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Geographic Association Between Incidence of Acute Appendicitis and Socioeconomic Status

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IMPORTANCE Some studies based on proportions of patients with perforated appendicitis (PA) among all patients with acute appendicitis (AA) have found an association between socioeconomic status (SES) and risk of perforation. A potential limitation is their use of proportions, which assumes that incidence of AA is evenly distributed across populations at risk. This assumption may be invalid, and SES may have a more complex association with both AA and PA.

OBJECTIVE To generate population-based incidences of AA and PA and to examine geographic patterns of incidence alongside geographic patterns of SES.

DESIGN, SETTING, AND PARTICIPANTS Retrospective study of data from Washington's Comprehensive Hospital Abstract Reporting System and the 2010 US census. Geographic methods were used to identify patterns of age- and sex-standardized incidence in Washington State between 2008 and 2012. The study included all patients discharged with *International Classification of Diseases, Ninth Revision* codes for AA or PA. Data were analyzed between November 2016 and December 2018.

EXPOSURES Location of primary residence.

MAIN OUTCOMES AND MEASURES Age- and sex-standardized incidence for AA and PA was generated for each census tract (CT). Global spatial autocorrelation was examined using Moran index (0.0 = completely random incidence; 1.0 = fully dependent on location). Clusters of low-incidence CTs (*cold spots*) and high-incidence CTs (*hot spots*) were identified for AA. Census-based SES data were aggregated for hot spots and cold spots and then compared.

RESULTS Statewide, over the 5-year study period, there were 35 730 patients with AA (including 9780 cases of PA), of whom 16 574 were women (46.4%). Median age of the cohort was 29 years (IQR, 16-48 years). Statewide incidence of AA and PA was 106 and 29 per 100 000 person-years (PY), respectively. Crude incidence was higher within the male population and peaked at age 10 to 19 years. Age- and sex-standardized incidence of AA demonstrated significant positive spatial autocorrelation (Moran index, 0.30; $P < .001$), but autocorrelation for PA was only half as strong (0.16; $P < .001$). Median incidence of AA was 118.1 per 100 000 PY among hot spots vs 86.2 per 100 000 PY among cold spots ($P < .001$). Socioeconomic status was higher in cold spots vs hot spots: mean proportion of college-educated adults was 56% vs 26% ($P < .001$), and mean per capita income was $44 691 vs $30 027 ($P < .001$).

CONCLUSIONS AND RELEVANCE Age- and sex-standardized incidence of appendicitis is not randomly distributed across geographic subunits, and geographic clustering of AA is twice as strong as PA. Socioeconomic advantages, such as higher income and secondary education, are strongly associated with lower incidence of AA. These findings challenge conventional views that AA occurs randomly and has no predisposing characteristics beyond age/sex. Socioeconomic status, and likely other geographically circumscribed factors, are associated with incidence of AA.

Invited Commentary

Supplemental content

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Many investigators have considered acute appendicitis (AA) a useful model to study disparities in access to high-quality surgical care.[1-3] One study noted that "[AA] is considered an ideal subject of investigation regarding health care disparities because it is characterized by a consistent natural history ... and an absence of known biological predisposition to perforation in any racial or ethnic group."[2] Other investigators have disputed the concept that AA is random in onset and consistent in progression.[4,5] Luckmann[4] raised the provocative "possibility that perforating and nonperforating appendicitis may represent two distinct entities."[4] He used California discharge data from 1984 to generate age-adjusted incidences of appendicitis and found significant age-related variation in the incidence of nonperforating appendicitis but much less variation in the incidence of perforated appendicitis (PA) and abscess. He hypothesized that a high proportion of PA among elderly individuals was associated more with a lower overall incidence of appendicitis than with a higher risk for perforation.[4] Livingston et al[6] examined secular trends from the 1970s to 2000s and showed that the population-based incidences of perforating and nonperforating appendicitis followed different patterns. He suggested that the relatively recent increase in nonperforating appendicitis was owing to early diagnosis of mild appendicitis on computed tomography and lower thresholds to take patients to surgery in the era of laparoscopic appendectomy. This is compared with previous management strategies in which patients with an indeterminate diagnosis of appendicitis would be observed, and, under this hypothesis, some patients with mild appendicitis would resolve with supportive care.[6] Andersson et al[7] evaluated 7 population-based studies and found that the incidence of non-PA varied widely among populations based on aggressiveness of surgical approach to suspected appendicitis, but that the incidence of PA was essentially unchanged. Similarly, Addiss et al[8] noted in 1990 that "differences in surgical practices may also have played a role [in geographic variation], since the regions with the highest rates of appendicitis were generally also highest for negative and incidental appendectomy."[8]

