Dyspepsia in general practice: incidence, risk factors, comorbidity and mortality

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Background. Many individuals consulting their GP with upper abdominal symptoms are initially classified as having dyspepsia. Few studies have described the incidence of dyspepsia or the comorbidities, risk factors or prognosis associated with this diagnosis.

Methods. We used the UK General Practice Research Database to find patients with a new diagnosis of dyspepsia in 1996 (n = 6913) and a control cohort ($n = 11\ 036$). We determined the incidence of dyspepsia, potential risk factors and comorbidity, and the risk of new onset morbidity in the year following the index date.

Results. The incidence of dyspepsia was 15.3 per 1000 person-years. An increased probability of a dyspepsia diagnosis was associated with chest pain [odds ratio (OR): 2.4], general pain (OR: 1.8), sleep disorders (OR: 1.5), angina (OR: 1.5), osteoarthritis/rheumatoid arthritis (OR: 1.4) and smoking (OR: 1.2). There was only a borderline association with obesity (OR: 1.1). Patients with dyspepsia had an increased likelihood of a diagnosis of irritable bowel syndrome (IBS) (OR: 264), gastroesophageal reflux disease (GERD) (OR: 62.8) or peptic ulcer disease (PUD) (OR: 27.2) during the following year.

Conclusions. The commonest diagnosis to emerge after an initial consultation for dyspepsia was IBS, followed by GERD and PUD.

Keywords. Diagnosis, dyspepsia, gastroesophageal reflux disease, incidence, irritable bowel syndrome.

Introduction

Upper gastrointestinal (GI) symptoms are common in the community, affecting between one quarter and one half of the UK population at least once every 6 months.^{1,2} Approximately 1.5% of the UK population consults a GP for stomach diseases each year, accounting for 2.2% of the GP's workload, and consultation rates are increasing.³ The direct costs of managing dyspepsia total £130 million for endoscopies and over £500 million for prescription, pharmacy-only and over-the-counter medication each year.⁴ These direct costs are likely to be dwarfed by the indirect social costs of impaired work productivity.⁵

Upper GI symptoms are typically described in terms of pain/burning and discomfort in the upper abdomen

and range from heartburn and acid regurgitation to nausea and vomiting. Upper GI symptoms cause problems with sleep, employment, physical and social activities and the consumption of food and drink.⁶ Consequently, they are associated with significant impairment of health-related quality of life.⁷ Potential causes of upper GI symptoms include gastroesophageal reflux disease (GERD), gastroenteritis, gastritis, peptic ulcer disease (PUD), gastric or esophageal cancer, diseases of the gallbladder and pancreas and diabetes mellitus. A number of these can be identified endoscopically, but as many as half of all patients with upper GI symptoms in general practice have no obvious findings to explain their symptoms.⁸

In the UK, the term dyspepsia, meaning 'poor digestion', has for many years been applied to all upper GI

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symptoms.⁹ However, today, both specialists and GPs increasingly subdivide these patients into different diagnoses such as GERD and functional dyspepsia, based on revised definitions of upper GI diseases [Rome III (functional GI disorders); Montreal (GERD) and Maastricht (PUD)]. The consensus criteria for functional dyspepsia developed by an international working group (Rome III) excluded patients whose dominant symptom was heartburn or acid regurgitation, on the basis that they were likely to have GERD.¹⁰ GERD is recognized in the International Classification of Diseases (ICD)¹¹ and covers the range of symptoms and forms of tissue damage secondary to the reflux of gastric contents into the esophagus.¹²

At least in part as a consequence of this terminological confusion, epidemiological studies have produced differing estimates of the incidence and prevalence of dyspepsia, and of its association with potential risk factors and complications.^{4,13–15} Most data on the epidemiology of dyspepsia have come from populationbased cross-sectional studies. Although these have provided much valuable information, they give no information on the temporal relationship between the onset of dyspepsia and potential risk factors and complications. The chronic, relapsing and remitting nature of upper GI symptoms is an additional difficulty. Consequently, the clinical relevance of potential risk factors and complications of dyspepsia is difficult to interpret.

