Diabetes and Its Effect on Abdominal Aortic Aneurysm Growth Rate in Hispanic Patients

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Background: The growth rate of abdominal aortic aneurysms (AAA) can vary depending on age, baseline diameter, blood pressure, race, and history of smoking. Paradoxically, previous studies show evidence of a protective effect of diabetes on the rate of AAA expansion despite its well-established role in the morbidity and mortality of cardiovascular disease. This study aims to investigate the impact diabetes plays on AAA growth within a Hispanic population.

Methods: Data were collected from patients who were predominantly Mexican-American at a single hospital site. Baseline and follow-up measures for AAA diameter were obtained from serial imaging studies. Demographics, medical history, the presence of type 2 diabetes, and medication use were extracted from hospital records. Linear mixed-effects growth models were used to calculate the overall AAA growth rate and to assess the difference in AAA growth rate between demographics, comorbidities, and medication use.

Results: The study comprised 201 patients (70.4% male) with a mean baseline age of 79.1 years, of whom 43.2% were diabetic. The average monthly AAA growth rate across all study participants was 0.15 mm (SE = 0.02 mm). Independently, the average AAA expansion rate for the diabetic and nondiabetic groups was 0.07 mm (SE = 0.04 mm) and 0.21 mm (SE = 0.03 mm) per month, respectively. This demonstrates a 65% lower linear AAA expansion rate per month in patients with diabetes.

Conclusions: This study confirms a difference of AAA physiology between diabetics and nondiabetics in the Hispanic community. The observed significant difference in AAA growth rate may be a combination of factors associated with race/ethnicity, prevalence of diabetes mellitus, and low compliance with diabetic control exhibited in the Mexican-American population.

INTRODUCTION

It is estimated that 11,000 Americans died from an abdominal aortic aneurysm (AAA) in 2014.[1] Risk factors associated with AAA formation include male gender, individuals older than 65 years, white race, family history of the disorder, obesity, hypertension, hyperlipidemia, and smoking.[2] The expansion rate of AAAs can vary depending on baseline diameter, current smoking, and diastolic blood pressure.[3] Recently, it has been observed that diabetes mellitus, despite its many

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cardiovascular complications, is inversely associated with AAA expansion.[4–7] In 2008, Golledge et al. found that diabetic patients with an AAA had an average growth rate of 0.63 mm per year in diameter compared with 1.2 mm per year in nondiabetic patients.[8]

Although the protective effect of diabetes on AAA growth rate has been demonstrated, differences in expansion rates between diabetics and nondiabetics seem to fluctuate greatly, from −0.596 mm[9] to −1.2 mm per year for patients with diabetes,[10] thereby making it difficult to definitively characterize AAA growth in diabetics. Furthermore, some studies have shown that medications used to treat diabetes mellitus have attenuated the development and growth rate of AAAs.[11,12]

Despite a greater propensity for acquiring risk factors, the Hispanic population experiences lower rates of morbidity and mortality from cardiovascular diseases compared with the general population.[13] With respect to AAAs, however, Hispanics are more likely than non-Hispanic whites (NHWs) to suffer ruptured AAAs and experience a higher likelihood of mortality when ruptured.[14] Because this condition is usually asymptomatic and death from rupture is 80–90%,[15] elucidating the progression of AAAs has significant implications in detection, surveillance, and intervention of a potentially fatal condition.

The purpose of this study is to examine the protective role diabetes plays on AAA within the Hispanic population using retrospective longitudinal data from patients from a single hospital site.

METHODS

The study was approved by the University of Texas Rio Grande Valley Institutional Review Board. The study population consisted of patients selected from a single hospital site. All patients in the hospital medical record system with a history of an AAA using the International Classification of Diseases, Ninth Revision diagnosis code 4414 were screened for inclusion into the study. The inclusion criteria included Hispanic ethnicity, history of AAA, and least 2 preoperative measurements of the AAA at different time points. All scans were performed in the department of radiology.

An AAA was defined as dilatation of the abdominal aorta exceeding the normal vessel diameter by 50% or initial external cross-sectional measurement in any plane greater than 3.0 cm^2. The primary outcome variable of interest was the AAA growth rate.

