Hospitalizations for Acute Myocardial Infarction are Decreased Among Patients with Inflammatory Bowel Disease Using a Nationwide Inpatient Database

Edward L. Barnes, MD[1], **Renee Marchioni Beery, DO**[1], **Allison R. Schulman, MD**[1], **Ellen P. McCarthy, PhD, MPH**[2], **Joshua R. Korzenik, MD**[1], and **Rachel W. Winter, MD**[1]

[1]Division of Gastroenterology, Hepatology, and Endoscopy, Department of Medicine, Brigham and Women's Hospital, Boston, MA, USA

[2]Division of General Medicine and Primary Care, Department of Medicine, Beth Israel Deaconess Medical Center; Office for Diversity Inclusion and Community Partnership, Harvard Medical School, Boston, MA, USA

Abstract

Background: Questions remain regarding the true prevalence of cardiovascular events such as myocardial infarction (MI) among patients with Inflammatory Bowel Disease (IBD). Using the Nationwide Inpatient Sample (NIS), we aimed to compare the proportion of hospitalizations for acute MI among patients with IBD to that of the general population.

Methods: This study used data from years 2000–2011 in NIS, the largest publicly available all-payer inpatient database in the US. International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM) discharge codes were used to identify adult patients with discharge diagnoses of IBD (Ulcerative Colitis or Crohn's Disease), acute MI, and multiple comorbid risk factors for cardiovascular disease. The independent effect of a diagnosis of IBD on risk of acute MI was examined using a multivariable logistic regression model controlling for multiple confounders. Data were analyzed using SAS survey procedures and weighted to reflect national estimates.

Results: We identified 567,438 hospitalizations among patients with IBD and 78,121,000 hospitalizations among the general population. Patients with IBD were less likely to be hospitalized for acute MI than patients in the general population (1.3% vs. 3.1%, p<0.001). In adjusted analyses, the odds of hospitalization for acute MI among patients with IBD were decreased when compared to the general population (OR 0.51, 95% CI 0.50 – 0.52).

Conclusions: Despite prior reports of a potentially increased risk of acute MI among patients with IBD, in a nationwide inpatient database, lower rates of acute MI were demonstrated in the IBD population when compared to the general population.

Corresponding Author: Edward L. Barnes, MD, Brigham and Women's Hospital, 75 Francis Street, Boston, MA 02115, ebarnes1@partners.org, Phone: 617-732-6389, Fax: 617-566-0338.

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Keywords

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Introduction:

Patients with chronic inflammatory conditions such as rheumatoid arthritis (RA), ankylosing spondylitis, and systemic lupus erythematosus (SLE) are at increased risk for the development of endothelial dysfunction, atherosclerosis and ultimately atherothrombotic events.[1–9] In particular, an inflammatory protein, C-reactive protein (CRP), has been identified as a predictor of cardiovascular risk given its association with increased morbidity and poor outcomes among patients with vascular disease as well as its potential role in atherogenesis.[10–16] The Inflammatory Bowel Diseases (IBDs), including Crohn's Disease (CD) and Ulcerative Colitis (UC) have been similarly associated with increased cardiovascular risks, including coronary artery disease (CAD) and thromboembolic events of the venous and arterial systems.[17–20] Patients with IBD often have long periods of remission with intermittent relapses of intestinal inflammation, and these increased cardiovascular risks have been documented during periods of both IBD flare and remission.

The risk burden for cardiovascular disease in IBD is unique and perhaps less well-defined relative to that of the general population. Traditional risk factors such as hyperlipidemia, obesity, and diabetes mellitus are less common among patients with IBD,[21] and thus the increased risk profile of cardiovascular disease in IBD is poorly defined.[22] Additional potential risk factors for cardiovascular disease in IBD include elevated levels of inflammatory cytokines (tumor necrosis factor α, CRP, and interleukin-6) and hypercoagulable states.[22] Prior meta-analyses have not consistently demonstrated an association between IBD and an increased risk for cardiovascular disease related mortality.[19,23] Thus, the actual risk of cardiovascular disease, in particular myocardial infarction (MI), in IBD has become a growing area of clinical and scientific interest. In this context, we sought to compare the rate of acute MI among patients with IBD to that of the general United States (US) population using a nationwide sample of inpatient hospitalizations between 2000 and 2011.

