prevalences of grouped symptoms (gastroesophageal reflux symptoms, epigastric pain, dyspepsia, overall abdominal pain, and irritable bowel syndrome) are shown in table 2. Thirty-three persons with PUD (80.5 percent) reported symptoms. Nausea was significantly associated with duodenal ulcer and PUD, as were gastroesophageal reflux symptoms and dyspepsia.

Dyspepsia was the only weekly symptom associated with PUD (odds ratio (OR) = 2.16, 95 percent CI: 1.11, 4.19). Daily abdominal pain was associated with duodenal ulcer (OR = 3.96, 95 percent CI: 1.39, 11.29) and with PUD (OR = 3.26, 95 percent CI: 1.49, 7.13).

Eleven subjects (1.1 percent, 95 percent CI: 0.5, 1.7) with PUD—four with gastric ulcer and seven with duodenal ulcer—reported “atypical PUD symptoms” but not dyspepsia or epigastric pain. Eight (72.7 percent) of these persons were aged 50 years or more, and nine (81.8 percent) were women.

The prevalence of asymptomatic PUD was 0.8 percent (95 percent CI: 0.2, 1.4) (six gastric ulcers and two duodenal ulcers).

Risk and protective factors for PUD

Of the 1,001 subjects in the esophagogastroduodenoscopy study, 62 had taken NSAIDs during the past 3 months,
### TABLE 1. Three-month period prevalence (%) of individual gastrointestinal symptoms and their associations with age, gender, and peptic ulcer disease in a randomized, population-based endoscopic study of peptic ulcer disease (the Kalixand Study), Kalix and Haparanda, Sweden, December 1998–June 2001