Baseline and follow-up measures for AAA diameter were obtained from serial imaging studies. For each patient, the maximum transverse and anteroposterior diameter of the AAA was extracted from the radiology report. AAA diameter and time in months between the imaging studies were collected for all subsequent imaging studies for each patient. All imaging studies before surgical intervention were included in the analysis, regardless of the number and time between studies. None of the measurements were excluded based on size as long they met the initial diagnosis requirement and AAA definition.

The predictor variable for this analysis was the presence of diabetes mellitus type 2. Diabetes was defined as a fasting blood glucose $\geq$126 mg/dl, treatment by a physician for diabetes with oral agents and/or insulin, or history of diabetes mellitus noted in the patient medical record.

Other variables analyzed in this study were demographic characteristics and comorbidities that have been referenced in previous studies as potential factors associated with aneurysm growth such as hypertension, hyperlipidemia, current smoking, history of coronary artery disease (CAD), history of cerebral vascular accidents (CVAs), chronic kidney disease (CKD), chronic obstructive pulmonary disease, peripheral artery disease, and medication use.

Statistical Analysis

Univariate and bivariate analyses were conducted to describe and compare the distribution of basic demographics, baseline comorbidity, and medication use characteristics of the study data by diabetes and nondiabetes groups. Continuous variables were described with mean and standard deviation, and categorical variables were described using frequency and percentages. The analyses of baseline data were conducted using Pearson's chi-square test or Fisher's exact test for categorical variables and Student's *t*-test for continuous variables.

To account for repeated AAA measures over time, we fitted linear mixed-effects models for AAA diameter growth with a subject-specific random intercept and a subject-specific random slope for time with unstructured covariance matrix using SAS PROC MIXED.[16] The linear mixed-effects model handles unbalanced data, directly models the covariance structure, and provides valid standard errors and efficient statistical tests.[17] The participants' follow-up time was measured in months, and it was analyzed as a continuous variable. Different covariance structures, accounting for the correlation between the

Table I. Baseline demographic, comorbidity, and medication use characteristics of the study population

Characteristics	All $n = 125$	Diabetes $n = 54$	No diabetes $n = 71$	P value
Age (years)	79.1 (11.3)	79.3 (8.4)	78.5 (13.7)	0.7052
AAA diameter (mm)	38.7 (10.53)	40.2 (12.07)	38.1 (9.71)	0.2695
Male	88 (70.4)	41 (75.9)	47 (66.2)	0.2379
Female	37 (29.6)	13 (24.1)	24 (33.8)	
Comorbidities				
Hypertension	113 (91.1)	50 (94.3)	63 (88.7)	0.2773
Coronary artery disease	81 (65.3)	38 (71.7)	43 (60.6)	0.1975
Hyperlipidemia	94 (75.8)	44 (83.0)	50 (70.4)	0.1052
Cerebral vascular accident	22 (17.9)	12 (23.1)	10 (114.1)	0.1986
Chronic kidney disease	51 (41.5)	25 (47.2)	26 (37.1)	0.2637
Chronic obstructive pulmonary disease	37 (30.1)	14 (2,649)	23 (32.9)	0.4404
Peripheral artery disease	42 (34.2)	20 (38.5)	22 (31.0)	0.3878
History of smoking	64 (52.0)	22 (42.3)	42 (59.2)	0.0647
Medication use				
Metformin	14 (12.1)	13 (26.0)	1 (1.5)	**< 0.0001**
Insulin	10 (8.6)	10 (20.0)	0 (0)	**0.0001**
Sulfonyl	7 (6.0)	7 (14.0)	0 (0)	**0.0021**
Statin	68 (58.6)	35 (70.0)	33 (50.0)	**0.0303**
Beta-blocker	68 (58.6)	34 (68.0)	34 (51.5)	0.0884
Calcium channel blocker	27 (23.5)	13 (26.0)	14 (21.5)	0.5758
ACE inhibitor	52 (45.2)	24 (49.0)	28 (42.4)	0.4849

Bold values are statistically significant.

repeated measures and the random effects, were explored and selected using restricted maximum likelihood (REML).[17] Multicollinearity and interaction effects between the covariates included in the models were assessed during the model building process. In the analyses, while controlling for the effect of person-level covariates, we were interested to test for changes in AAA diameter over time; to test for the overall differences in AAA diameter between diabetes and other patient characteristics' defined groups, such as hypertension, hyperlipidemia, and smoking status; and to assess the interaction between these characteristics and time to evaluate differences in AAA growth rate between the characteristics' defined groups over time. Using maximum likelihood estimation methods, the most parsimonious model was selected based on the smallest value of Akaike's Information Criterion (AIC).[18] Final model parameters were estimated using REML estimators. All statistical analyses were conducted using SAS 9.4 (SAS Institute, Cary, NC). All statistical testing was two-sided and was performed using a significance (alpha) level of 0.05.