Materials and Methods:

Data source:

We conducted a retrospective cross-sectional study using consecutive years of the Nationwide Inpatient Sample (NIS) database. NIS is the largest publicly available all-payer inpatient database in the United States. It is designed to be nationally representative, and for the years 2000 through 2011, NIS captured discharges from a 20% stratified probability sample of community hospitals in the United States.[24] In 2011, NIS contained information from over 8 million discharges from 1049 hospitals across 46 states.[25] The NIS contains information on demographic characteristics and insurance status, up to 15 diagnostic and procedure codes based on the International Classification of Diseases, 9th Revision, Clinical

Modification (ICD-9-CM), hospital characteristics, and outcomes including hospital length of stay, inpatient death, and total costs. Due to the de-identified nature of the data, this study was determined exempt from review by the Partners Healthcare Institutional Review Board.

Study sample:

We examined the discharges of patients age 18 and older. Patients were determined to have IBD if they had presence of ICD-9-CM diagnosis codes indicating CD (555.xx) or UC (556.xx). To minimize misclassification of subgroups of IBD (CD or UC), we excluded patients with discharge codes for both CD and UC (n=3751).

Outcomes of Interest:

The primary outcome of interest, acute MI, was determined by the presence of ICD-9-CM codes 410.xx. We were also interested in the risk of developing coronary artery disease (CAD) among patients with IBD; however, we had concerns that existing CAD may be underdiagnosed/undercoded in patients admitted with known IBD but without cardiovascular complications at the time of a current admission. For this reason we chose to evaluate acute MI as the primary outcome and CAD as a secondary outcome. CAD was determined by presence of ICD-9-CM codes 414.xx.

Covariates:

Patient demographics included age, sex, and race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, Asian or Pacific Islander, Native American, other, and unknown), primary insurance/payer for the admission, and year of admission. ICD-9-CM diagnosis codes were used to identify additional clinical factors known to be independent predictors or confounders of the relationships between IBD and acute MI/CAD including hypertension, diabetes, hyperlipidemia, obesity (defined as Body Mass Index [BMI] > 30 kg/m^2), and tobacco use (Appendix 1). Additionally, ICD-9-CM diagnosis codes were used to identify other known inflammatory conditions, RA (714.xx) and SLE (710.0). We used the Deyo-modification of the Charlson Comorbidity Index as a marker of comorbid illness.[26] When this comorbidity index was used in the evaluation of the primary outcome (acute MI), the index was further modified to remove acute MI and old MI from the scoring system.

Statistical Analysis:

All analyses were performed using survey procedures in SAS version 9.3 (SAS Institute, Cary, NC, USA) to account for the complex survey design. Data were weighted to reflect estimates of the national population including means and proportions presented herein. Chi-Square test and Student t-test were used to compare proportions and continuous variables (under the assumption of the Central Limit Theorem), respectively. First, a logistic regression model was constructed to estimate the unadjusted odds of a diagnosis of acute MI among patients with IBD compared to those patients without IBD. Based on clinical knowledge, a multivariable logistic regression model was then constructed to evaluate the adjusted association between IBD and acute MI, adjusting for the covariates identified above. All factors were included in the model due to their clinical relevance, with diabetes

included in the Charlson Comorbidity Index. These analyses were then repeated to examine the relationship of IBD to the secondary outcome of diagnosis of CAD.

Given concerns that diagnoses of IBD might be underrepresented among patients hospitalized with acute MI, we also evaluated acute MI among two patient populations with other chronic inflammatory diseases (RA and SLE). A two tailed p-value of 0.05 was chosen as the threshold for statistical significance for all tests. Odds ratios (OR) and 95% confidence intervals (CI) are presented.

Results:

From 2000–2011, NIS included a total of 563,687 discharges for patients with IBD and 78,121,000 discharges for the general population, representing an estimated 2.77 million hospitalizations among patients with IBD and 382.79 million hospitalizations among the general population. Of the 563,687 discharges for patients with IBD, 359,098 (63.7%) had CD and 204,589 (36.3%) had UC. As noted above, 3,751 discharges recorded diagnosis codes pertaining to both CD and UC and were excluded from the analyses.