These researchers all used population-based methods to study incidence of AA and rates of perforation. However, most studies of risk factors for perforation have based their findings on proportional methods: generally, the number of cases of PA divided by all cases of AA. Such proportion-based studies have identified disparities in the rate of perforation associated with race/ethnicity,[9,10] insurance status,[1,3,9,11] age,[12] relationships with primary care clinicians,[13-15] rural residence,[16,17] and even hospital resource levels.[14,18] Each of these studies demonstrated an association between markers of reduced access to health care and an increase in proportion of perforation. This association became so accepted that several articles were written demonstrating that improved health care access eliminated disparities in perforation.[19,20]

Two assumptions underlie these proportion-based studies: (1) appendicitis has no predisposing characteristics (other than age and sex) and (2) all cases of appendicitis progress toward perforation without intervention. However, a close reading of the epidemiologic data presented by Luckmann,[4] Andersson et al,[7] Livingston et al,[6] and others suggests that those assumptions should be revisited. We hypothesized that in a statewide data set with near-100% capture of hospital admissions, we could detect patterns in the epidemiology of AA that would clarify the validity of these previous assumptions. Furthermore, we hypothesized that areas of higher socioeconomic status (SES) would have a higher incidence of AA (because of greater access to early diagnosis) but that rates of PA would be more constant. A substantial body of high-quality research has documented the pervasive influence of economic, racial/ethnic, sex, and other social determinants on health care outcomes. The purpose of this study is not to dispute whether such disparities exist in AA, but to clarify more accurately how they may influence outcomes.

Key Points

Question Is the population-based incidence of acute appendicitis (AA) or perforated appendicitis (PA), at the census tract level, uniformly distributed across a densely populated geographic area?

Findings In this study, AA and PA were clustered geographically into high-incidence and low-incidence regions, but geographic autocorrelation was twice as strong for AA compared with PA. Areas of low AA incidence have significantly increased secondary education and income vs high-incidence areas.

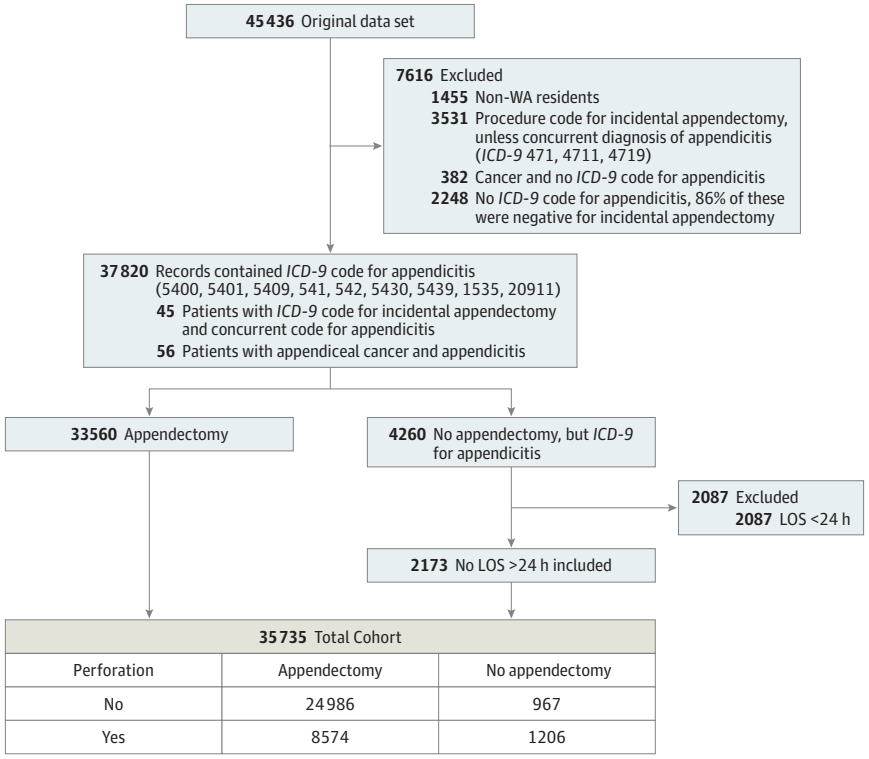
Meaning These findings challenge the conventional view that AA occurs randomly and has no predisposing characteristics beyond age/sex; higher socioeconomic status is associated with lower incidence of both AA and PA.