RESULTS

In total, 201 patients were included in the final retrospective analysis after screening 2,000 patient records with documented AAA by ICD-10 code. After exclusion of 61 patients with only one AAA diameter measurement and 15 patients with missing type 2 diabetes status, a total of 347 AAA diameter measurements were taken on 125 patients between November 2006 and August 2016.

In the study population, 88 (70.4%) were male and 37 (29.6%) were female, with a mean baseline age of 79.1 years (SE = 11.30) (Table I). At the baseline assessment, there were 54 (43.2%) diabetic and 71 (56.8%) nondiabetic patients. The mean AAA diameter at initial measurement was 38.7 mm (SE = 10.53 mm). There was no significant difference in age, sex, hypertension, CAD, hyperlipidemia, CVA, CKD, smoking history, beta-blocker use, calcium channel blocker use, or use of angiotensin-converting enzyme (ACE) inhibitors between diabetic and nondiabetic patients (Table I).

Table II. Parameter estimates from linear mixed-effects model for AAA growth ($n = 325$)

Fixed effects	Estimate	Standard error	Pr > |t|
Intercept	−2.19	1.71	0.20
Time (months)	0.21	0.03	<0.0001
Type 2 diabetes	0.20	0.45	0.6574
No type 2 diabetes	Reference		
Time*type 2 diabetes	−0.14	0.05	0.0035
Time*no type 2 diabetes	Reference		
Baseline age (years)	0.01	0.02	0.4280
Female	−0.06	0.47	0.9002
Male	Reference		
Maximum transverse diameter (cm)	10.20	0.20	<0.0001
Hyperlipidemia	1.55	0.58	0.0088
No hyperlipidemia	Reference		
Hypertension	−0.42	0.86	0.6247
No hypertension	Reference		
Smoke	0.36	0.43	0.3989
Do not smoke	Reference		
Cerebral vascular accident	0.24	0.46	0.6069
No cerebral vascular accident	Reference		
Statin medication use	−0.58	0.4459	0.2005
No statin medication use	Reference		
ACE inhibitor	−0.38	0.44	0.3815
No ACE inhibitor	Reference		
Sulfonyl medication use	−0.18	0.94	0.8474
Sulfonyl medication use	Reference		
AAA growth rate estimates based on the interaction effect type 2 diabetes*time			
Growth rate for diabetics	0.07	0.04	<0.0001
Growth rate for nondiabetics	0.21	0.03	0.0486

The average monthly AAA growth rate across all study participants, estimated based on the unconditional linear mixed-effects growth model, was 0.15 mm (SE = 0.02 mm). In a multivariable linear mixed-effects growth model using person-level covariates, the estimated average monthly AAA growth rate was 0.21 mm (SE = 0.03 mm) while controlling for the effect of diabetes status, the interaction between diabetes and time variable, sex, baseline age, baseline measure of AAA diameter, hyperlipidemia, hypertension, smoking status, CVA, ACE inhibitor, statin, and sulfonyl medications (Table II). In the overall study population, there was no significant difference in mean AAA diameter between the diabetes and nondiabetes groups. However, the interaction between time and diabetes groups variables was statistically significant ($P = 0.0035$), indicating that there was a significant decrease in monthly AAA growth rate of −0.14 mm (SE = 0.05 mm) in the diabetes groups over time (Table II). In particular, the average AAA expansion rate for the diabetic group was 0.07 mm (SE = 0.04 mm) per month or 0.84 mm per year and the mean expansion rate for the nondiabetic group was 0.21 mm (SE = 0.03 mm) per month or 2.5 mm per year as shown in Table II.

Table III shows the estimated AAA growth rate in mm/month according to patient risk factors and comorbidities. There was no significant crude difference in AAA growth rate between smokers and nonsmokers ($P = 0.0814$). However, after controlling for the effect of diabetes status, sex, baseline age, baseline measure of AAA diameter, hyperlipidemia, hypertension, CVA, ACE inhibitor, statin, and sulfonyl medications, the difference in AAA monthly growth diameter between smokers and nonsmokers was 0.10 mm (0.01 mm, 0.19 mm) ($P = 0.0417$) (Table III).