The aim of this study was to determine the incidence of dyspepsia in UK general practice and to identify comorbidities, potential risk factors, subsequent diagnoses and mortality associated with this diagnosis.

Patients and methods

Data source

Data for this study were extracted from the General Practice Research Database (GPRD), one of the world's largest longitudinal general practice databases. It contains information entered by around 1500 GPs covering a population of about three million individuals representative of the UK general population.¹⁶ Participating GPs hold the complete medical records of all individuals registered with them, including demographics, diagnoses, prescriptions and referrals. Prescriptions are automatically produced from the computer and recorded on the patient's computerized file. Diagnoses are coded using Oxford Medical Information Systems (OXMIS) and READ codes that can be mapped to codes from the 8th edition of the ICD (ICD-8). This information is anonymized and sent to the Medicines and Healthcare products Regulatory Agency, which makes it available for use in research projects. A number of studies have validated the accuracy and completeness of the GPRD.^{16,17}

Study population

The selection of patients for the dyspepsia and control cohorts is illustrated in Figure 1. To identify patients with a new diagnosis of dyspepsia in 1996, we first identified a study source population of all individuals aged 20-79 in 1996, who had been registered with the GP for at least 2 years and who had at least one entry in the database during the previous 2 years. We identified those patients within the source population who had a recorded diagnosis of dyspepsia, epigastric pain or indigestion recorded in 1996, using OXMIS dictionary codes (5369C, 5369CD, 5369F, 5369FN, L5369FD, 7855E, 7855ED, 7855DB, 7855EM, 7855ER) and READ dictionary codes (J16y400-412, 1972.00, 195.0, 195Z.00, 1954.00, R090500). We assigned the date of the diagnosis as the index date. To select new cases of dyspepsia, patients with prior dyspepsia, GERD or a related upper GI diagnosis recorded in the 2 years before the index date were excluded. Patients with a prior organic upper or lower GI diagnosis that might explain their symptoms, such as esophageal, pancreatic or bowel disease, were also excluded. Patients with prior cancer, alcoholism or drug dependence recorded within 3 months of the index date, as well as women pregnant during 1996, were excluded. We also excluded patients with long-term (greater than 1 year) use of acid-suppressive drugs before the index date as this could mask prior upper GI conditions not recorded.

An age- and sex-matched comparison cohort without a dyspepsia diagnosis during 1996 was randomly sampled and to ensure an even distribution of followup periods, a random date in 1996 was assigned to each patient as the index date. We applied the same eligibility criteria as were applied to the dyspepsia cohort.

In order to control for consultation rate and make the cohorts more comparable, we restricted the analysis to those patients in the two cohorts who had at least two visits to their GP in the 2 years prior to the index date. The final dyspepsia cohort comprised 6913 patients and the control cohort 11 036. The mean number of consultations per patient in the year prior to the index date was 7.0 (SD: 5.5) for patients in the dyspepsia cohort and 5.3 (SD: 4.2) for patients in the control cohort.

In order to assess time trends in the treatment of patients with dyspepsia, we also sampled a cohort of patients with a new diagnosis of dyspepsia in 1999, following the same procedure for the 1996 cohort. The final 1999 dyspepsia cohort included 4897 patients.

Data collection

For both dyspepsia and control cohorts, information on demographic and lifestyle characteristics [body mass index (BMI), smoking status and alcohol consumption] and comorbidities (diagnoses recorded in the year before the index date) were collected from the computer files. An association between functional

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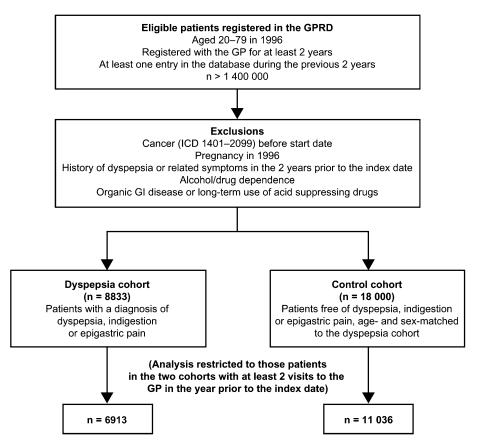


FIGURE 1 Flow diagram showing study design and patient exclusion criteria

GI disorders and pain has been shown in the past,¹⁸ and we included the same pain variable in our study by grouping together the prior pain diagnoses detailed in the footnote to Table 1.