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Loss of weight</th>
<th>Poor appetite</th>
<th>Uncomfortable feeling of fullness</th>
<th>Difficulties in swallowing</th>
<th>Retching</th>
<th>Acid regurgitation</th>
<th>Early satiety</th>
<th>Nausea</th>
<th>Vomiting</th>
<th>Heartburn</th>
<th>Pain behind the breastbone</th>
<th>Burning feeling rising</th>
<th>Constipation</th>
<th>Diarrhea</th>
<th>Alternating constipation and diarrhea</th>
<th>Feeling of incomplete evacuation</th>
<th>Pain/discomfort upon defecation</th>
<th>Pain/discomfort relieved by defecation</th>
<th>Straining</th>
<th>Urgency</th>
<th>Flatus</th>
<th>Borborymi (gurgling sounds)</th>
<th>Abdominal distension</th>
<th>Urge to defecate during the night</th>
<th>Black stools</th>
<th>Blood stains in stool</th>
<th>Mucus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men (48.8%) Ages 20–49 years (n = 178)</td>
<td>0.0%</td>
<td>1.3%</td>
<td>8.1%</td>
<td>2.8%</td>
<td>6.0%</td>
<td>16.8%</td>
<td>19.6%</td>
<td>21.7%</td>
<td>21.4%</td>
<td>27.0%</td>
<td>16.1%</td>
<td>19.4%</td>
<td>14.9%</td>
<td>15.4%</td>
<td>21.3%</td>
<td>20.4%</td>
<td>11.9%</td>
<td>7.9%</td>
<td>12.0%</td>
<td>17.2%</td>
<td>22.7%</td>
<td>21.8%</td>
<td>19.7%</td>
<td>12.0%</td>
<td>5.5%</td>
<td>7.0%</td>
<td>5.5%</td>
</tr>
<tr>
<td>Men (48.8%) Ages 50–81 years (n = 310)</td>
<td>0.8%</td>
<td>2.9%</td>
<td>13.0%</td>
<td>20.7%</td>
<td>7.7%</td>
<td>28.5%</td>
<td>19.6%</td>
<td>21.7%</td>
<td>21.4%</td>
<td>27.6%</td>
<td>19.4%</td>
<td>19.8%</td>
<td>14.9%</td>
<td>15.4%</td>
<td>19.6%</td>
<td>20.4%</td>
<td>11.9%</td>
<td>7.9%</td>
<td>12.0%</td>
<td>17.2%</td>
<td>22.7%</td>
<td>21.8%</td>
<td>19.7%</td>
<td>12.0%</td>
<td>5.5%</td>
<td>7.0%</td>
<td>5.5%</td>
</tr>
<tr>
<td>Men (48.8%) Ages 20–49 years (n = 488)</td>
<td>4.8%</td>
<td>7.5%</td>
<td>24.3%</td>
<td>20.7%</td>
<td>7.7%</td>
<td>25.7%</td>
<td>19.6%</td>
<td>21.7%</td>
<td>21.4%</td>
<td>27.6%</td>
<td>19.4%</td>
<td>19.8%</td>
<td>14.9%</td>
<td>15.4%</td>
<td>19.6%</td>
<td>20.4%</td>
<td>11.9%</td>
<td>7.9%</td>
<td>12.0%</td>
<td>17.2%</td>
<td>22.7%</td>
<td>21.8%</td>
<td>19.7%</td>
<td>12.0%</td>
<td>5.5%</td>
<td>7.0%</td>
<td>5.5%</td>
</tr>
<tr>
<td>Men (48.8%) Ages 50–81 years (n = 325)</td>
<td>3.5%</td>
<td>4.3%</td>
<td>22.0%</td>
<td>20.7%</td>
<td>7.6%</td>
<td>25.7%</td>
<td>19.6%</td>
<td>21.7%</td>
<td>21.4%</td>
<td>27.6%</td>
<td>19.4%</td>
<td>19.8%</td>
<td>14.9%</td>
<td>15.4%</td>
<td>19.6%</td>
<td>20.4%</td>
<td>11.9%</td>
<td>7.9%</td>
<td>12.0%</td>
<td>17.2%</td>
<td>22.7%</td>
<td>21.8%</td>
<td>19.7%</td>
<td>12.0%</td>
<td>5.5%</td>
<td>7.0%</td>
<td>5.5%</td>
</tr>
<tr>
<td>Total (n = 513)</td>
<td>2.2%</td>
<td>3.6%</td>
<td>17.6%</td>
<td>15.2%</td>
<td>7.6%</td>
<td>25.7%</td>
<td>19.6%</td>
<td>21.7%</td>
<td>21.4%</td>
<td>27.6%</td>
<td>19.4%</td>
<td>19.8%</td>
<td>14.9%</td>
<td>15.4%</td>
<td>19.6%</td>
<td>20.4%</td>
<td>11.9%</td>
<td>7.9%</td>
<td>12.0%</td>
<td>17.2%</td>
<td>22.7%</td>
<td>21.8%</td>
<td>19.7%</td>
<td>12.0%</td>
<td>5.5%</td>
<td>7.0%</td>
<td>5.5%</td>
</tr>
<tr>
<td>p value</td>
<td>NS*</td>
<td>0.008†</td>
<td>&lt;0.001†</td>
<td>&lt;0.001†</td>
<td>&lt;0.001†</td>
<td>&lt;0.001†</td>
<td>&lt;0.001†</td>
<td>&lt;0.001</td>
<td>&lt;0.001†</td>
<td>&lt;0.001†</td>
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<td>&lt;0.001†</td>
<td>&lt;0.001†</td>
<td>&lt;0.001†</td>
<td>&lt;0.001†</td>
<td>&lt;0.001†</td>
<td>&lt;0.001</td>
<td>&lt;0.001†</td>
<td>&lt;0.001†</td>
</tr>
<tr>
<td>95% CI</td>
<td>5.10, 1.18, 24.01</td>
<td>NS NS NS</td>
<td>NS NS</td>
<td>NS NS</td>
<td>NS NS</td>
<td>NS NS</td>
<td>NS NS</td>
<td>NS NS</td>
<td>NS NS</td>
<td>NS NS</td>
<td>NS NS</td>
<td>NS NS</td>
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<td>NS NS</td>
<td>NS NS</td>
<td>NS NS</td>
<td>NS NS</td>
<td>NS NS</td>
</tr>
</tbody>
</table>