Baseline characteristics for patients with and without IBD are shown in Table 1. Compared to patients without IBD, patients with IBD tended to be younger (mean age 51.6 years vs. 57.0 years) and were less likely to have diagnosed comorbidities of hyperlipidemia, diabetes mellitus, hypertension, and obesity (all p-values <0.001).

Overall, patients with IBD were less likely to have a diagnosis of acute MI compared to patients without IBD (1.3% vs 3.1%, p<0.001), with unadjusted OR 0.42, 95% CI 0.41 – 0.43. After adjusting for age, sex, race, hypertension, hyperlipidemia, obesity, tobacco abuse, modified Charlson Comorbidity Index, year of hospitalization, and primary insurance/payer, patients with IBD had a 0.51 fold odds of diagnosis of acute MI when compared to patients without IBD (adjusted OR 0.51, 95% CI 0.50 – 0.52, Table 2). The overall trends for acute MI per 100,000 hospitalizations, analyzed by hospitalization year, are seen in Figure 1.

When evaluating only patients with IBD, patients with UC were more likely to have a diagnosis of acute MI than patients with CD (1.8% vs. 1.1%, p<0.001). In an unadjusted analysis compared to patients without IBD, patients with UC (OR 0.57, 95% CI 0.55 – 0.59) and CD (OR 0.34, 95% CI 0.33 – 0.35) demonstrated decreased odds of being diagnosed with acute myocardial infarction. After adjusting for age, sex, race, hypertension, hyperlipidemia, obesity, tobacco abuse, modified Charlson Comorbidity Index, year of hospitalization, and primary insurance/payer, patients with both UC (OR 0.61, 95% CI 0.59 – 0.64) and CD (OR 0.44, 95% CI 0.43 – 0.46) continued to demonstrate significantly decreased odds of being diagnosed with acute myocardial infarction when compared to patients without IBD.

In evaluating the secondary outcome, 10.7% of patients with IBD had a discharge diagnosis of CAD compared to 18.8% of patients without IBD (p<0.001). In both an unadjusted analysis (OR 0.52, 95% CI 0.51 – 0.52) and an adjusted analysis controlling for age, sex, race, hypertension, hyperlipidemia, obesity, tobacco abuse, Charlson Comorbidity Index,

year of hospitalization, and primary insurance/payer (OR 0.71, 95% CI 0.70 – 0.71) patients with IBD demonstrated lower odds of a discharge diagnosis of CAD when compared to patients without IBD (Table 3). When compared to patients without IBD, patients with UC demonstrated a decreased odds of diagnosis of CAD in both unadjusted (OR 0.65, 95% CI 0.65 – 0.66) and adjusted analyses controlling for age, sex, race, hypertension, hyperlipidemia, obesity, tobacco abuse, Charlson Comorbidity Index, year of hospitalization, and primary insurance/payer (OR 0.74, 95% CI 0.73 – 0.75). Patients with CD also demonstrated a decreased odds of diagnosis of CAD when compared to patients without IBD in both an unadjusted analysis (OR 0.44, 95% CI 0.44 – 0.45) and an adjusted analysis controlling for age, sex, race, hypertension, hyperlipidemia, obesity, tobacco abuse, Charlson Comorbidity Index, year of hospitalization, and primary insurance/payer (OR 0.71, 95% CI 0.70 – 0.72).

Hospitalization for acute MI occurred more frequently among patients with RA (3.0%) and SLE (2.3%) than among patients with IBD (1.3%, p<0.001). Additionally, in both unadjusted and multivariable regression analysis controlling for age, sex, race, hypertension, hyperlipidemia, obesity, tobacco abuse, modified Charlson Comorbidity Index, year of hospitalization, and insurance/payer, patients with RA (OR 1.45, 95% CI 1.41 – 1.49) and SLE (OR 1.72, 95% CI 1.67 – 1.77) had significantly higher odds of discharge with a diagnosis of acute MI than patients with IBD (Table 4).