Exposure to the following prescription medications at the index date was determined: non-steroidal antiinflammatory drugs (NSAIDs), aspirin, oral steroids and disease-modifying anti-rheumatic drugs (DMARDs). Current use was defined as a supply of the most recent prescription lasting until the index date or ending in the previous 3 months. Non-use was defined as no prescription recorded before the index date.

Individuals in the 1996 group were followed from their index date until a minimum of 1 year or death. We collected information on deaths and specific morbidities such as GI, respiratory, esophageal and cardiac diseases during the follow-up year. We also recorded the new treatment that patients received during the year after the recorded diagnosis. For this analysis, we excluded patients who had any prescription of these specific drug groups in the year prior to diagnosis.

Analyses

The incidence of dyspepsia was calculated for both sexes and for both the 1996 and 1999 datasets in six 10-year age groups, as the ratio of the number of dyspepsia patients to the total number of patient-years within that group. The distribution of demographic characteristics, current treatment patterns and comorbidity among newly diagnosed dyspepsia patients in 1996 was compared with their distribution in the control group without a dyspepsia diagnosis. Unconditional logistic regression analysis was used to estimate the odds ratios (ORs) and 95% confidence intervals (CIs) for the association of the studied factors with a new diagnosis of dyspepsia, adjusting for age, sex and other potential risk factors.

We also ascertained the risk of development of specific conditions during the 1-year follow-up period, using a separate logistic regression model for each of the studied outcomes, adjusting for other covariables. To be included in each of these analyses, patients had to be free of the corresponding outcome prior to the index date. The risk of death during the follow-up year was estimated by Cox regression analysis.

Results

Incidence

The overall incidence of dyspepsia in 1996 was 15.3 (95% CI: 15.0–15.6) per 1000 person-years (8833 patients per 577 273 person-years). The incidence was greater in women (16.0 per 1000 person-years; 95%

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	Dyspepsia cohort ($n = 6913$)		Control cohor	OR ^a	95% CI	
	n	%	n	%		
BMI (kg/m ²) ^b						
<20	1041	15.1	1650	15.0	1.1	1.0-1.3
20-24.9	1612	23.3	2644	24.0	1.0	_
25-29.9	2053	29.7	3147	28.5	1.1	1.0 - 1.1
30+	1023	14.8	1419	12.9	1.1	1.0 - 1.2
Smoking status ^b						
Non-smoker	3822	55.3	6453	58.5	1.0	_
Smoker	1777	25.7	2348	21.3	1.2	1.2-1.3
Ex-smoker	579	8.4	801	7.3	1.2	1.0-1.3
Alcohol consumption (units p	er week) ^b					
None	2588	37.4	3665	33.2	1.0	_
1–20	2683	38.8	4418	40.0	0.9	0.8-0.9
21–36	200	2.9	347	3.1	0.8	0.6-0.9
37+	109	1.6	143	1.3	1.0	0.7-1.3
Comorbidity in prior year ^c						
General pain ^d	3848	55.7	4190	38.0	1.8	1.7–1.9
Chest pain	542	7.8	325	2.9	2.4	2.1-2.7
Osteoarthritis/RA	537	7.8	558	5.1	1.4	1.2-1.6
Angina	193	2.8	176	1.6	1.5	1.2-1.8
Asthma	368	5.3	472	4.3	1.0	0.9-1.2
COPD	110	1.6	117	1.1	1.1	0.8 - 1.4
Stress	113	1.6	129	1.2	1.2	0.9–1.5
Sleep disorders	249	3.6	215	1.9	1.5	1.2 - 1.8
Pneumonia	28	0.4	35	0.3	0.9	0.6 - 1.6
Cough	764	11.1	926	8.4	1.1	1.0-1.3
Laryngitis/hoarseness	91	1.3	112	1.0	1.2	0.9–1.6
Sinusitis	407	5.9	53	0.5	1.3	1.1-1.5
Otitis	126	1.8	93	0.8	0.9	0.7 - 1.1
Current prescription drug use	e					
NSAIDs	1047	15.1	886	8.0	1.8	1.7-2.0
Aspirin	477	6.9	528	4.8	1.3	1.2-1.5

