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Factors Associated with the Progression of Aortic Aneurysms: A Single-Center Experience

Aort Anevrizmaları Progresyonu ile İlişkili Faktörler: Tek Merkez Deneyimi

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Öz

Aort anevrizmaları (AA), yetişkinlerde önemli mortalite sebebidir. Çalışmamızda, torasik aort anevrizması (TAA) ve abdominal aort anevrizması (AAA) ile ilişkili etyolojik faktörleri araştırmayı amaçladık. Çalışmamız 181 bireyi (98'i TAA ve AAA tanılı, 83'ü kontrol grubu) içermektedir. Aort anevrizma (AA) grubunda %72.4'ü erkekti. AA grubunda %57.1'i TAA, %42.9'u AAA tanılıydı. Aorta çapı 41-108 mm aralığında olanlar AA grup içine alındı. İnisiyal serum biyokimya değerleri ve 2 ardışık bilgisayarlı tomografi anjiografi ölçümü değerlendirildi ve her birey için yıllık aort çapı genişlemesi ölçüldü. AA grup ve kontrol grubu ortalama yaşı sırasıyla 62.89±13.55 ve 68.10±11.69 idi (p=0.007), hipertansiyon oranı (p=0.021) daha yüksekti. TAA grubu daha yüksek AST (p=0.016) ve trombosit değerlerine (p=0.010) sahipti. AAA grubunda ise yüksek nötrofil/lenfosit oranı (NLR) (p=0.044) mevcuttu. AAA grubunda erkek oranı %90.5'ti. Sigara kullanımı AAA grubunda daha belirgindi (p=0.08). Bir cm/yıldan daha hızlı anevrizma büyüme hızı oranı, TAA ve AAA gruplarında sırasıyla %25 ve %75 idi. TAA ile yüksek AST ve yüksek trombosit değeri arasında korelasyon mevcuttu. AAA ise erkek cinsiyet, sigara kullanımı ile yakından ilişkili bulundu.

Anahtar Kelimeler: Abdominal Aort Anevrizması, ALT, AST, Nötrofil/Lenfosit Oranı, Torasik Aort Anevrizması

Abstract

Aortic aneurysms (AA) have a significant mortality rate in population. We aimed to identify the etiologic factors associated with thoracic aortic aneurysms (TAAs) and abdominal aortic aneurysms (AAAs). Our study included 181 patients; 98 patients diagnosed with TAA or AAA made up the aortic aneurysm (AA) group, 83 patients without either condition made up the control group. Within the AA group, 72.4% of the patients were male, the patients had been diagnosed with TAA and AAA in ratio of 57.1% and 42.9% respectively. All AA group patients had an aortic diameter in range from 41 to 108 mm. Initial serum biochemical measures, two consecutive computed tomography angiography measurements were recorded, the enlargement rate per year for the aorta was calculated for all. The mean age in the AA group was 62.89±13.55 compared to 68.10±11.69 in the control group (p=0.007), higher ratio for hypertension (p=0.021). TAA patients had a higher AST level (p=0.016) and platelet counts (p=0.010) compared to control group. AAA patients had a higher mean neutrophil/lymphocyte ratio (NLR) (p=0.044) compared to control group. Among the patients with AAA, 90.5 % were male. Smoking was more prevalent in the abdominal AA group (p=0.08). An enlargement rate of more than 1 cm/year was detected in 25% of the patients with TAA and in 75% of the patients with AAA. TAA was associated with higher AST levels and higher platelet counts than were in the AAA group, whereas AAA showed strong relationships with male gender and smoking.

Keywords: Abdominal Aortic Aneurysm, ALT, AST, Neutrophil/Lymphocyte Ratio, Thoracic Aortic Aneurysm

Introduction

Aortic aneurisms (AA) have been recognized for centuries; they were even depicted in hieroglyphics from ancient Egypt (1). First try in AA was endoaneurysmorrhaphy by Rudolph Mattas in 1881 (1). AAs remained untreatable until 1951. The first AA case presented by Dr. De Bakey represented a substantial surgical advance, and the manufacturing of material used in aortic replacement has aided surgical progress. The first utilization of a homograft was also an achievement of by Dr. DeBakey, which took place in 1952 (1). Many surgeons around the world have contributed their expertise to addressing this life-threatening disease, and today successful results please patients suffering AA.

Etiological factors related to AA include smoking, older age, male gender, hypertension, chronic obstructive pulmonary disease, hyperlipidemia, atherosclerosis, white race, and a family history of AA, as well as some connective tissue diseases and syndromes such as Ehlers-Danlos type IV, Marfan, and Loeys-Dietz syndrome (2). Furthermore, some infectious diseases such as tuberculosis, syphilis, bacterial and fungal infections, in addition to inflammatory diseases, may cause AA (2). The majority of AA cases are identified without any definitive cause (3).

In the current study, we aimed to identify the etiologic factors that affect aneurysm progression in any segment of the aorta.

