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Factors that contribute to blood loss in patients with colonic angiodysplasia from a population-based study

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Abstract

Background & Aims—Most studies of angiodysplasia are small and performed at a single center. We investigated the epidemiology and management of colonic angiodysplasia using a national endoscopy database.

Methods—Colonoscopy reports (n=229,727; generated from January 2000 to December 2002) from patients with documented angiodysplasia (n=4159) were retrieved from the Clinical Outcomes Research Initiative. Predictors of occult or overt blood loss and endoscopic treatment were identified using multivariate logistic regression.

Results—Most patients with documented angiodysplasia were more than 60 years old (73%) or had right-sided lesions (62%). There was evidence of blood loss in 56% of patients with angiodysplasia. Predictors of blood loss included inpatient status (odds ratio [OR]: 8.74; 95% confidence interval [CI]: 5.42–14.10], 2–10 angiodysplasias (OR: 1.50, 95% CI: 1.29–1.75), more than 10 lesions (OR: 2.18, 95% CI: 1.69–2.80), black race (OR:1.95, 95% CI: 1.46–2.62, severe illness (OR: 1.97, 95% CI: 1.62–2.41), Hispanic ethnicity (OR: 1.71, 95% CI: 1.32–2.22), and age greater than 80 years (OR: 1.32, 95% CI: 1.06–1.63). Endoscopic therapy was given to 28% of patients with evidence of blood loss and in 68% with active bleeding. Endoscopic treatment increased among patients in a university practice setting (vs. community setting, OR: 2.53, 95% CI: 1.96–3.27) and decreased in Northwest geographic locations (vs. Southwest, OR: 0.60, 95% CI: 0.4–0.84].

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Study concept and design (JH, DL, GE, LS); acquisition of data (JH, DL, GE, LS); analysis and interpretation of data (ND, JH, DL, GE, LS); drafting of the manuscript (ND, LS); critical revision of the manuscript for important intellectual content (ND, JH, DL, GE, LS); statistical analysis (JH, LS); obtained funding (DL, GE, LS)

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Conclusions—Predictors of blood loss in patients with colonic angiodysplasia include inpatient status, comorbities, age, race/ethnicity, and lesion number. Endoscopic therapy for angiodysplasia varied according to practice setting and region.

Keywords

CORI; lower gastrointestinal bleeding; practice patterns

INTRODUCTION

Angiodysplasia is an important cause of occult and acute gastrointestinal blood loss, particularly in the elderly. Estimates of prevalence vary from 2 to 50% in studies of colonoscopy performed for different indications.[1–9] In studies evaluating severe, ongoing lower intestinal bleeding, angiodysplasia is found in as many as 37–50% of patients.[7–9] In one study of 964 healthy, asymptomatic patients, angiodysplasia was seen in less than 1%.[10]

Angiodysplasia is thought to result from chronic, intermittent, low-grade obstruction of submucosal veins.[11] It may also be a complication of decreased mucosal blood flow and local ischemia.[12] Most lesions are found in the cecum and ascending colon possibly due to increased wall tension of the right colon, but angiodysplasia can be found throughout the gastrointestinal tract.[1, 6, 13] Patients with angiodysplasia are usually older than 60 years of age, with a more than 200 fold increase in incidence between the third and ninth decades of life.[3] This increased incidence with age supports the theory that acquired angiodysplasias are due to degenerative changes.[11]

Most colonic angiodyplasia are diagnosed by endoscopy. Colonoscopy has a sensitivity of greater than 80% when the entire colon is examined.[13] Endoscopic therapy for bleeding angiodysplasia is successful,[9, 14–18] using a variety of treatment modalities.[18] Therapy is generally indicated only when there is evidence of blood loss or active bleeding from a lesion and not for incidentally found, asymptomatic angiodysplasia.[19]

Most studies of angiodysplasia are small and reflect a single center's experience. The aims of this study were to use the Clinical Outcomes Research Initiative (CORI) database to describe the epidemiology and examine the endoscopic treatment practices of colonic angiodysplasia.