 TABLE 1 Demographic and lifestyle characteristics, comorbidity and drug use in the dyspepsia and control cohorts and their associations with a new dyspepsia diagnosis

RA, rheumatoid arthritis; COPD, chronic obstructive pulmonary disease.

^aOR estimates are adjusted by age, sex, smoking, alcohol consumption, BMI and prior visits to a GP using logistic regression and are presented with 95% CIs.

^bIn 18.7% of the patients, BMI could not be calculated; in 12.1% of the patients, smoking status was unknown and in 21.1% of the patients, alcohol consumption was not recorded.

^cComorbidity was measured in the year prior to the index date. The reference category for each comorbidity was absence of the diagnosis.

^dGeneral pain includes the following: headache, neuralgia, menstrual and intermenstrual pain, female genital pain, eye, ear, nose, throat or mouth pain, chest pain, pain on respiration, abdominal and rectal pain, urinary pain, penile and testicular pain, skin pain, breast pain, limb pain, muscular and joint pain, neck pain and generalized pain.

^eA prescription lasting until the index date or ending in the previous 3 months.

CI: 15.6–16.5) than men (14.5 per 1000 person-years; 95% CI: 14.1–15.0), and increased with age for both men and women. In 1999, the overall incidence had increased slightly, but insignificantly, to 15.5 (95% CI: 15.1–15.9) per 1000 person-years (4897 patients per 316 004 person-years). In both years, incidence increased with age for women and men (Fig. 2). Overall, half (52%) of the 1996 study population was identified with a recorded code for dyspepsia, 28% with a code for epigastric pain and 20% with a code for indigestion. The distribution of age and sex was different among the different diagnostic code groups. Of those patients identified with a record of epigastric pain, 61% were women. No gender difference was seen in the other diagnostic code groups. We also found that

patients with indigestion were disproportionately old (half of the patients with an indigestion code were over 60 years old). The distribution of ages among the other diagnostic code groups was more homogeneous, which could explain the higher prevalence of comorbidity found among patients with indigestion compared with the other diagnostic code groups (data not shown).

Potential risk factors

Smoking and obesity (BMI $\ge 30 \text{ kg/m}^2$) were associated with a slightly increased risk of dyspepsia, although the association with obesity was of only borderline significance (Table 1). Alcohol consumption did not increase the likelihood of receiving a dyspepsia

diagnosis. A 1-year prior morbidity of chest pain, general pain, angina, sinusitis, osteoarthritis/rheumatoid arthritis or sleep disorders was associated with a newly recorded entry of dyspepsia (Table 1). There was no association of a new diagnosis of dyspepsia with preexisting asthma, chronic obstructive pulmonary disease, cough, otitis, laryngitis, pneumonia or stress.