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Material and Method

Patient Population

This retrospective study included patients diagnosed with AA in any segment of the aorta between November 2018 and June 2022 in Muğla Sıtkı Koçman University Medical Faculty Cardiovascular Surgery Department. Ethical approval was obtained from a local review board (22/06/2022 12/II), and this study was conducted in accordance with the principles of the Declaration of Helsinki.

Inclusion criteria allowed patients who were diagnosed with an AA with a diameter of at least 41 mm and who were at least 18 years of age to be included in this study. Exclusion criteria consisted of having an aneurysm that was less than 40 mm in diameter or being younger than 18 years of age.

A power analysis performed in the G Power 3.1.2 program with a type 1 error rate of 0.05 and statistical power of 0.80 gave an effect size of 0.470. Accordingly, it was calculated that a total of 144 patients, with at least 72 patients in both groups, should be included in the planned study.

Patients (n=181) were divided into 2 groups: the aortic aneurysm (AA) group (n=98) and the control group (n=83). The AA group was divided into 2 subgroups: patients who received an endovascular intervention (EVAR/TEVAR) or surgery, and medical treatment. A flow chart of the study is shown in Figure 1.

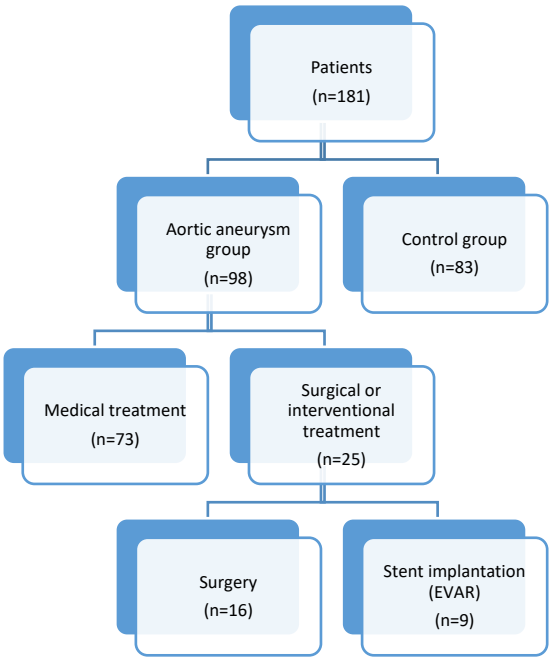

Figure 1. Demonstration of patients according to their diagnosis and treatment they received.

Two consecutive computed tomography angiography (CTA) assessments—performed with no less than 1 year between the two screenings—were evaluated for each patient. Institutional electronic database about hemogram and biochemistry, demographic features were also recorded.

Laboratory Findings

Blood samples were collected into ethylene diamine tetraacetic acid (EDTA) and analyzed using a Sysmex XN1000 hematology analyzer (Sysmex, Kobe, Japan) for white blood cell (WBC), hemoglobin (Hb), mean cell hemoglobin (MCH), red blood cell (RBC) count, platelet count, mean platelet volume (MPV), total bilirubin and direct bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT) measurements. The neutrophil/lymphocyte ratio (NLR) was calculated by dividing the total number of neutrophils by the total number of lymphocytes. Concentrations of low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides (tgl) were determined by enzymatic methods on a COBAS 8000 (c702) biochemical analyzer (Roche Diagnostics GmbH; Meinheim, Germany).

Imaging Analysis

Two consecutive measurements of AA diameters using cross-sectional CTA images were performed for each patient (no less than 1 year between two screenings). Images were obtained using a 256-slice dual energy CTA scanner (Siemens Definition Flash, Berlin, Germany) with a slice thickness of 1 mm.

Statistical Analysis

Data were analyzed with SPSS 28.0 (SPSS Inc., Chicago, IL). The Kolmogorov–Smirnov test was used to evaluate whether the distribution of continuous variables was normal. Continuous variables were compared between independent samples using independent samples t-test, and categorical variables were compared using the χ^2 test. Spearman correlation analysis was also performed to determine the level of correlation between quantitative variables. Descriptive statistics of quantitative variables were described as mean±standard deviation, median (25th–75th percentiles), and minimum–maximum. Univariate and multivariate logistic regression analyses were performed to determine the effects of risk factors on the AA enlargement rate. Receiver operating characteristic (ROC) curve analysis was applied to determine cut-off values for aortic aneurysm. Frequency and percentage (%) were given for each quantitative variable. A p value <0.05 was considered statistically significant.

Results

Demographic data for the patients in this study are shown in Table 1. Males were more common than females in both groups. The mean age was 65.28±12.96 years for all patients, 62.89±13.55

years in the AA group, and 68.10±11.69 in the control group (p=0.007). In AA group, hypertension was more frequent in ratio of 47% (p=0.021). There was no significant difference between the AA and control groups in terms of biochemical values (p>0.05). Two patients in the AA group were dead.

Table 2 shows comparative statistics for the abdominal AA (AAA) and thoracic AA (TAA) groups. Smoking habit and NLR were significantly higher in the AAA group (p=0.003 and p=0.044). However, platelet count and AST were higher in TAA group than in the AAA group (p=0.010, and p=0.016, respectively).