* CI, confidence interval; OR, odds ratio; NS, not significant.
† p value from logistic regression analysis (significance level: p < 0.05).
‡ More common in women.
§ A burning feeling rising from the stomach or lower chest towards the neck.
<table>
<thead>
<tr>
<th>Symptom group</th>
<th>Men (48.8%)</th>
<th>Women (51.2%)</th>
<th>All subjects (n = 1,001)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ages 20–49 years</td>
<td>Ages 50–81 years</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>Gastroesophageal reflux symptoms</td>
<td>44.9</td>
<td>32.6</td>
<td>42.7</td>
</tr>
<tr>
<td>Epigastric pain/discomfort</td>
<td>18.5</td>
<td>11.3</td>
<td>13.9</td>
</tr>
<tr>
<td>Dyspepsia</td>
<td>42.2</td>
<td>31.5</td>
<td>36.9</td>
</tr>
<tr>
<td>Irritable bowel syndrome</td>
<td>95% CI: 0.001, 0.001</td>
<td>0.001</td>
<td>NS</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>55.6</td>
<td>38.4</td>
<td>46.7</td>
</tr>
</tbody>
</table>

CI, confidence interval; OR, odds ratio; NS, not significant.

| p value from logistic regression analysis (significance level: p < 0.05). |

More common in women.

H. pylori infection were independent risk factors for gastric ulcer (OR = 3.12, 95 percent CI: 1.13, 8.64, OR = 4.15, 95 percent CI: 1.31, 13.13, and OR = 7.44, 95 percent CI: 2.78, 19.93, respectively). Smoking, overall aspirin intake, and H. pylori infection were independent risk factors for duodenal ulcer (OR = 2.84, 95 percent CI: 1.11, 7.27, OR = 4.28, 95 percent CI: 1.52, 12.10, and OR = 3.56, 95 percent CI: 1.40, 9.09, respectively).

The presence of esophagitis was an independent risk factor for duodenal ulcer (OR = 3.39, 95 percent CI: 1.17, 9.86) and PUD (OR = 3.47, 95 percent CI: 1.57, 7.69).

Low-dose aspirin use was an independent risk factor for both gastric ulcer (OR = 8.88, 95 percent CI: 2.64, 29.88) and duodenal ulcer (OR = 9.38, 95 percent CI: 2.71, 32.46), while standard-dose aspirin use was a risk factor for gastric ulcer only (OR = 4.85, 95 percent CI: 1.25, 18.83). Use of NSAIDs or acetylsalicylic acid did not change the outcome.

One person with gastric ulcer (5.0 percent), a 57-year-old woman, had taken NSAIDs, and eight persons with gastric ulcer (40.0 percent) had taken aspirin. None of the subjects with duodenal ulcer had used NSAIDs; six (28.6 percent) had used aspirin.

Fifty-nine persons who underwent esophagogastroduodenoscopy (5.9 percent) reported former, previously treated PUD (28 gastric ulcers, 21 duodenal ulcers, and 10 with no given localization) before the study started, and 15 of them had received H. pylori eradication therapy. Seven of these 59 subjects had PUD (four gastric ulcers, three duodenal ulcers) in this study, and none had received eradication therapy before.

Idiopathic ulcers

Altogether, five (25.0 percent) of the persons with gastric ulcer and four (19.0 percent) of the persons with duodenal ulcer were found to have no known risk factors (NSAID/aspirin use or H. pylori infection) for PUD, and hence their cases were...
considered idiopathic. The prevalence of idiopathic PUD was 0.9 percent (95 percent CI: 0.3, 1.5), and six of the nine subjects (0.6 percent, 95 percent CI: 0.1, 1.1) did not have histologic signs of former \textit{H. pylori} infection or serologic evidence of former \textit{H. pylori} infection. Five of them had chemical-reactive gastritis in the antrum, and one had normal histology. None of the nine subjects had any antral granulocyte activity, but one of them had the lowest degree of activity in the corpus. Only four of the nine subjects with idiopathic ulcer smoked, and one had an elevated gastrin-17 level (76 pmol/liter) but a low pepsinogen-1 level (7.2 µg/liter), suggesting a low gastric acid output.