METHODS

Clinical Outcomes Research Initiative

CORI is a consortium of gastroenterology practices designed to study outcomes of endoscopic procedures in routine practice. The CORI consortium during the study period included 73 gastroenterology practice sites from 27 states including private community practices (71%), university hospitals (17%), and Veterans Affairs hospitals (12%). Participating physicians (525 for this study) use a computerized endoscopic procedure report generator which electronically transmits de-identified reports to a centralized data repository. The data then undergo a series of quality control measures. The CORI database is reviewed annually by the institutional review board at Oregon Health and Science University. Given the use of pre-existing, de-identified data, this study was exempt from institutional review board review at the University of Washington (45 CFR 46.102(f)).

Study Patients

We identified all unique patients in CORI who were documented to have at least one angiodysplasia during colonoscopic examination between January 1, 2000 and December

31, 2002. Patients were excluded if they were younger than 20 years of age or if the endoscopy report noted radiation proctitis and angiodysplasia located in the rectum only (n=103).

We collected data entered by the endoscopists including patient demographics, ASA classification (American Society for Anesthesiologist), exam location (inpatient/outpatient), exam indications, practice type and location, as well as endoscopic findings and treatment. The endoscopic report generator prompts physicians to provide detailed descriptors of angiodysplasia including number, size, location, and whether these lesions were bleeding at the time of the exam. Angiodysplasia number and size (without formal measurement) were estimated by the endoscopists. Details of endoscopic treatment are also collected. In addition, we reviewed 1,781 free text comment fields for additional information regarding evidence of blood loss, angiodysplasia characteristics, and endoscopic treatment.

Statistical Analysis

The main outcomes of interest were evidence of overt or occult blood loss and receipt of endoscopic treatment. Evidence of blood loss was defined as the report of anemia (including iron deficiency anemia), positive fecal occult blood, hematochezia, or melena. We also identified individuals with active bleeding or oozing at the time of colonoscopy.

We classified angiodysplasia according to a classification system proposed by Schmit and colleagues which groups lesions according to location (all colon in this study), size (< 2 mm in diameter, 2 to 5 mm in diameter, > 5 mm in diameter) and number (1, 2 to 10 and > 10)[20].

We used multivariate logistic regression with backward selection to determine independent predictors of each outcome starting with univariate predictors with a significance level of $p < 0.05$. Categories were created for missing variables. To assess the importance of provider characteristics, region and site type were added individually to the final model of treatment. We assessed variables with a univariate significance of < 0.1 as potential confounders. We further examined the relationship between age and comorbid disease given that patients at the extremes of age appeared to be at an increased risk of bleeding (the OR for age 20–39 years was 1.34 but was not statistically significant). We created cross product terms between dichotomized ASA score (< 3 vs. ≥ 3) and each of the age categories, and these terms were added to the multivariable model.

We present the adjusted odds ratios (OR) and 95% confidence intervals (CI). A p value of < 0.05 was considered statistically significant. We performed all analyses using SAS, Version 9.2 (SAS Institute, Cary, NC).

RESULTS

Of the 229,727 colonoscopies performed between January 2000 to December 2002, 4,159 patients (1.8%) were documented to have angiodysplasia. Table 1 summarizes the baseline characteristics and demographics of patients. The majority of patients (73%) were older than 60 years, 55% of patients were male and 86% were Caucasian. Severe comorbid illness (ASA class ≥ III) was present in 16% of cases. Other findings on colonoscopy in addition to angiodysplasia included diverticula (54%), polyps (40%), colitis (4%), and malignancy (0.8%). The majority of colonoscopies in patients with colonic angiodysplasia were performed as outpatients (84%) in community-based practices (69%). Fifteen percent of colonoscopies were performed in a University hospital and 14% in a Veterans Administration (VA) hospital.

Of the 4,159 patients with colonic angiodysplasia, 2,320 (56%) were noted to have evidence of occult or overt blood loss including one or more of the following indications for colonoscopy hematochezia (41%), anemia (37%, including iron deficiency anemia in 2%), positive fecal occult blood test (33%), or melena (7%).