Current users of NSAIDs or aspirin were at an increased risk of receiving a new diagnosis of dyspepsia (Table 1). There was also an increased risk of dyspepsia among current users of biphosphonates (OR: 2.3; 95% CI: 1.3–4.2) and DMARDs (OR: 1.6; 95% CI: 1.1–2.3). However, very few patients took these drugs (biphosphonates were currently used by 0.4% of the dyspepsia cohort and 0.2% of the control cohort; DMARDs were currently used by 1.1% of the dyspepsia cohort and 0.6% of the control cohort). Among

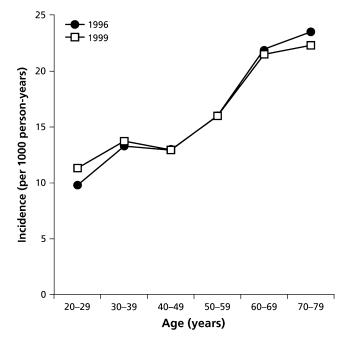


FIGURE 2 The incidence of a new diagnosis of dyspepsia in UK general practice in 1996 and 1999

women, the risk of a new dyspepsia diagnosis was slightly increased among those with a current history of hormone replacement therapy, although this was of only borderline significance (OR: 1.1; 95% CI: 1.0–1.3).

Treatment patterns among patients with dyspepsia in 1996 and 1999

The proportion of patients with dyspepsia who received proton pump inhibitors (PPIs) in the 3 months following their diagnosis was higher in 1999 (25%) than 1996 (15%) (Table 2). There was a decrease in the proportion of patients receiving H₂-receptor antagonists (H₂RAs) and antispasmodics in 1999 compared with 1996 (Table 2). We observed that the majority of patients starting treatment with PPIs after a dyspepsia diagnosis received treatment for less than 1 month (64%). This proportion was similar in the two time periods studied.

Potential complications and mortality

In the follow-up year, patients with dyspepsia had an over 60 times greater risk of receiving a subsequent new diagnosis of GERD than the control cohort (Table 3). There were 152 new cases of irritable bowel syndrome (IBS) in the follow-up year, and all but one were in the dyspepsia cohort, resulting in a very strong association between the recorded diagnosis of dyspepsia and a subsequent diagnosis of IBS (Table 3). Also, patients with dyspepsia more frequently had a new diagnosis of PUD and gallbladder disease. We also found a more than two-fold increased risk of having chest pain or angina in the follow-up year (Table 3). Cough was the most frequent new diagnosis in both cohorts, but dyspepsia patients carried an increased risk compared with the control cohort (Table 3).

We observed that 21% of all dyspepsia patients visited their GP with dyspepsia symptoms again within 6 months and 6% revisited in the following 6-month period. During the year after the index date, 74% of the patients did not consult again with dyspepsia.

During the follow-up year, 107 (1.5%) patients died in the dyspepsia cohort and 84 (0.8%) in the control

TABLE 2 New medication use in the year after diagnosis of dyspepsia in the 1996 and 1999 dyspepsia cohorts

	1996 dyspepsia cohort ($n = 6913$)				1999 dyspepsia cohort ($n = 4897$)					
	N ^a	≤3 months ^b		>3 months ^b		N^{a}	$\leq 3 \text{ months}^{b}$		>3 months ^b	
		n	%	n	%		n	%	n	%
PPI H ₂ RA Antispasmodics	6680 6361 6403	1025 2435 435	15.3 38.3 6.8	291 188 281	4.4 3.0 4.4	4675 4543 4601	1161 1625 259	24.8 35.8 5.6	234 95 159	5.0 2.1 3.5

^aFor each treatment group, we only analyzed new users (excluding those that had any prescription in the year before the dyspepsia diagnosis). ^bTime after index date that medication use started.