Prior studies[27] have raised concerns that women and younger patients with IBD may have greater risk of development of acute MI; thus, we performed subgroup analyses of these populations. In a comparison of diagnosis of acute MI stratified by sex, both women (1.0% vs. 2.2%, p<0.001) and men (1.7% vs. 4.5%, p<0.001) with IBD were less likely to have a diagnosis of acute MI than their respective populations without IBD. While patients with IBD who were diagnosed with acute MI had a lower mean age (68.4 years) than patients without IBD who were diagnosed with acute MI (69.3 years, p <0.001), when divided into age groups of greater than 50 years and less than 50 years, patients with IBD were less likely to have a diagnosis of acute MI than patients without IBD in both age groups (2.3% vs. 4.5% and 0.26% vs. 0.84%, respectively, p<0.001).

Discussion:

In this study we utilized a large, nationwide database to demonstrate a significant decrease in the odds of being discharged with a diagnosis of acute MI among patients with IBD compared to patients without IBD. In both a crude analysis and in a multivariable analysis adjusting for comorbidities associated with increased risk of cardiovascular disease and MI, we found that patients with IBD demonstrated significantly lower odds of being diagnosed with acute MI and CAD when compared to patients without IBD. The decreased odds of being diagnosed with acute MI or CAD were demonstrated in patients with both UC and CD when the IBD subtypes were evaluated independently.

The association between venous thromboembolic disease and IBD has been well established, as patients with IBD carry an estimated three-fold increased risk of venous thromboembolic events.[20] However, the potential for IBD to act as a risk factor for

development of CAD and the relationships between IBD and other cardiovascular outcomes such as mortality is less clear.[19] While recent large database studies have demonstrated an increased risk of ischemic heart disease and MI among patients with IBD, particularly during times of increased disease activity,[17,28] questions remain regarding the actual risk of MI in this population.[29,30]

Several mechanisms linking the pro-inflammatory state associated with active IBD and increased cardiovascular risk have been proposed, including derangements in the coagulation cascade,[31] elevations in inflammatory mediators such as CRP[32] and homocysteine,[33–35] and alterations in endothelial, microvascular, and macrovascular function.[36] As these factors would be suspected to carry the greatest risk during flares of disease, there remains the potential that the lack of clinical information regarding relapse/remission of disease and the cross sectional design of our study did not allow for an accurate comparison of odds of acute MI during the periods of greatest risk. However, the potential also remains that any increased risk associated with the pro-inflammatory state is offset by a lack of other risk factors such as obesity, hypertension, and hyperlipidemia, which were lower in the IBD population. The higher rate of tobacco abuse documented in patients with IBD may act in multiple ways among the patients with separate sub-types of IBD. While smoking is known to increase overall risk of cardiovascular disease,[37] smoking among some patients with UC is associated with a milder disease course and thus less inflammatory burden overall.[38–40] Alternatively, among patients with CD, smoking is associated with an increase in both disease activity and complications.[41]

In 2008, one of the first large studies evaluating the risk of ischemic heart disease in patients with IBD was published using a large registry database from Canada. Patients with IBD were found to have a significantly increased risk of ischemic heart disease when compared to non-IBD patients (IRR 1.26, 95% CI 1.11 – 1.44).[42] Subsequently, a nationwide registry-based study of 4.6 million Danish patients, including 28,833 patients with IBD, revealed a modestly increased risk of ischemic heart disease in patients with IBD.[28] This risk was particularly increased in the first year after diagnosis,[28] perhaps indicating a different underlying pathophysiology of CAD in the IBD population. A historical cohort study of IBD patients undergoing cardiac catheterization found that patients with IBD developed CAD at a younger age, although their post-percutaneous coronary intervention outcomes were similar to those patients without IBD.[27]

One potential reason for the differences in findings between our study and prior database studies from Canada[42] and Denmark[28] relates to the nature of NIS as an inpatient hospitalization database. The Manitoba Health administrative database utilized by Bernstein, et al.[42] contains information regarding both inpatient hospitalizations and outpatient physician visits. The Danish study cohort was derived from the Danish National Patient Register, which contains individual patient information about outpatient visits and inpatient hospitalizations.[28] Similarly, a retrospective longitudinal cohort study from the US that demonstrated an increased risk for development of CAD among patients with IBD included both inpatient and outpatient evaluations.[21] If there were an increased likelihood for more than one disease to be coded in an outpatient setting as opposed to an inpatient setting, this would not be reflected in NIS as it is an inpatient only database. Additionally, the mean ages

of both the IBD and non-IBD patients in our study population were greater than the mean ages of those populations the prior database studies from Canada[42] and Demark.[28] This increased age would seem to favor an increased likelihood of acute MI in our population, given the mean ages of arterial thromboembolic disease demonstrated in the Manitoba Health administrative database.[42] However, fundamental differences in the baseline demographics of these studies should be noted, as they are potentially indicative of the differences between an inpatient only population and a database that utilizes inpatient and outpatient visits.