Methods

Generating the Washington State Comprehensive Hospital Abstract Reporting System Appendicitis Data Set

We obtained data from the Washington State Comprehensive Hospital Abstract Reporting System (CHARS) for 5 years, 2008 to 2012, chosen because of temporal proximity to the 2010 US census. Some population-based studies have used procedure codes for appendectomy to identify patients with AA,[21] but not all patients with appendicitis undergo operations at their index admission, in particular, those with advanced disease such as abscess or phlegmon. Therefore, we queried CHARS using *International Classification of Diseases, Ninth Revision (ICD-9)* diagnosis codes in addition to procedure codes (eFigure 1 in the Supplement). We began with all patients with diagnosis codes for AA or procedure codes for appendectomy (**Figure 1**). Non-Washington residents and patients coded for incidental appendectomy were excluded. The *ICD-9* codes for appendiceal cancer are 153.5 and 209.11, and these patients were included only if they also had a code for AA. We excluded patients who had no *ICD-9* code for AA but underwent a nonincidental appendectomy. These patients make up the "negative appendectomy" segment of the overall appendectomy population (eTable 1 in the Supplement). Perforated appendicitis was defined as an *ICD-9* code of 540.0 or 540.1. In this article, AA includes all cases of appendicitis, including nonperforated and perforated; moreover, PA includes all

Figure 1. Study Flowchart of Inclusion and Exclusion Criteria



Perforation	Appendectomy	No appendectomy
No	24 986	967
Yes	8574	1206

This figure illustrates how the initial data set of 45 436 patients was narrowed to a study cohort of 35 735 patients, as well as their designation as cases of perforated appendicitis or acute appendicitis. *ICD-9* indicates *International Classification of Diseases, Ninth Revision (ICD-9)* and LOS, length of stay.

instances of complicated appendicitis including perforation, abscess, and inoperable phlegmon.

Including patients who had an appendicitis *ICD-9* code but not an appendectomy risked including patients who were admitted with possible appendicitis but ultimately diagnosed as having something else. However, to keep patients in the data set who had complicated appendicitis treated nonoperatively, we could not rely on lack of appendectomy as an exclusion criteria. Therefore, we excluded patients who had an *ICD-9* code for AA but did not have an appendectomy and were discharged within 24 hours. These patients were unlikely to have AA (and certainly not PA) because this data set is from well before antibiotics-only treatment of uncomplicated appendicitis was common.

Generating Geographic Data

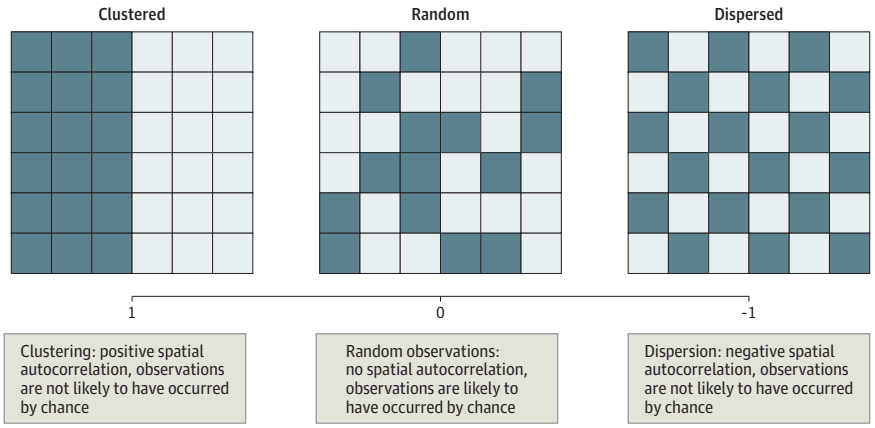
The CHARS data set includes patient zip code but not home address. Zip codes are not ideal to spatially analyze epidemiologic data because they are based on linear postage routes and not neighborhoods.[22] Thus, we chose to examine incidence at the census tract (CT) level. Census tracts contain greater statistical accuracy for measuring populations because they are relatively permanent and more representative of neighborhoods. Because zip codes are larger than CTs, data must be disaggregated from zip codes into CTs. We used a Census Bureau–approved method to systematically allocate patients within zip codes to CTs based on percentages of occupied residential housing units per CT.

Substantial rural portions of Washington are too sparsely populated for incidence to be reliably calculated. The National Center for Health Statistics does not publish rates if the absolute number of cases is less than 20.[23] Thus, although we generated statewide data, detailed analysis of geographic incidence patterns and SES was performed only within the 3 counties immediately adjacent to the Puget Sound region (King, Snohomish, and Pierce), hereafter referred to as the tricounty area. This area contains 51% of Washington's population, with just more than 3.6 million residents, and represents a racially and socioeconomically diverse urban and dense suburban population. If a tricounty resident had appendicitis care at a hospital in another county, data for that patient would be correctly designated to the county of residence. Furthermore, none of these counties are on a state line, making it unlikely a tricounty resident would travel out of state for AA care.