	Dyspepsia cohort			Control cohort			OR^b	(95% CI)
	N^{a}	New cases		N^{a}	New cases			
		п	%		n	%		
Pneumonia	6752	25	0.4	10 821	22	0.2	1.7	0.9–3.0
Asthma	6042	56	0.9	9905	69	0.7	1.1	0.8 - 1.6
COPD	6643	36	0.5	10 723	41	0.4	1.2	0.8 - 1.8
Cough	4964	368	7.4	8633	406	4.7	1.5	1.3-1.7
Laryngitis/hoarseness	6536	30	0.5	10 604	58	0.5	0.9	0.6-1.3
Otitis	6307	77	1.2	10 174	91	0.9	1.3	1.0 - 1.8
Sinusitis	5575	134	2.4	9529	182	1.9	1.2	0.9-1.5
Angina	6219	68	1.1	10 282	42	0.4	2.7	1.8-4.0
Chest pain	6371	348	5.5	10 711	233	2.2	2.3	2.0-2.8
Gallbladder disease	6775	19	0.3	10 929	8	0.1	4.0	1.7–9.4
GERD	6509	304	4.7	10 761	7	0.1	62.8	31.1-127.0
PUD	6890	35	0.5	11 030	2	0.02	27.2	6.5-113.7
IBS	6536	151	2.3	10 706	1	0.01	264.0	36.9–1187
Stress	6519	89	1.4	10 612	62	0.6	2.1	1.5-2.9
Sleep disorders	5991	121	2.0	10 086	103	1.0	1.8	1.4-2.3

 TABLE 3
 ORs for a first time diagnosis of various diseases during the year after the index date among the dyspepsia cohort compared with the control cohort

The analysis was performed among those without a diagnosis of these diseases before the index date. An independent logistic model was built for each of the outcomes. COPD: chronic obstructive pulmonary disease.

^aNumber of patients without the specified diagnosis in the 12 months before the index date.

^bOR estimates were adjusted by age, sex, smoking, alcohol consumption, BMI and visits to a GP.

cohort. The risk of death among patients with dyspepsia was increased compared with the general population (relative risk: 1.6; 95% CI: 1.2–2.2) after adjustment for age, sex, smoking, alcohol use, BMI, visits to the GP and prior morbidities.

Discussion

We found an overall incidence of dyspepsia in 1996 of 15.3 per 1000 person-years, with little change by 1999, where the incidence was 15.5 per 1000 person-years. This is in line with the incidence reported in a previous study from UK general practice (12.8 per 1000 person-years at risk).³ It is, however, lower than that reported in most population-based symptom surveys (annual incidence $3.2-11.5\%^{19-21}$; 56.1 per 1000 person-years²²). This most likely reflects low consultation rates for upper GI symptoms in general practice.²³

The results of this study confirm the association of upper GI symptoms with NSAID^{2,24,25} and aspirin use.^{24,26} Lifestyle factors, however, were found to have only a minor impact on the risk of dyspepsia. Smoking had a small but significant association with the risk of receiving a dyspepsia diagnosis, while the slight association of dyspepsia with obesity was of only borderline significance. Previous studies have also reported a weak association between dyspepsia and smoking^{2,24,27} and BMI.^{28–30} Our findings are in agreement, therefore, with an accumulating body of literature that shows that lifestyle choices are unlikely to have a major role in the development of upper GI symptoms.⁴

We found that patients with a range of pre-existing disorders had a slightly increased likelihood of receiving a new diagnosis of dyspepsia. The association between dyspepsia and chest pain or ischemic heart disease may represent a change in the diagnosis of pre-existing upper abdominal symptoms or a link with underlying GERD.^{31,32} The association with general pain has been noted previously in this patient population,³³ and may be related to the psychosocial issues and stress reported by some patients with dyspepsia.^{20,34,35} The association with osteoarthritis and rheumatoid arthritis, however, could be a consequence of the association with treatments for these diseases, such as aspirin, NSAIDs, steroids and DMARDs.

In the present study, we found a strong association between recorded symptoms of dyspepsia and a subsequent diagnosis of IBS. Overlap of these two conditions has been reported and population-based studies have found the prevalence of dyspepsia among patients with IBS to be 29–87%.^{21,36–38} Some researchers have even suggested that functional GI disorders such as IBS and dyspepsia could represent a single syndrome.^{39–41} We also found that dyspepsia patients had an increased risk of receiving a new GERD diagnosis (with or without esophagitis) in the year after a dyspepsia diagnosis. This is in agreement with previous population studies showing an association between symptoms of GERD and PUD or dyspepsia.^{24,42} This increased risk may be partly linked to diagnostic practice in primary care, where time is used as a diagnostic tool.⁴³ Patients may be given a diagnosis of dyspepsia

until the presence of a more specific GI disease such as GERD or IBS begins to crystallize over time. We found that dyspepsia patients were also more likely to be diagnosed with angina, chest pain and other extraesophageal conditions, some of which carry an increased risk of mortality.⁴⁴ This could explain in part the increased risk of death found for those with dyspepsia compared with controls in this study. There was also an increased risk of cough in the year following a dyspepsia diagnosis, and in absolute terms, this was the most common subsequent diagnosis (7.4%).