Other studies have demonstrated risks of CAD among patients with IBD that are more similar to the population as a whole. A population-based study utilizing a large claims database from the US demonstrated an increased risk of MI only among a subgroup of women with IBD aged more than 40 years, after adjustment for many confounders including hyperlipidemia, diabetes, and hypertension.[43] Similarly, a study from the United Kingdom (UK) evaluating 15,498 patients with UC and 9,829 patients with CD found an increased risk of acute MI among patients with IBD, however this risk was attenuated in a multivariable model.[29] Meta-analyses have reported conflicting results, as cardiovascular disease (including ischemic heart disease) was increased in patients with IBD (OR 1.18, 95% CI 1.08 – 1.31) in one meta-analysis[44] and not significantly increased in another meta-analysis (RR 1.23, 95% CI 0.94 – 1.62).[19]

In a study of cardiovascular morbidity among hospitalized patients with IBD that used a single year of data from NIS, Sridhar, et al. demonstrated lower odds of ischemic heart disease among patients with both UC and CD, after adjusting for hypertension, hyperlipidemia, and diabetes.[30] Though we report a similar outcome of patients with IBD demonstrating decreased odds of being diagnosed with CAD, our analysis differs from the prior published study as we evaluated outcomes over a longer period of consecutive years. In addition, we attempted to control for several additional factors in our multivariable analysis, including the modified Charlson Comorbidity Index, and insurance/payer status. Given our concerns that CAD may potentially be underdiagnosed among inpatients with IBD, we also chose to evaluate acute MI as our primary outcome. In a comparison not used in the prior study, we also compared the rate of acute MI among inpatients with IBD to other chronic inflammatory conditions (RA and SLE).

Our study has several important results. The decrease in odds of being discharged with a diagnosis of acute MI among patients with IBD is an important finding, particularly as this was seen in both an unadjusted analysis and after adjustment for multiple potential confounders. While these results differ from two large studies from Canada[42] and Denmark,[17] they do mirror the results of database studies from the UK[29] and the lack of overall difference demonstrated in at least one meta-analysis.[19] Additionally, the lower frequency of diagnosis of acute MI among patients with IBD when compared to other chronic inflammatory conditions such as RA and SLE may indicate a difference in pathophysiology among these diseases.

This study does have multiple limitations. Notably, the NIS does not include information regarding an individual patient's medication profile prior to admission or during the

hospitalization. Thus, we were unable to evaluate the potential relationship between steroid use or other immunosuppressive therapy and risk of acute MI, an area where differing results have previously been reported.[45,46] The NIS does not contain data regarding the severity of an individual patient's IBD at the time of admission. In a large, nationwide Danish cohort study,[17] the risk of MI was among patients with IBD was increased only during periods of flare and persistent disease activity and not during periods of disease remission. Additionally, the NIS does not contain data regarding the duration of IBD, and thus we were unable to evaluate the effect of prolonged inflammation on development of CAD or risk of acute MI.

With a large database such as NIS, there is potential for misclassification of both our exposure and outcome. We attempted to limit this by excluding any patient with an ICD-9-CM code for both UC and CD; however, this bias may still exist. While International Classification of Disease codes have been used in other studies examining the relationship between cardiovascular disease and IBD,[17,18,21,27,28,30,42] the NIS does not contain personal identifiers, and thus we are unable to validate the ICD-9-CM diagnosis codes of individual patients using chart review. ICD-9-CM coding has demonstrated accuracy in IBD[47,48] and other studies of gastrointestinal disorders,[49] however this remains a concern in our population given mixed performance within studies of patients with IBD in the Veterans Affairs system.[50,51] There also exists the potential for underdiagnosis of other risk factors, particularly tobacco use and obesity.[52]