Age- and Sex-Standardized Incidence

Several articles have demonstrated age and sex differences in the incidence of AA.[5,8,24] There is substantial age variation across the tricounty Puget Sound area, with several university districts and certain neighborhoods with higher proportions of elderly residents. Thus, we performed direct age and sex standardization using the overall state population as the standard population. Standardization was performed at zip code level prior to disaggregation.

Figure 2. Illustration of Moran Index for Spatial Autocorrelation



This figure illustrates the range of possible results for Moran index for Spatial Autocorrelation and how these values (from −1 to 1) are interpreted.

Geographic Analysis

Incidences of AA and PA were first examined using the Moran index to determine whether the spatial distribution of incidence was owing to chance. The output of Moran index is a value between −1.0 (dispersion) and 1.0 (clustering), and 0 indicates a random distribution (**Figure 2**). Because geospatial distribution was not random, we then proceeded to perform a Getis-Ord Gi* analysis. This is a regression-based technique to assess whether the incidence in one CT is influenced by nearby CTs at a particular confidence level. We identified groups of high-incidence CTs that are spatially related or clustered (designated as *hot spots*) and groups of clustered low-incidence CTs (*cold spots*).

Socioeconomic Status and Incidence of AA

Socioeconomic status data for each CT were taken from the 2010 census. Socioeconomic status data were aggregated for hot spots and cold spots. All data on race/ethnicity are reported as designated by the US Census Bureau. Importantly, percentages of race/ethnicity are aggregate variables per CT (as are all SES variables reported in this study); these variables are not based on patient data reported in the CHARS data set. Means were compared via *t* tests, with statistical significance set at a 2-sided *P* value greater than .05. The analytic plan and reporting of results for this study accord with the STROBE statement.[25]

Human Participants Review

Human participants review was waived by the San Francisco State University institutional review board because the data set provided by the Washington Department of Health contains no direct identifiers.

Sensitivity Analysis

To ensure that our results were not confounded by race/ethnicity, we used multivariable linear regression to study the association between incidence of AA/PA and SES variables across all CTs in the study area (ie, not just hot spot vs cold spot CTs). Based on prior work, census variables were included in the model if they had a univariate association with incidence of AA or PA at a significance level *P* of less than .05, which included race/ethnicity, median home value, median income, and percentage bachelor's degree. All *P* values were 2-sided. Not all census categories of race/ethnicity had a univariate association with incidence, but because some did, all were included in the multivariable model.