Non-ulcer dyspepsia is a chronic and remitting disorder.⁴⁵ We found that a quarter of patients in the 1996 dyspepsia cohort consulted their GP again with dyspepsia in the year after their first diagnosis, reflecting the chronicity of the condition. A similar proportion of patients with abdominal complaints have been found to have persistent symptoms in previous studies.^{46,47} Furthermore, we found that many patients were prescribed GI drugs in the year after a diagnosis of dyspepsia, with 39% receiving H₂RAs and 15% PPIs in the first 3 months. In the 1999 dyspepsia cohort, the percentage of patients who received PPIs had increased to 25%, while H₂RA and antispasmodics use had decreased. This change in prescribing habits reflects growing evidence that acid-suppressive therapies, and among them PPIs in particular, are effective for the treatment of dyspepsia.^{48,49}

The results of this study should be considered in the light of various limitations inherent in an observational database study of this type. The most important of these is the limited information regarding the definition of dyspepsia applied to patients in the study. The type, frequency and severity of symptoms which lead to a diagnosis of dyspepsia are not recorded in the GPRD. Rather, the definition of dyspepsia was determined by the codes used by the GP and included both uninvestigated and functional dyspepsia. In any case, the definition covers a range of upper GI symptoms of unspecified origin recorded by GPs. We also excluded GERD, IBS, PUD and other GI disorders from both cohorts prior to analysis. We excluded the main organic GI disorders in order to limit our study to functional dyspepsia, even though this has been reported to be a difficult if not impossible task based on clinical history.^{50,51} Therefore, we were not able to investigate GERD, PUD or IBS as risk factors for dyspepsia. However, we were able to measure the association with a new diagnosis of these disorders in the year after diagnosis. The very strong association between dyspepsia and a subsequent diagnosis of IBS is likely to be in part due to the low incidence of IBS in the control group used in our study. However, a recent study using the GPRD found an incidence of IBS of 1.9 per 1000 in men and 5.8 per 1000 in women,⁵² which would still equate to a substantially increased likelihood of IBS in patients with dyspepsia in our study.

Further research is needed to elucidate potential risk factors for unspecific dyspepsia. There is also a need to understand how the differential diagnosis of dyspepsia is made in UK general practice and, in particular, how this symptom complex is distinguished from GERD. The range of comorbidities experienced by dyspepsia patients emphasizes the potentially life-impairing effects of upper GI symptoms, and a recent systematic review has highlighted the need for further investigations, particularly those with relevance to general practice.⁵³ Recognition of the associations between dyspepsia and certain comorbidities and medication use may assist with the management of upper GI symptoms in general practice.

Conclusions

Over 1% of the UK general population consults a GP with newly presenting dyspeptic symptoms each year. Our study shows that lifestyle factors such as obesity, smoking and alcohol consumption are unlikely to have a major role in the development of dyspepsia. The link between NSAID therapy and dyspepsia reinforces the need to consider appropriate GI therapy for patients receiving chronic NSAID treatment.⁵⁴ The association between dyspepsia and a subsequent diagnosis of IBS, GERD and PUD shows that a proportion of patients who initially receive a non-specific diagnosis of dyspepsia are subsequently given a firmer diagnosis of the underlying cause of their symptoms. Patients with dyspepsia also have an increased risk of receiving potentially serious diagnoses such as chest pain or angina. Our findings also reflect the difficulties that GPs inevitably encounter in trying to make a firm diagnosis in clinical practice.

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Declaration

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