Perhaps the most significant limitation of the use of administrative coding relates to the potential for an "undercoding" of IBD among patients that are experiencing acute MI. If a patient has an ICD-9-CM code for acute MI as a primary diagnosis, and an ICD-9-CM code for UC or CD is not included as a secondary diagnosis, this could lead to the appearance of fewer occurrences of acute MI among patients with IBD. To evaluate this limitation, we compared the frequency of acute MI among patients with IBD to two other chronic inflammatory conditions (RA and SLE). Although the clinical presentation and underlying disease processes differ among patients with RA and SLE, patients with these diseases share an underlying chronic inflammation that has been linked to increased cardiovascular risk.[53] Patients with RA have demonstrated an increased risk of acute MI compared to the general population.[54,55] Similarly patients with SLE have demonstrated an increased risk of acute MI,[7] and in some cases acute MI can precede the diagnosis of SLE.[56] In this evaluation, patients with IBD were less likely to have a diagnosis of acute MI than patients with RA or SLE. The differences in risk for arterial thromboemboli when comparing patients with IBD to patients with RA or SLE may be due to a difference in platelet dysfunction that leads to more venous thrombosis than arterial thrombosis among patients with IBD,[29] though the biologic mechanisms underlying the different risk profiles associated with these chronic inflammatory conditions are not well understood.

In conclusion, using a large, nationwide inpatient database, we demonstrated decreased odds of diagnosis of acute MI among patients with IBD compared to patients without IBD. Additionally, patients with IBD were discharged with a diagnosis of acute MI less frequently than patients with other chronic inflammatory conditions such as RA and SLE. Although

providers should be cognizant of cardiovascular risk factors among patients with IBD, the risk of acute MI may not be as significant as reported in other studies.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgement of Support:

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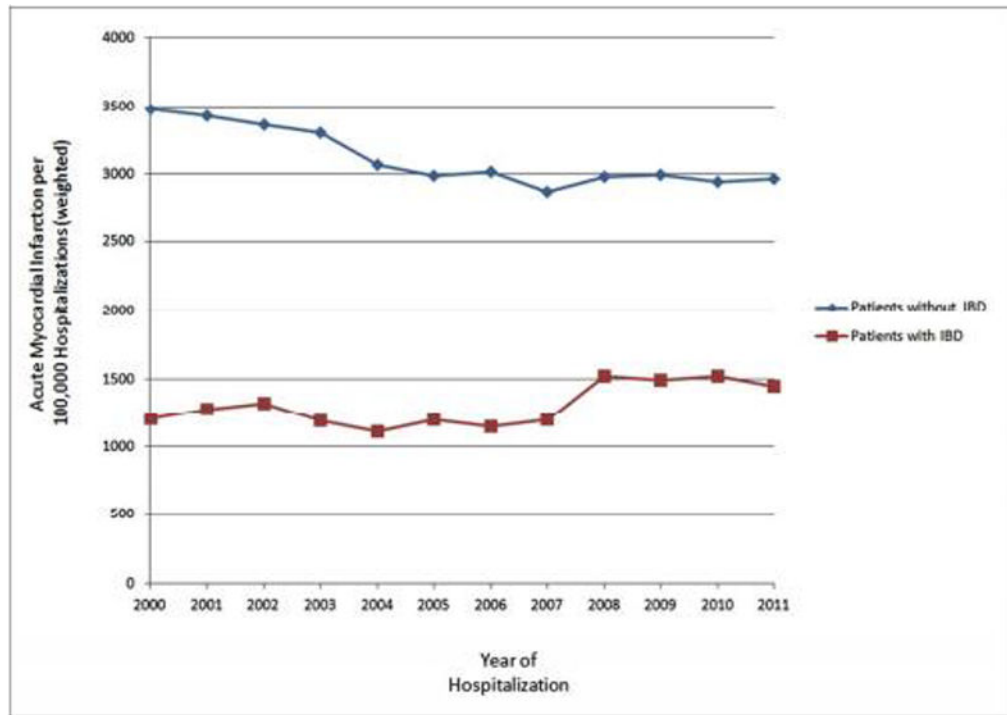
Figure 1.
Number of Acute Myocardial Infarctions per 100,000 Hospitalizations among Patients with Inflammatory Bowel Disease compared to Patients without Inflammatory Bowel Disease
84×59mm (300 × 300 DPI)

Table 1.