There was no significant association between idiopathic PUD and gastroesophageal reflux symptoms, epigastric pain, dyspepsia, irritable bowel syndrome, obesity, or smoking. The only individual symptoms significantly associated with idiopathic PUD were stated weight loss ($p = 0.015$) and loss of appetite ($p = 0.041$) (Fisher’s exact test).

**DISCUSSION**

In this randomly selected population of adults aged 20 years or more, we found a point prevalence of 4 percent for PUD, but the symptomatology did not conform to a classical pattern; that is, “epigastric pain or discomfort” alone did not predict PUD, while nausea and gastroesophageal reflux symptoms did, as did loss of weight. An unexpectedly high proportion of persons with duodenal ulcer were \textit{H. pylori}-negative; 25.0 percent of the gastric ulcers and 19.0 percent of the duodenal ulcers were idiopathic. Continuous use of low-dose aspirin was a risk factor for PUD. In addition, obesity was a risk factor for gastric ulcer. The subjects who underwent endoscopy had a mean age that was approximately 4 years higher than the mean ages of the original adult study population and the Swedish general population. Most of the difference was due to a lower recruitment rate among the youngest quarter of participants (age <35 years) and the fact that symptomatic subjects under 50 years of age were somewhat more willing to respond than asymptomatic people (17). This selection bias might have caused a slight overestimation of PUD prevalence.

The upper age limit of 80 years in this study was decided by the ethical committee because of the risk of complications during esophagogastroduodenoscopy and concerns about obtaining informed consent.

The two study communities, located in northern Sweden, have a slightly lower socioeconomic status than the Swedish average (17–19). Most other relevant studies have shown that socioeconomic differences of similar magnitude do not affect gastrointestinal morbidity (17). \textit{H. pylori} prevalence, another indirect indicator of socioeconomic status, decreases with greater prosperity. The prevalence of \textit{H. pylori} seropositivity in this study was 43.0 percent, and this is comparable with other Northern European countries (36). We do not know of any population-based data from Northern Europe on the prevalence of current infection, which was 34 percent in this study, although available data from Southern Europe show prevalence that is markedly higher (37).

The minimum requirement for symptom reporting in this study was that symptoms should be troublesome. The prevalence of most symptoms was higher than has been reported before in Sweden (22), and it was also higher than in some investigations conducted elsewhere (38–40), although prevalence rates of a similar magnitude have been reported by other investigators (41). Atypical symptoms in PUD patients, especially among the elderly, have also been reported before (42).

There is debate as to whether the concept of dividing dyspepsia symptoms into “ulcer-like” and “dysmotility-like” symptoms is valid (24). The proportion of patients with PUD has been found to be approximately the same in both symptom groups (10, 43), suggesting that those symptom profiles are not useful predictors of PUD. Our study supports the concept that “epigastric pain or discomfort” does not predict PUD, while the dysmotility-like symptom nausea was a weak predictor. We also found, as have other investigators (10), that PUD was common in patients with gastroesophageal reflux symptoms. This suggests that treating all patients with symptomatic gastroesophageal reflux empirically by acid suppression may not represent optimal management. In addition, we found that 34 percent of subjects with unknown PUD were taking acid-reducing drugs.

The high risk of gastric ulcer among obese people has not been described before, to our knowledge. \textit{H. pylori} infection, use of NSAIDs or aspirin, serum gastrin-17 level, and smoking habits did not appear to explain this observation. It remains unknown whether higher acid secretion rates, increased stress, or mechanical factors could explain excess gastric ulcer disease in obese persons.