DISCUSSION

The subjects in this study are from the Rio Grande Valley (RGV), an area along the Texas-Mexico border at the southernmost tip of Texas that has a population of 1.4 million. Most residents are

Table III. Analysis of AAA growth rate (mm/month) according to risk factors and comorbidities

	Mean crude difference in growth rate (mm/month)		Mean adjusted[a] difference in growth rate (mm/month)	
Characteristic	Mean (95% CI)	*P* value	Mean (95% CI)	*P* value
Male gender	0.04 (−0.04, 0.11)	0.3542	0.04 (−0.06, 0.14)	0.4764
Diabetes	**−0.08 (-0.15, -0.01)**	**0.0283**	**−0.14 (-0.23, -0.05)**	**0.0035**
Smoker	0.06 (−0.01, 0.13)	0.0814	**0.10 (0.01, 0.19)**	**0.0417**
Hypertension	−0.004 (−0.16, 0.16)	0.9684	−0.07 (−0.28, 0.14)	0.5331
Hyperlipidemia	0.05 (−0.3, 0.13)	0.2046	0.05 (−0.06, 0.17)	0.3496
Coronary artery disease	−0.01 (−0.08, 0.07)	0.8545	−0.03 (−0.14, 0.07)	0.6374
Cerebral vascular accident	0.04 (−0.05, 0.12)	0.3795	0.03 (−0.09, 0.15)	0.5344
Chronic kidney disease	0.02 (−0.05, 0.10)	0.4932	0.01 (−0.08, 0.11)	0.8018
Chronic obstructive pulmonary disease	−0.02 (−0.09, 0.06)	0.6742	−0.04 (−0.14, 0.07)	0.4553
Peripheral artery disease	−0.05 (−0.12, 0.02)	0.16945	−0.06 (−0.16, 0.04)	0.22263
Statin use	−0.05 (−0.12, 0.03)	0.2642	−0.06 (−0.16, 0.03)	0.1809
Beta-blocker	−0.01 (−0.10, 0.07)	0.7255	−0.03 (−0.13, 0.06)	0.5338
Insulin	−0.11 (−0.25, 0.04)	0.2546	−0.13 (−0.30, 0.04)	0.1353

Bold values are statistically significant.
[a]Adjusted for age, sex, history of smoking, diabetes, hyperlipidemia, hypertension, coronary artery disease, statin use, ACE inhibitors, use of sulfonylureas.

Hispanic (90%) and predominantly of Mexican descent (85%).[19] It is well documented that Hispanics in the RGV have a disproportionately higher prevalence of obesity and type 2 diabetes mellitus (30%) than any other population around the country, even among other Hispanics (12%).[20,21] Moreover, studies indicate that when compared with NHWs, Mexican-Americans with diabetes are a particularly vulnerable population, exhibiting higher rates of nonadherent behavior, inappropriate diet, less than recommended physical activity, blood glucose monitoring less than 4 times per day, and a lower percentage of glycemic control.[22,23] In addition, Piven et al. found that nonadherent behavior worsens and identifies a 0.07% decline in glycemic control and diabetes self-care for every year of age.[24] Diabetes mellitus is a leading risk factor for many cardiovascular diseases but various studies have identified a relationship between diabetes and a substantial reduction in AAA growth rate.[4,6,9,11]

Our finding of a significant association between diabetes and a 65% lower linear AAA expansion rate is consistent with previous findings reported in a 2012 meta-analysis of factors affecting small AAA expansion rates.[9] The magnitude of difference in AAA growth rates in diabetic and nondiabetic groups has varied greatly between studies although a reduction in growth by 1.63 mm/year observed in diabetic patients in this study is one of the greatest differences reported thus far.[7–10,25] In addition, while examining growth rates from previous studies (Table IV), it was observed that ethnicity appeared to have an effect on the growth rate in the diabetic population. This is demonstrated by similar AAA growth rates in articles published in the United Kingdom[5,7,9] and comparable rates found in our Hispanic population and the study by Vega de Ceniga et al. from Spain.[25] There were insufficient data from the published studies to perform statistical analyses on the correlation between AAA expansion rates and Hispanic ethnicity. However, in the present study (99% Hispanic), AAA growth rate among nondiabetics was on the higher end of all published data, as was the rate in the study by Vega de Ceniga et al.