Baseline Demographic and Clinical Characteristics among Hospitalized Patients in the Nationwide Inpatient Sample

	Patients with IBD (n=563,687)	Patients without IBD (n=78,121,000)	p-value
Age[a]	51.6 (0.15)	57.0 (0.002)	<0.001
Sex:			<0.001
Female	326153 (57.8%)	47408967 (60.7%)	
Male	237111 (42.1%)	30623519 (39.2%)	
Race/ethnicity:[b]			<0.001
Non-Hispanic White	363699 (64.5%)	42418256 (54.3%)	
Non-Hispanic Black	41378 (7.3%)	8446106 (10.8%)	
Hispanic	21735 (3.8%)	6549449 (8.3%)	
Asian or Pacific Islander	3933 (0.7%)	1382922 (1.7%)	
Native American	1479 (0.3%)	325294 (0.4%)	
Other	8965 (1.6%)	1737149 (2.2%)	
Unknown	122498 (21.7%)	17261824 (22.1%)	
Primary Insurance/Payer:			<0.001
Medicare	202096 (36.0%)	34839556 (44.8%)	
Medicaid	55608 (9.9%)	11031686 (14.2%)	
Private Insurance	258431 (45.9%)	25246763 (32.3%)	
Self-Pay	25757 (4.6%)	3908146 (5.0%)	
No Charge	2874 (0.5%)	380445 (0.5%)	
Other	17687 (3.1%)	2544927 (3.2%)	
Hyperlipidemia	71023 (12.6%)	14350542 (18.4%)	<0.001
Diabetes Mellitus	69802 (12.4%)	16061332 (20.6%)	<0.001
Hypertension	152018 (27.0%)	27256308 (34.9%)	<0.001
Tobacco Abuse	68985 (12.2%)	7961496 (10.2%)	<0.001
Obese	26545 (4.7%)	5223999 (6.7%)	<0.001
Modified Charlson Comorbidity Index[c]			<0.001
Index Score 1	476989 (84.6%)	57175337 (73.2%)	
Index Score 2	49775 (8.8%)	11261611 (14.4%)	
Index Score 3	36923 (6.6%)	9684052 (12.4%)	

[a]Age is a continuous variable, presented as mean (Standard Deviation) Categorical variable presented as number with associated (%)

[b]Race/ethnicity is a missing variable for 22% of patients in sample, listed as unknown

[c]Charlson Comorbidity Index modified to remove acute MI and old MI from scoring

Table 2.

Unadjusted and Adjusted Odds Ratios for the Relationship between Inflammatory Bowel Disease and Myocardial Infarction

	Odds Ratio (95% CI)
Unadjusted Analysis	
Inflammatory Bowel Disease	0.42 (0.41 – 0.43)
Adjusted Analysis (Mutivariable Model)	
Inflammatory Bowel Disease	0.51 (0.50 – 0.52)
Age:	
Age 18–39	0.12 (0.12 – 0.12)
Age 40–60	Reference
Age 60–80	1.69 (1.68 – 1.70)
Female Sex	0.56 (0.56 – 0.56)
Race/ethnicity:	
Non-Hispanic White	Reference
Non-Hispanic Black	0.79 (0.79 – 0.79)
Hispanic	0.92 (0.92 – 0.93)
Asian or Pacific Islander	1.12 (1.11 – 1.13)
Native American	1.11 (0.99 – 1.04)
Other	1.20 (1.19 – 1.21)
Unknown	1.01 (1.01 – 1.02)
Hyperlipidemia	2.19 (2.19 – 2.20)
Hypertension	0.90 (0.90 – 0.91)
Obese	1.07 (1.06 – 1.07)
Tobacco Abuse	1.84 (1.83 – 1.85)
Modified Charlson Comorbidity Index[a]:	
Index Score 1	Reference
Index Score 2	1.11 (1.10 – 1.11)
Index Score 3	0.94 (0.94 – 0.95)
Primary Insurance/Payer:	
Medicare	Reference
Medicaid	0.79 (0.79 – 0.80)
Private Insurance	1.03 (1.02 – 1.03)
Self-Pay	1.37 (1.36 – 1.38)
No Charge	1.21 (1.19 – 1.24)