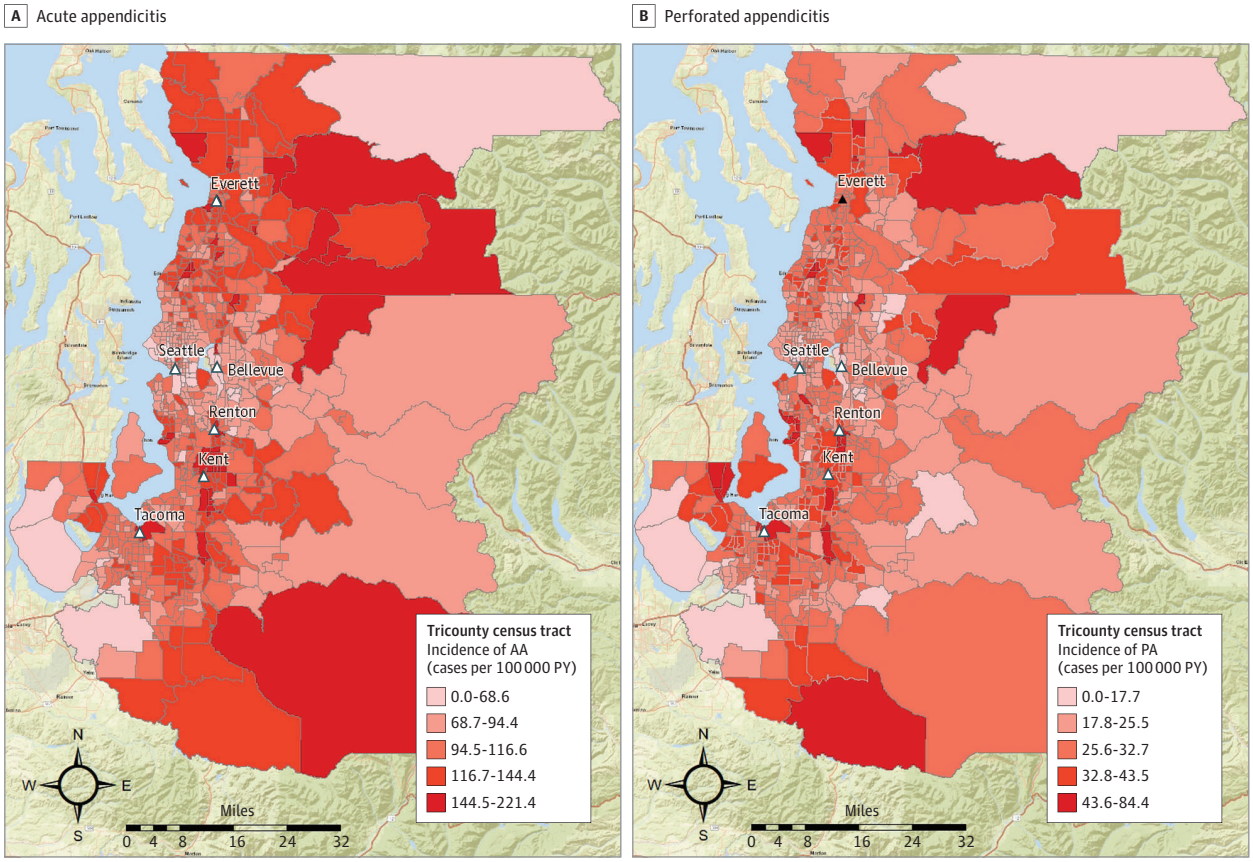
Results

Incidence of AA across Washington State was 106.0 cases per 100 000 person-years (PY) and was 102.4 per 100 000 PY for the tricounty Puget Sound area. For PA, this was 29.0 and 28.2 per 100 000 PY, respectively. Both AA and PA were more common within the male population compared with the female population, with respective incidences of 114.0 and 31.4 per 100 000 PY (male population) and 94.3 and 24.5 per 100 000 PY (female population). For both sexes, the age decile with the highest incidence of AA was 10 to 19 years; children 9 years and younger had the lowest incidence (eTable 2 in the Supplement).

Incidence of AA and PA both demonstrated significant nonrandom spatial variability (**Figure 3**); however, the maps depict a higher degree of geographic autocorrelation for AA compared with PA, a finding that was determined statistically using Moran index: AA = 0.30 ($P < .001$) and PA = 0.16 ($P < .001$). Geographic clustering was twice as robust for AA compared with PA, with a less than 1% chance that these spatial distributions were owing to chance. Getis-Ord Gi* analysis identified 235 CTs within cold spots and 234 CTs within hot spots at the 99% confidence level (**Figure 4**).

Census tracts with high AA incidence were concentrated in the northern and southern ends of the study area (ie, Tacoma and Everett). The Seattle and Bellevue metropolitan areas had the lowest incidence. Perforated appendicitis incidence reflects a similar nonrandom pattern of distribution, although overall incidence is more homogenous. Median standardized incidence of AA was 86.2 per 100 000 PY among cold spots and 118.1 per 100 000 PY among hot spots. For PA, incidence between cold and hot spots was 24.4 per 100 000 PY

Figure 3. Incidence of Acute Appendicitis (AA) and Perforated Appendicitis (PA) in the Tricounty Puget Sound Region



This figure depicts the population-based incidence of AA and of PA in the study area. The Moran index of 0.30 ($P < .001$) for AA and of 0.16 ($P < .001$) for PA indicate a statistically significant nonrandom geographic distribution of incidence for both conditions. The strength of the association between geographic location and incidence is twice is strong for AA as for PA.

and 29.5 per 100 000 PY, respectively. This incidence gap (the proportional difference from cold spots to hot spots) was almost double for AA compared with PA (21% for PA vs 37% for AA). (**Table**; eFigure 2 in the Supplement).