The characteristics of the angiodysplastic lesions are described in Table 2 using a modification of a previously proposed endoscopic classification scheme[20]. Forty-one percent of angiodysplasia were solitary, 30% were multiple (2–10 lesions), and 9% were diffuse (greater than 10 lesions). Half of angiodysplasia were estimated to be intermediate in size ranging from 2 to 5 mm. Twenty percent of lesions were larger than 5mm. Sixty-two percent of angiodysplasia were found in the cecum and ascending colon. Active bleeding from angiodyplasia was reported in 328 cases (7%).

In the multivariable analysis, factors associated with evidence of overt or occult blood loss in patients with colonic angiodysplasia included inpatient status, age greater than 80 years, severe comorbid illness (ASA class ≥ III), black race, Hispanic ethnicity, and multiple or diffuse lesions (Table 3). Forward and backward selection identified the same independent predictors. Multivariable analysis of active bleeding seen at endoscopy identified similar predictors, with the addition of right-sided location (OR, 1.85: 95% CI [1.34, 2.56], p <0.001), and lesion size greater than 5mm (OR, 1.38: 95% CI [1.00, 1.90], p= 0.048). In addition, race and ethnicity were not predictive of active bleeding.

The interaction between ASA score and age 60–79 years was significant in the multivariable model of blood loss (p=0.007) and between ASA score and age >80 years was of borderline significance (p=0.059). We also stratified the analysis by ASA score (< 3 vs. ≥3) and found that in patients with a high ASA score, the relationship between age and bleeding was more linear in nature – patients <40 years of age were not at increased risk of bleeding (OR 0.59: 95%CI [0.15–2.43]), patients 40–59 were at moderate risk (OR 1.66: 95% CI [1.00–2.76]), and patients >80 years were at the highest risk (OR 2.35: 95% CI [1.24–4.43]), although the confidence interval was wide for young patients.

A total of 17% of patients with documented angiodysplasias were treated endoscopically. Endoscopic treatment of angiodysplasia was utilized in 27% of patients with evidence of overt or occult blood loss. Sixty-eight percent of actively bleeding angiodysplasia were treated, as were 47% of angiodysplasia in hospitalized patients with hematochezia. Five percent of patients without an indication of blood loss received endoscopic treatment. The most common treatment modality was thermal coagulation (62%), followed by argon plasma coagulation (17%), injection with epinephrine and/or saline (3.2%), and the use of both injection and thermal coagulation (2.6%). In the multivariable analysis predictors of treatment included patients with evidence of blood loss, active bleeding at the time of endoscopy, young (20–39 years) and very old (> 80 years) age, inpatient status, size > 5 mm, and right-sided location (Table 4). These same independent predictors were identified using a forward selection process. Treatment was more likely to occur at a university (adjusted OR, 2.53: 95% CI [1.96, 3.27], p<.001) than at a community site. Lesions were less likely to be treated in the Northwest (adjusted OR, 0.60: 95% CI [0.43, 0.84], p=0.003) than in the Southwest. Independent predictors of receipt of endoscopic treatment for actively bleeding lesions were size > 5mm (OR, 3.47: 95% CI [1.45, 8.30], p=0.005) and inpatient exam (OR, 4.30: 95% CI [1.56, 11.88], p=0.005). Patients with more than ten lesions were less likely to receive treatment for active bleeding (OR, 0.36: 95% CI [0.15, 0.84], p=0.018).

DISCUSSION

To our knowledge, this is the largest study of patients with angiodysplasia reported to date. Data from over 70 practice sites across the United States provide a unique population-based perspective and enable the analysis of treatment practices.

Of more than 200,000 colonoscopies performed for various indications, 2% of patients were found to have colonic angiodysplasia. This finding confirms that angiodysplasia is a relatively infrequent colonoscopic finding[3–4, 6, 10]. We did not examine angiodysplasia in the context of acute lower intestinal bleeding or in the elderly specifically, but we presume that the prevalence in these select populations would be higher than that seen in all patients undergoing colonoscopy. The highest prevalence of angiodysplasia (40–50%) is reported in studies of patients with severe, ongoing hematochezia[7–9]. As in previous studies, the majority of lesions were located in the right colon, supporting the role of increased wall tension in the pathogenesis of angiodysplasia. In our study, the majority of patients with angiodysplasia were older than 60 years of age. This may reflect the fact that most patients undergoing colonoscopy are over 50 years of age, and we did not have data on individuals without angiodysplasia with which to compare. Nonetheless, our findings are consistent with the literature and suggest that angiodysplasia is an age-related degenerative lesion. Indeed, more than half of patients with angiodysplasia also had diverticulosis, a condition that similarly occurs with aging.