	Odds Ratio (95% CI)
Other	0.90 (0.80 – 0.91)
Year of Hospitalization:	
2000	Reference
2001	0.95 (0.94 – 0.95)
2002	0.91 (0.91 – 0.92)
2003	0.88 (0.87 – 0.88)
2004	0.80 (0.87 – 0.88)
2005	0.74 (0.73 – 0.74)
2006	0.73 (0.73 – 0.73)
2007	0.68 (0.68 – 0.69)
2008	0.68 (0.67 – 0.68)
2009	0.66 (0.66 – 0.67)
2010	0.64 (0.64 – 0.65)
2011	0.62 (0.62 – 0.63)

[a]Charlson Comorbidity Index was modified to remove acute MI and old MI from scoring, as acute MI is the primary outcome being modeled

Table 3.

Unadjusted and Adjusted Odds Ratios for the Relationship between Inflammatory Bowel Disease and Coronary Artery Disease

	Odds Ratio (95% CI)
Unadjusted Analysis	
Inflammatory Bowel Disease	0.52 (0.51 – 0.52)
Adjusted Analysis (Mutivariable Model)	
Inflammatory Bowel Disease	0.71 (0.70 – 0.71)
Age:	
Age 18–39	0.09 (0.09 – 0.09)
Age 40–60	Reference
Age 60–80	2.09 (2.09 – 2.10)
Female Sex	0.51 (0.51 – 0.51)
Race/ethnicity:	
Non-Hispanic White	Reference
Non-Hispanic Black	0.75 (0.75 – 0.76)
Hispanic	0.87 (0.87 – 0.88)
Asian or Pacific Islander	0.78 (0.78 – 0.79)
Native American	1.04 (1.03 – 1.05)
Other	0.97 (0.96 – 0.97)
Unknown	1.35 (1.35 – 1.35)
Hyperlipidemia	3.25 (3.25 – 3.25)
Hypertension	1.35 (1.35 – 1.35)
Obese	1.10 (1.10 – 1.11)
Tobacco Abuse	1.24 (1.24 – 1.25)
Charlson Comorbidity Index:	
Index Score 1	Reference
Index Score 2	1.85 (1.85 – 1.86)
Index Score 3	1.94 (1.94 – 1.94)
Primary Insurance/Payer:	
Medicare	Reference
Medicaid	0.73 (0.73 – 0.73)
Private Insurance	0.70 (0.70 – 0.70)
Self-Pay	0.67 (0.66 – 0.67)
No Charge	0.64 (0.63 – 0.65)
Other	0.64 (0.63 – 0.64)

	Odds Ratio (95% CI)
Year of Hospitalization:	
2000	Reference
2001	1.01 (1.01 – 1.02)
2002	0.97 (0.97 – 0.98)
2003	0.97 (0.97 – 0.98)
2004	0.92 (0.92 – 0.92)
2005	0.87 (0.87 – 0.87)
2006	0.89 (0.89 – 0.89)
2007	0.83 (0.82 – 0.83)
2008	0.82 (0.82 – 0.82)
2009	0.81 (0.81 – 0.82)
2010	0.74 (0.74 – 0.75)
2011	0.74 (0.73 – 0.74)

Table 4.

Unadjusted and Adjusted Odds Ratios for the Relationship between Rheumatoid Arthritis, Systemic Lupus Erythematosus, Inflammatory Bowel Disease and Myocardial Infarction

	Odds Ratio (95% CI)
Unadjusted Analysis	
Inflammatory Bowel Disease	Reference
Systemic Lupus Erythematosus	1.74 (1.69 – 1.80)
Rheumatoid Arthritis	2.32 (2.26 – 2.38)
Adjusted Analysis[a]	
Inflammatory Bowel Disease	Reference
Systemic Lupus Erythematosus	1.72 (1.67 – 1.77)
Rheumatoid Arthritis	1.45 (1.41 – 1.49)

[a]Adjusted for age, sex, race, hypertension, hyperlipidemia, obesity, tobacco abuse, modified Charlson Comorbidity Index, year of hospitalization, and insurance/payer