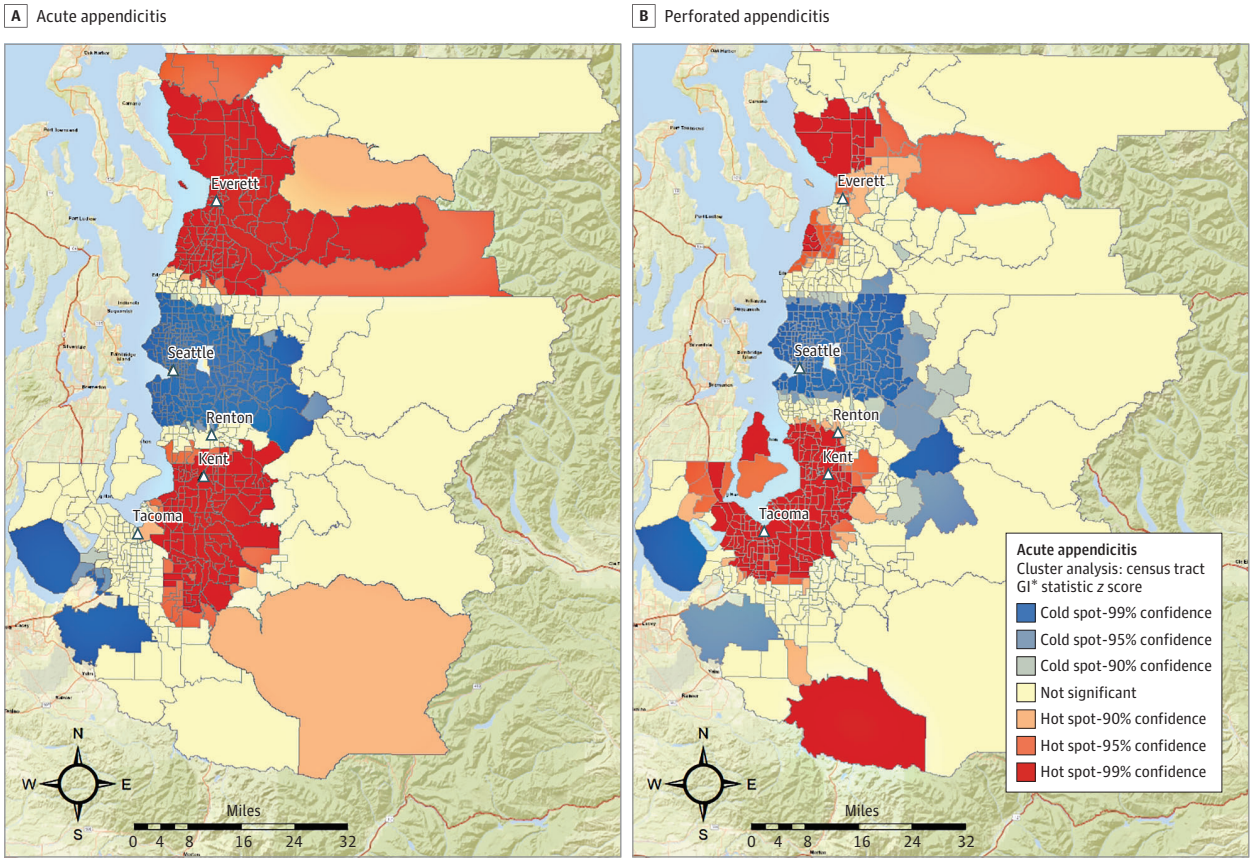
Finally, we compared socioeconomic characteristics of AA hot spots vs cold spots. Socioeconomic status markers, such as mean proportion of adults with a college education (56% vs 26%, $P < .001$), mean median income ($79 841 vs $70 110; $P < .001$), and mean per capita income ($44 691.62 vs $30 027.60; $P < .001$), were substantially higher in cold spots compared with hot spots. Other SES markers, including rates of high school graduation, employment, and public assistance, were also significantly different, although to a lesser degree, and followed a similar pattern: areas of higher SES had lower incidence of both AA and PA. Racial and ethnic differences were statistically significant, likely because of the large data set, but the differences were small and provide somewhat of a mixed picture. More affluent, low-incidence CTs (cold spots) had more African American residents but fewer Latino residents; they had a higher nonwhite population and more residents not born in the United States (Table).

After adjustments via our multivariable linear regression model, increasing percentage of bachelor's degrees within CTs was independently associated with reduced incidence of both AA and PA. Increasing proportion of black race/ethnicity was also associated with reduced incidence of AA, but the other race/ethnicity variables had no association with incidence. The AA model had an adjusted R^2 value of 0.20. For PA, in addition to bachelor's degrees, increases in median income and proportion of black and Latino residents were all associated with decreases in incidence of PA. The PA model had an adjusted R^2 value of 0.11 (eTable 3 in the Supplement). Therefore, approximately 20% of the variation in incidence of AA but only 11% of the variation in incidence of PA across the entire study area was explainable by the SES and race/ethnicity variables included in our 2 models.