We examined risk factors associated with blood loss in patients with documented angiodysplasia. We recognize that use of NSAIDs or anti-coagulation could promote bleeding from these lesions. In this analysis, we did not have accurate information about drug use. What we report are important patient characteristics which were linked to bleeding. We found that in patients with colonic angiodysplasia, inpatient status, advanced age, comorbid illnesses, black race, Hispanic ethnicity, and the presence of multiple lesions were associated with evidence of blood loss. We suspect that anti-platelet or anticoagulants may be more likely to promote bleeding in these higher risk patients.

The majority of patients with angiodysplasia had concomitant comorbid illness and severe comorbidity, as assessed by ASA class, was a predictor of bleeding. In addition, we found a significant interaction between age and comorbid illness and blood loss suggesting that the interplay of age and illness is important. An increased incidence of angiodysplasia has been described with a number of comorbid illnesses including aortic stenosis, renal failure, von Willebrand's disease, cirrhosis, and pulmonary disease[19]. It remains to be determined whether these illnesses increase the risk of blood loss and thus the detection of angiodysplasia (e.g. via coagulopathy or platelet dysfunction), or whether they are involved in the development of angiodysplasia through mechanisms such as ischemia.

We also noted that patients with multiple (>10) lesions were more likely to have evidence of blood loss and patients with large lesions (>5mm) were more likely to have active bleeding at the time of colonoscopy. Patients with multiple lesions would be expected to have a cumulatively higher risk of blood loss from each lesion. As for the association between larger angiodysplasia and bleeding, we speculate that the local factors that influence the development of these vascular lesions (obstruction of submucosal veins, ischemia, increased wall tension[11–12]) may be even more pronounced in larger lesions and may increase the chance of bleeding. It is also possible that larger or multiple lesions were simply more likely to be seen or recorded by the endoscopist.

This is the first study to suggest a relationship between race and ethnicity and angiodysplasia. These findings may indicate an inherited tendency for angiodysplasia formation or bleeding or perhaps reflect a higher prevalence of other risk factors that were

not accounted for in our adjusted analysis. For example, chronic kidney disease is more prevalent among African American and Hispanic populations[21] and might confer increased bleeding risk via platelet dysfunction or vascular disease. Importantly, we could not account for sociodemographic factors in our analysis. The study of race and disease is complex and deserves further investigation in the context of angiodysplasia.

Our findings suggest that endoscopic treatment of angiodysplasia is inconsistent across regions, practice settings, and clinical scenarios. A total of 27% of patients with any evidence of blood loss and 68% with actively bleeding lesions received endoscopic treatment. Though this may be suggestive of incomplete data acquisition, we speculate that the utilization of endoscopic treatment is an important element of the endoscopy report and is unlikely to be omitted if performed. Furthermore, to account for differences in endoscopic reporting practices, we reviewed all comment or free text fields within the endoscopic reports. This process identified 57 additional patients with active bleeding and 227 who received endoscopic treatment. Nonetheless, our data is limited to the colonoscopic encounter and we may not have accounted for credible reasons for withholding endoscopic therapy such as coagulopathy, or other sources of blood loss (e.g. an upper tract source in a patient also found to have nonbleeding colonic angiodysplasia). However, the latter reason would not account for failure to treat actively bleeding angiodysplasia. Another explanation for the variation in treatment practices could be the lack of conviction among endoscopists that treating isolated angiodysplastic lesions, particularly in patients with chronic anemia, will have significant clinical impact. Angiodysplasia may be present elsewhere in the gastrointestinal tract and tend to recur after treatment. Furthermore, treatment of incidentally noted lesions lacks convincing outcomes data. Endoscopic therapy was less likely to be used in community practice and in the Northwest. Patients with more comorbid illness and thus more likely to have bleeding may be seen preferentially in an academic setting, although the multivariable model adjusted for age, ASA classification and active bleeding. We also speculate that the case loads may be lighter in the academic setting and this may facilitate endoscopic treatment especially when several lesions are present. Overutilization of therapy in the university setting or in the Southwest cannot be excluded. However, we would have expected financial incentives to favor treatment in the community setting. The reasons for regional variation in treatment practices are not clear but may reflect differences in the training of endoscopists and their perceptions as to the utility of treating angiodysplasia.