The formation of aneurysms can be directly correlated to the development of localized arterial wall weakness. Focally weak segments within the vasculature are not able to provide adequate resistance in response to the pulsatile pressure they are subjected to, leading to the formation of aneurysms. In diabetes, characterized by a hyperglycemic state, glycosylation of proteins in the media layer of the abdominal aorta has been shown to downregulate the immune response within the vascular medial layer.[8] Thus, the pathogenicity of immune-mediated degradation within the muscular layer of the arterial wall is attenuated, providing a protective effect on the development

Table IV. Abdominal aortic aneurysm growth rate from previously published literature

Study (location)	Total sample size	Mean overall growth rate (mm/year)	Non-diabetes mellitus sample size	Non-diabetes mellitus growth rate (mm/year)	Diabetes mellitus sample size	Diabetes mellitus growth rate (mm/year)	Difference	*P* value
Sweeting et al., 2012 (UK, Canada, Sweden, Denmark)	5,697	2.21	5,074		623		−0.596 mm	0.001
Schlösser et al., 2008 (Netherlands)	147	2.5	112		35		−1.2 mm	0.057
Gollege et al., 2008 (Australia)	198		178	1.2 ± 1.16	20	0.63 ± 0.79	−0.57 mm	0.02
Thompson et al., 2010 (UK)	1,232	1.97	1,163	1.7 (Mean SE 0.23)	69	0.74 (Mean SE 0.40)	−0.95 mm	0.01
Vega de Ceniga et al., 2006 (Spain)	106	2.87	91	5.22 ± 6.11	15	1.69 ± 3.51	−3.53 mm	
Brady et al., 2004 (UK)	1,743	2.6	1,668		75		−0.79 mm	
Betancourt-Garcia et al., 2018 (South Texas-Hispanic)	140	1.8	78	2.52	60	0.878	−1.63 mm	

of arterial wall weakness and subsequent aneurysm formation. The counterintuitive finding of apparent mitigation of AAA expansion risk in patients with diabetes may be explained by the significant correlation of diabetes with reduced extensibility and increased stiffness of the aorta.[11,12] Low compliance or nonadherence in the Mexican-American population[22,23] may actually further slow the progression of AAAs by maintaining high glucose levels.

The effect of smoking on aneurysm growth rates in these subjects was consistent with previous studies and was significantly associated with an increased AAA expansion rate (0.10 mm/month) compared with nonsmokers.[3,5,9]

Although other studies have shown an association between statins and decreased growth rates, none of the medications (statins, ACE inhibitors, beta blockers, insulin) in this study demonstrated significant associations with aneurysm growth. However, this study was not powered to detect a difference in growth rates in relation to medication use.

Screening recommendations from the U.S. Preventive Services Task Force for AAAs is a one-time screening for men and women aged 65 years or older who have ever smoked, selective screening for men aged 65−75 years who have never smoked, and against routine screening for women who have never smoked.[26] Screening programs are estimated to reduce the incidence of ruptured AAA by 64%.[27] Surveillance guidelines propose monitoring aneurysm growth rates every 3 years for those with a diameter of 3.0 to 3.4 cm and every 6 months for those with an aneurysm diameter of 4.5−5.4 cm.[28] Our study demonstrates that nondiabetic patients with AAAs are prone to increases in expansion rate and may benefit from early and frequent surveillance. Studies have shown that an increase in AAA monitoring is associated with a reduction in rates of AAA rupture and AAA-related death.[29] Studies with larger sample sizes in targeted populations with known AAA prevalence and those conducting a cost-effective analysis of surveillance programs would be useful in determining the effect of surveillance on survival outcomes in both diabetic and nondiabetic patients.

CONCLUSION

This study has confirmed that diabetes has a protective effect on AAA growth rate, attenuating the rate by roughly 65%. The large difference seen between diabetics and nondiabetics in this study population may be a combination of factors associated with

race/ethnicity, prevalence of diabetes mellitus, and decreased medical compliance exhibited in the Mexican-American population. Higher powered studies should be conducted to examine the factors affecting growth rates of AAAs in both diabetic and nondiabetic populations.

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