Discussion

This analysis identified significant geographic clustering in the incidence of AA, a disease thought to be random in onset, and weaker, although still present, geographic clustering in PA, a disease thought to have substantial associations with SES and other social determinants of health. Consistent with the observation that PA incidence was more stable across regions, the

Figure 4. Getis-Ord Gi* Clustering Analysis of Hot Spots and Cold Spots of Incidence of Acute Appendicitis (AA) and Perforated Appendicitis (PA)



Getis-Ord Gi* is a regression-based geographic method that identifies clusters of spatially associated geographic subunits such as census tracts at 90, 95, and 99 percent confidence levels. A, Analysis for AA and identifies hot and cold spots for age- and sex-standardized incidence of AA. B, Analysis for PA and identifies hot and cold spots for age- and sex-standardized incidence of PA.

incidence rate ratio of PA to AA was higher within cold spots for AA. Put another way: lower incidence of overall appendicitis, even among affluent CTs, did not result in proportionally lower incidence of PA. Moreover, contrary to our hypothesis, the incidence of AA was not higher in regions of higher SES. In fact, college education and income were substantially higher in areas of low incidence. This analysis focused particularly on education and income because these 2 parameters plus occupation are considered the chief components of overall SES.[26-28] Indeed, some public health investigators consider education to be the best single marker of SES as it relates to health,[26,29] and the difference in college education between AA hot spots and cold spots was especially large at 26% vs 56%. As with higher education, per capita income was substantially higher in cold spots vs hot spots (nearly 50% higher). In fact, the only income/wealth variable that did not follow the trend was home ownership, which was 10% higher in hot spots, and we suspect this is associated with the extremely expensive real estate market in Seattle and a young, tech-based work force that is more likely to rent than buy (this is supported by the small but significant difference in rent-to-income ratio, suggesting that cold spot renters pay less of their income in rent than those who rent in hot spots). In summary, essentially all of the education and income data support the conclusion that areas with low AA incidence are more educated and wealthier compared with those with high incidence. In a multivariable model of incidence over the entire study area, secondary education remained independently and robustly associated with incidence after adjustments for race/ethnicity and income.

Previous studies have identified educational attainment as a potent predictor of health outcomes,[26,27,29-31] and similar to our own study, other investigators have demonstrated associations between geography, SES, and health in the Puget Sound region.[32] These have suggested that geography can function as its own marker of SES, with direct, measurable associations between where populations live and their health and health care outcomes.[33] Although our study detected an unexpected association between the incidence of AA and SES, it is not the first to do so. A 2018 Canadian study[17] (notably, in the setting of universal health care) used a similar geographic method to ours (although proportional, not population-based rates were calculated) and reported that high SES was associated with nearly twice the odds of perforation compared with the lowest quartile of SES. Our finding that AA has an age- and sex-standardized incidence with such significant, nonrandom geographic variation is a novel and important observation,

Table. Comparing Incidence and Socioeconomic Factors Between High-Incidence and Low-Incidence Census Tract Clusters[a]

Variable	Census Tracts		*P* Value
	Low Incidence (235 Observations)	High Incidence (234 Observations)	
Mean incidence/person-years (crude)			
AA	83.0/100 000	120.2/100 000	<.001
PA	24.7/100 000	30.2/100 000	<.001
Mean rate ratio	29.9	25.1	<.001
Standardized incidence/person-years, mean (SD)			
AA	87.8/100 000 (19.1)	120.8/100 000 (21.6)	<.001
PA	25.3/100 000 (6.5)	30.7/100 000 (6.4)	<.001
Mean rate ratio	29.1	25.6	<.001
Standardized incidence/person-years, median (IQR), y			
AA	86.2/100 000 (76.2-98.7)	118.1/100 000 (104.9-132.1)	<.001
PA	24.4/100 000 (21.3-28.3)	29.5/100 000 (26.4-33.7)	<.001
Socioeconomic indicators			
Average %			
High school diploma	93.4	89.9	<.001
Bachelor's degree	55.8	25.6	<.001
Median income, $	79 841.73	70 110.62	<.001
Per capita income, $	44 691.62	30 027.60	<.001
Employed	66.1	62.5	<.001
Public assistance	27.3	39.4	<.001
Non-Hispanic white	66.5	70.6	.008
Black	5.6	3.8	.001
Home ownership	57.5	67.4	<.001
Rent-to-income ratio	29.0	30.9	.001
Latino	6.5	10.2	<.001
Married	49.3	54.4	<.001
Non-US born	20.6	14.6	<.001

Abbreviations: AA, acute appendicitis; IQR, interquartile range; PA, perforated appendicitis.

[a] Both a mean and median standardized incidence was calculated for hot spot census tracts and cold spot census tracts (See also eFigure 2 in the Supplement for medians and IQRs). Means are compared via *t* test and medians via the Mann-Whitney test.

and the association between incidence and SES requires validation in other populations along with investigations to ascertain mechanisms.