A primary strength of this study is the large population of more than 4,000 patients with angiodysplasia. Data were gathered on the vast majority of patients from each practice site using the standardized CORI report generator designed for research purposes. As we examined all angiodysplasia and not just those found in colonoscopies performed for acute hematochezia, we are able to broadly describe the epidemiology and risk factors for bleeding in colonic angiodysplasia. The variety of practice settings across the United States also enabled us to evaluate treatment practices.

Our study has several important limitations. As noted above, the retrospective data is limited to the endoscopy encounter and thus specifics such as the presence of comorbid illness (beyond ASA class), contraindications to therapy, potential confounding variables, other sources of blood loss, or previous endoscopic treatment are not available for analysis. These factors likely account in part for the apparent infrequent utilization of endoscopic therapy particularly in patients without active bleeding at the time of colonoscopy. In addition, variation in endoscopic reporting may have led to misclassification, although we accounted for all comment and free text fields. There may also have been a tendency for endoscopists to report the presence of angiodysplasia in those patients with evidence of blood loss as opposed to those undergoing screening colonoscopy. Therefore, the true incidence of angiodysplasia may be higher than reported, whereas the prevalence of blood loss in patients

with angiodysplasia may be overstated. We also rely on the accuracy of endoscopic interpretation as to the presence or absence of angiodyplasia and the size and number of lesions since neither photo nor video confirmation is available in this retrospective analysis. We were unable to assess the prevalence of angiodysplasia in specific populations such as those with acute hematochezia or in specific age groups. In the subanalyses of active bleeding and endoscopic treatment, smaller numbers of outcomes may have affected the stability of the multivariable models. Lastly, the use of aspirin, NSAIDs, or other anticoagulants is an important potential confounder that we were unable to account for in our analysis, and that could alter the associations seen between variables such as age and comorbid disease and bleeding.

In summary, our results show that angiodysplasia is an uncommon finding in all patients undergoing colonoscopy. Older patients with angiodysplasia, and those with comorbid illness or multiple lesions are more likely to have evidence of blood loss. Endoscopic treatment is appropriately reserved for symptomatic patients but treatment of patients with overt or occult bleeding is inconsistent. Variations in endoscopic treatment practices point to the need for prospective studies in order to develop a more standardized approach to the treatment of colonic angiodysplasia.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

1. Hochter W, Weingart J, Kuhner W, et al. Angiodysplasia in the colon and rectum. Endoscopic morphology, localisation and frequency. Endoscopy. 1985; 17:182–5. [PubMed: 3876926]
2. Richter JM, Christensen MR, Kaplan LM, et al. Effectiveness of current technology in the diagnosis and management of lower gastrointestinal hemorrhage. Gastrointest Endosc. 1995; 41:93–8. [PubMed: 7721024]
3. Longstreth GF. Epidemiology and outcome of patients hospitalized with acute lower gastrointestinal hemorrhage: a population-based study. Am J Gastroenterol. 1997; 92:419–24. [PubMed: 9068461]
4. Rockey DC, Koch J, Cello JP, et al. Relative frequency of upper gastrointestinal and colonic lesions in patients with positive fecal occult-blood tests. N Engl J Med. 1998; 339:153–9. [PubMed: 9664091]
5. Heer M, Sulser H, Hany A. Angiodysplasia of the colon: an expression of occlusive vascular disease. Hepatogastroenterology. 1987; 34:127–31. [PubMed: 2440788]
6. Danesh BJ, Spiliadis C, Williams CB, et al. Angiodysplasia–an uncommon cause of colonic bleeding: colonoscopic evaluation of 1,050 patients with rectal bleeding and anaemia. Int J Colorectal Dis. 1987; 2:218–22. [PubMed: 3500991]
7. Jensen DM, Machicado GA. Diagnosis and treatment of severe hematochezia. The role of urgent colonoscopy after purge. Gastroenterology. 1988; 95:1569–74. [PubMed: 3263294]