The reason why some ulcers are asymptomatic is unknown. In controlled trials, both asymptomatic duodenal ulcers and gastric ulcers have been found in a substantial proportion of cases (44, 45). We found that eight subjects (19.5 percent of all ulcer patients and 0.8 percent of the study population) had asymptomatic PUD. Similarly, in the Sørreisa Gastrointestinal Disorder Study, 1 percent of persons who underwent upper endoscopy had asymptomatic PUD (39). The clinical implications of asymptomatic peptic ulcers in our study are uncertain; evaluation of this issue would require a longitudinal natural history study.

To our knowledge, there have been no large-scale studies of PUD in a randomly selected adult population that have had a satisfactory participation rate (46). The Sørreisa Gastrointestinal Disorder Study, from the 1980s, was population-based (47), but a case-control design was applied for the endoscopies. Those investigators found prevalences of PUD of 4 percent among controls and 8 percent among persons with dyspepsia (47). The only other comparable study was performed in Sweden (48) and had a participation rate of 25 percent; 3 percent of those subjects had current PUD, and a further 3 percent had evidence of past ulcers. Few other data are available. Ihämäki et al. (49) found a less than 2 percent prevalence of PUD and a 4 percent prevalence of duodenal scars among healthy controls matched to cancer patients. Khuroo et al. (50) found a point prevalence of 4.7 percent for PUD and a lifetime prevalence of 11.2 percent in a population-based case-control study; most of those subjects had duodenal ulcer. Among monks, Katelaris et al. (51) found a 6 percent prevalence of duodenal ulcer, a 2 percent prevalence of gastric ulcer, and a 7 percent
prevalence of prepyloric or duodenal deformity. Lond et al. (52) found a prevalence of 9 percent for duodenal ulcers and 4 percent for gastric ulcers in a random population sample of persons reporting dyspepsia. In a recent preliminary report from Italy (53), the prevalence of PUD among adults was 4.5 percent, and one third (six of the 18 persons with duodenal ulcer and four of the 12 persons with gastric ulcer) were asymptomatic.

The proportion of H. pylori-negative duodenal ulcers was surprising and worrying, since such results could alter current dyspepsia management algorithms. A rising proportion of idiopathic ulcers among patients has been shown in recent studies (16, 54–56). Lanas et al. (57) have shown that the number of idiopathic ulcers may be overestimated if the participants with PUD underreport aspirin or NSAID use. In our study, aspirin or NSAID use for subjects with idiopathic ulcers was double-checked through an extra telephone interview and a review of those subjects' medical records. In total, use of either aspirin or NSAIDs during the past 3 months was confirmed for 169 subjects (16.9 percent), which is consistent with estimated use for the region (58). A small possibility of underreporting of NSAID use in older women remains, but this is unlikely. H. pylori infection and use of NSAIDs are well-recognized causes of PUD, and some studies have suggested a synergistic effect of these risk factors (59). We did not identify any synergism in the development of PUD. A higher risk of both gastric ulcer and duodenal ulcer with low-dose aspirin use as compared with use of standard-dose aspirin can probably be explained by the continuous use of low-dose aspirin.

Another indicator of aspirin or NSAID use can be antral chemical-reactive gastritis (30), although this can also be caused by bile reflux or excessive alcohol consumption (60). Earlier reports suggested that the sensitivity of this histologic finding for NSAID use is 73 percent (30), but in this study only 32 percent of our 169 subjects with reported use of aspirin or NSAIDs during the previous 3 months had chemical-reactive gastritis. The three cases in which H. pylori was detected by serologic analysis but not by histologic analysis or culture are another potential source of bias. None of those subjects had any granulocyte activity to indicate an ongoing but hidden infection.

We conclude that both gastric ulcer and duodenal ulcer are common in the general population and that persons with ulcer frequently show atypical symptoms. In this general adult population from Sweden, 22 percent of all cases of PUD were idiopathic, and almost 40 percent of duodenal ulcers were H. pylori-negative. Obesity was a risk factor for gastric ulcer, as was use of low-dose aspirin for PUD.

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