Our overall estimations of incidence (106 and 29 per 100 000 PY for AA and PA, respectively) are in line with the existing literature. Addis et al[8] evaluated 15 years of the National Hospital Discharge Survey (United States, 1970-1984) and calculated an overall incidence of AA of 110 per 100 000 PY[8] in a study that also detected regional differences. Luckmann's[4] 1984 data (California), with an age- and sex-adjusted incidence of 99.9 per 100 000 PY, were consistent with the temporal decrease observed in the Addis et al article.[8] Ilves et al[24] (Finland) also showed a decrease from 145 per 100 000 PY in 1987 to 98 per 100 000 PY in 2007,[24] the latter number quite similar to our tricounty incidence. Lee et al[19] (South Korea, 2005-2007) calculated an incidence of PA (29.1 per 100 000 PY) that is identical to our data.

These similarities provide external validity to our methods for calculating incidence, which strengthens our finding of such variation within the highly-populated tricounty study area. With the prevailing assumption that AA is random in onset, one would expect a more homogenous distribution across the state. Not only is there variability, but hot spot and cold spot CTs were located in statistically significant clusters. There are several possibilities for why that might be: environmental factors that increase susceptibility, infectious processes that are geographically circumscribed, microbiome differences, socioeconomic factors, health care–seeking behaviors, or practice patterns at nearby hospitals. Likely, these differences in incidence are multifactorial.

Regarding health care–seeking behaviors, Luckmann[4] proposed that some observed trends among hospitalized patients with AA could be explained by differences in the number of patients who present with non-PA rather than differences among those with PA. If some patients with mild or self-limiting AA get better on their own and do not present to the hospital, evaluating proportions of PA among all patients with AA who do present to the hospital may generate misleading conclusions (assuming that all or most patients with PA become sick enough to require hospitalization).[4] Inherent in this hypothesis is the notion that appendicitis can onset in more or less severe forms, and data are being accumulated to support this view, not only in terms of appendicitis that is successfully treated with antibiotics but also treated without antibiotics.[34] Fitz,[35] in the original description of appendicitis, indicated that up to one-third of the appendices in

autopsy specimens showed evidence of prior inflammation. The disease appendicitis is almost certainly more complex than an obstructive process that progresses inexorably to perforation, and the ways in which socioeconomic factors influence both presentation and outcomes also appear to be more complex than a simple association between diminished health care access and increased risk of perforation.[36] This population-based geographic analysis suggests that a more nuanced understanding of pathophysiology and the influence of SES is necessary to fully understand risk factors for both perforating and nonperforating appendicitis.

Limitations

This observational study has some limitations. Data sets based on discharge diagnoses can have misclassification. We designed our analysis to accommodate for this, but it is likely that some mischaracterization persists in the data set. Patients cared for at the Veterans Administration Medical Center or a military hospital in the state are not included, but prior research suggests this is only 0.5% of the cases of appendicitis in the state.[21] Anyone traveling out of state when they had appendicitis would not be included, slightly reducing our calculated incidence. Disaggregation from zip codes to CTs has limitations, and an ideal data set would provide street addresses so that disaggregation was unnecessary. Finally, our study population, residents of Washington State and the tricounty Puget Sound, have high rates of insurance coverage and access to health care. Given Luckmann's hypothesis[4] that some patients with mild or self-limiting appendicitis may not present to the hospital, and given the traditional hypothesis that some patients delay presentation because of inadequate insurance coverage, variations in insurance status within a population may be associated with incidence of AA or PA. Thus, if this study was to be repeated in a population with less insurance coverage, the results might differ.

Conclusions

In summary, population-based incidence of AA is not randomly or uniformly spread across geographic space. Not only is there wide variability in incidence, areas of high incidence and low incidence are geographically clustered. Areas with low incidence of AA are more prosperous and more educated compared to areas with high incidence. Incidence of PA was also nonrandomly distributed, but the measurable association between geography and PA was only half as strong compared with AA. Although most studies have focused on associations between SES and rates of PA, our data suggest that associations between SES and incidence of overall AA may be equally or even more relevant. This population-based analysis also suggests that using proportions of PA of all cases of AA as a marker of access to high-quality surgical care is not appropriate. Our understanding of the pathophysiology of appendicitis and what pathways may lead to perforation or resolution is limited, but future investigators should prioritize elucidating the biologic, behavioral, and socioeconomic factors that drive the epidemiologic patterns we have detected.

ARTICLE INFORMATION

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Concept and design: Golz, Flum, Liu, Donovan, Drake.
Acquisition, analysis, or interpretation of data: Golz, Sanchez, Liu, Donovan, Drake.
Drafting of the manuscript: Golz, Flum, Liu, Donovan, Drake.
Critical revision of the manuscript for important intellectual content: Golz, Sanchez, Liu, Donovan, Drake.
Statistical analysis: Golz, Liu, Drake.
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