8. Jensen DM, Machicado GA. Colonoscopy for diagnosis and treatment of severe lower gastrointestinal bleeding. Routine outcomes and cost analysis. Gastrointest Endosc Clin N Am. 1997; 7:477–98. [PubMed: 9177148]
9. Santos JC Jr, Aprilli F, Guimaraes AS, et al. Angiodysplasia of the colon: endoscopic diagnosis and treatment. Br J Surg. 1988; 75:256–8. [PubMed: 3258173]
10. Foutch PG, Rex DK, Lieberman DA. Prevalence and natural history of colonic angiodysplasia among healthy asymptomatic people. Am J Gastroenterol. 1995; 90:564–7. [PubMed: 7717311]
11. Boley SJ, Sammartano R, Adams A, et al. On the nature and etiology of vascular ectasias of the colon. Degenerative lesions of aging. Gastroenterology. 1977; 72:650–60. [PubMed: 300063]
12. Baum S, Athanasoulis CA, Waltman AC, et al. Angiodysplasia of the right colon: a cause of gastrointestinal bleeding. AJR Am J Roentgenol. 1977; 129:789–94. [PubMed: 410241]
13. Richter JM, Hedberg SE, Athanasoulis CA, et al. Angiodysplasia. Clinical presentation and colonoscopic diagnosis. Dig Dis Sci. 1984; 29:481–5. [PubMed: 6609803]
14. Davila RE, Rajan E, Adler DG, et al. ASGE Guideline: the role of endoscopy in the patient with lower-GI bleeding. Gastrointest Endosc. 2005; 62:656–60. [PubMed: 16246674]
15. Olmos JA, Marcolongo M, Pogorelsky V, et al. Long-term outcome of argon plasma ablation therapy for bleeding in 100 consecutive patients with colonic angiodysplasia. Dis Colon Rectum. 2006; 49:1507–16. [PubMed: 17024322]
16. Rutgeerts P, Van Gompel F, Geboes K, et al. Long term results of treatment of vascular malformations of the gastrointestinal tract by neodymium Yag laser photocoagulation. Gut. 1985; 26:586–93. [PubMed: 3874122]
17. Kwan V, Bourke MJ, Williams SJ, et al. Argon plasma coagulation in the management of symptomatic gastrointestinal vascular lesions: experience in 100 consecutive patients with long-term follow-up. Am J Gastroenterol. 2006; 101:58–63. [PubMed: 16405534]
18. Eisen GM, Dominitz JA, Faigel DO, et al. An annotated algorithmic approach to acute lower gastrointestinal bleeding. Gastrointest Endosc. 2001; 53:859–63. [PubMed: 11375618]
19. Sharma R, Gorbien MJ. Angiodysplasia and lower gastrointestinal tract bleeding in elderly patients. Arch Intern Med. 1995; 155:807–12. [PubMed: 7717788]
20. Schmit A, Van Gossum A. Proposal for an endoscopic classification of digestive angiodysplasias for therapeutic trials. The European Club of Enteroscopy. Gastrointest Endosc. 1998; 48:659. [PubMed: 9852467]
21. Collins AJ, RNFaCHea. Excerpts from the United States Renal Data System 2008 annual data report. Am J Kidney Dis. 2009; 53:S1–S374.

Table 1

Baseline Characteristics of Individuals with Colonic Angiodysplasia

Characteristic	Total n= 4159 (%)	Exam Indication	
		Bleeding[a] n= 2320 (%)	No Bleeding n= 1839 (%)
Age (y)			
20–39	104 (2.5)	65 (2.8)	39 (2.1)
40–59	1019 (24.5)	559(24)	460 (25)
60–79	2394(57.6)	1285 (55)	1109 (60)
>80	642 (15)	411(17.7)	231(12.6)
Male	2267 (54.5)	1273 (55)	994 (54)
Race/Ethnicity			
White	3590 (86)	1970 (85)	1620 (88)
Black	244 (6)	171 (7.4)	73 (4)
Hispanic	311 (7.5)	213 (9.2)	98 (5.3)
Other	62 (1.5)	38 (1.6)	24 (1.3)
Unknown	263 (6.3)	141 (6)	122 (6.6)
ASA Class			
I	948 (25)	454 (21)	494 (29)
II	2232 (58)	1196 (56)	1036 (61)
III	605 (16)	442 (21)	164 (10)
IV	36 (0.08)	30 (1.4)	6 (0.35)
Unknown	338 (8)	199 (8.6)	139 (7.6)
Inpatient Exam	266 (6.4)	247 (11)	19 (1)
Exam Site			
Community	2851 (69)	1544 (67)	1307 (71)
University	628 (15)	341 (15)	287 (16)
VA	592 (14)	391 (17)	201 (11)
Other	88 (2)	44 (2)	44 (2)
U.S. Region			
North Central	316 (8)	155 (7)	161 (9)
Northeast	883 (21)	486 (21)	397 (22)
Northwest	510 (12)	303 (13)	207 (11)
South Central	391 (9)	271 (12)	120 (7)
Southeast	526 (13)	315 (14)	211 (11)
Southwest	1532 (37)	790 (34)	742 (40)

[a] Overt or occult bleeding including hematochezia, melena, anemia or positive fecal occult blood.

Table 2

Characteristics of Colonic Angiodysplasia

Characteristic	n (%)[a]
Number	
Unique (1)	1898 (41)
Multiple (2–10)	1376 (30)
Diffuse (>10)	407 (9)
Unknown	935 (20)
Size (mm)	
Minute (<2)	112 (3)
Intermediate (2–5)	2311 (50)
Large (>5)	891 (19)
Unknown	1302 (28)
Location	
Cecum	1789 (40)
Ascending	1002 (22)
Transverse	331 (7)
Descending	1068 (24)
Rectum	278 (6)
Active bleeding	328 (7)

[a]Providers were able to enter multiple lines of data for a single patient. For 4159 unique patients, there were 4616 separate records of angiodysplasia.

Table 3

Multivariable Analysis of Factors Associated With Evidence of Overt or Occult Blood Loss in Patients with Colonic Angiodysplasia

Characteristic	Adjusted OR (95% CI)	*P* value
Inpatient Exam	8.74 (5.42–14.10)	<.0001
Age (y)		
20–39	1.34 (0.87–2.06)	0.18
40–59	1.0	Reference group
60–79	0.92 (0.79–1.07)	0.28
>80	1.32 (1.06–1.63)	0.01
Race		
White	1.0	Reference group
Black	1.95 (1..46–2.62)	<.0001
Other race	1.31 (0.76–2.25)	0.33
Ethnicity		
Not Hispanic	1.0	Reference group
Hispanic	1.71 (1.32–2.22)	<.0001
ASA Class		
I,II	1.0	Reference group
III,IV	1.97 (1.62–2.41)	<.0001
Number		
1	1.0	Reference group
2–10	1.50 (1.29–1.75)	<.0001
>10	2.18 (1.69–2.80)	<.0001

Table 4

Multivariable Analysis of Factors Associated With Endoscopic Treatment of Colonic Angiodysplasia

Characteristic	Adjusted OR (95% CI)	*P* value
Age (y)		
20–39	3.17 (1.88–5.36)	<.0001
40–59	1.0	Reference group
60–79	1.19 (0.93–1.52)	0.18
>80	1.51 (1.11–2.04)	0.008
Size (mm)		
< 2 mm	0.53 (0.27–1.04)	0.063
2–5 mm	1.0	Reference group
> 5 mm	1.88 (1.51–2.35)	<.0001
Location		
Left colon	1.0	Reference group
Transverse	1.04 (0.67–1.61)	0.88
Right colon	1.54 (1.22–1.95)	0.0003
Evidence of bleed[a]	5.36 (4.21–6.83)	<.0001
Active bleeding	12.26 (9.18–16.39)	<.0001
Inpatient	2.52 (1.88–3.37)	<.0001

[a]Overt or occult bleeding including hematochezia, melena, anemia or positive fecal occult blood.