Table 3 shows a comparison of enlargement rate between the AAA and TAA groups; there was no significant difference between groups (p>0.05).

A high progression rate was defined as the enlargement of an aneurysm by more than 1 cm/year. A comparison of aneurysm-enlargement rate between the AA and the control group showed that the AAA group had a significantly higher progression rate (p=0.002) (Table 4).

Table 1. Patient characteristics of the AA and control groups.

Variables	**AA** (n=98)	**Control** (n=83)	p
Gender			
Male	71 (72.4)	67 (80.7)	0.259c
Female	27 (27.6)	16 (19.3)	
Age	62.89±13.55	68.10±11.69	**0.007**[s]
Smoking	45 (45.9)	45 (54.2)	0.266[c]
Hypertension	54 (65.1)	47 (48)	**0.021**[c]
Medical treatment	73 (74.5)[b]	83 (100)[a]	**<0.001**[c]
AA graft replacement	8 (8.2)[b]	0 (0)[a]	**<0.001**[c]
EVAR	9 (9.2)[b]	0 (0)[a]	**<0.001**[c]
AAA graft replacement	8 (8.2)[b]	0 (0)[a]	**<0.001**[c]
Exitus	2 (2)	0 (0)	0.501[c]
WBC	8.04 (6.01-10.08)	7.65 (5.98-9.13)	0.367[m]
Hb	13.40 (11.40-14.73)	13.30 (12.10-14.90)	0.586[m]
MCH	28.90 (27.75-30.13)	29.20 (27.70-30.50)	0.409[m]
RBC	4.74 (4.02-5.24)	4.64 (4.21-5)	0.475[m]
PLTx1000	220 (176.75-259.25)	234 (180-278)	0.214[m]
MPV	10.80±1.18	10.70±0.88	0.509[s]
NLR	2.41 (1.65-4.01)	2.31 (1.68-3.59)	0.774[m]
AST	18 (16-23.50)	17 (13-22)	0.166[m]
ALT	16 (13-22.25)	17 (12-23)	0.899[m]
T blr	0.48 (0.33-0.64)	0.51 (0.34-0.68)	0.793[m]
D blr	0.21 (0.16-0.27)	0.21 (0.16-0.29)	0.731[m]
HDL	50.35 (39.75-63.25)	49.20 (41-58)	0.770[m]
LDL	106.05 (85.75-124.25)	103 (83-124)	0.963[m]
Tgl	114 (85-167.50)	114 (81-167)	0.629[m]

[c]: Chi-Square test, [s]: Independent samples t-test, [m]: Mann Whitney U test. Descriptive statistics are expressed as mean ± standard deviation, median (25th-75th percentiles), or frequency (percentage). The same letters in the same row indicate the similarity between column percentages, and different letters indicate the difference between column percentages. AA: Aortic aneurysm, EVAR: Endovascular aortic repairment, AAA: Abdominal aortic aneurysm. WBC (white blood cell), Hb (hemoglobin), MCH (mean cell hemoglobin), RBC (red blood cell), plt (platelet counts), MPV (mean platelet volume), NLR (neutrophile–lymphocyte ratio), AST (aspartate aminotransferase), ALT (alanine aminotransferase), t blr (total bilirubin), d blr (direct bilirubin), HDL (high- density lipoprotein), LDL (low-density lipoprotein), tgl (triglyceride).

HDL, LDL, tgl, AST, ALT, WBC, Hb, MCH, RBC; When the effect of PLT, MPV, NLR, t blr, d blr, initial diameter (D1) and second diameter (D2) values were analyzed by univariate logistic regression (LR) analysis to determine if these variables affect enlargement rate>1. The effects of HDL, WBC, D1 and D2 variables on enlargement rate were statistically significant (p<0.05) while other variables were not (p>0.05). HDL increase had a protective effect of 0.958 against an enlargement rate >1 cm. As WBC, D1 and D2 values increase, the risk of an enlargement rate >1 cm increases by 1.173, 1.529 and 2.148 times, respectively (p<0.05). The variables having statistically significant effect on enlargement rate regarding univariate LR were analysed by multivariate LR. However, since there is a high level of correlation between D1 and D2 (r=0.885, p<0.001), D2 variable is excluded from multivariate LR analysis. According to the final results, it was determined that HDL had no significant effect on enlargement rate (p=0.065). As WBC and D1 value increase, the risk of an enlargement rate >1cm increases by 1.173 and 1.560 times, respectively (p<0.05) (Table 5).

The cut-off point for WBC variable for diagnosis of the disease was calculated as >8.82 by ROC-curve analysis. The sensitivity and specificity rates were found to be 38.78% and 73.49%; respectively, however, it was not statistically significant (p=0.364) because the WBC variable had a low rate of determination on enlargement rate (Nagelkerke R2=6%).

Table 2. Comparative statistics of abdominal and thoracic aortic aneurysm subgroups.

Variables	**Diagnosis** Total n=98		P
	AAA (n=42)	**TAA** (n=56)	
Gender			
Male	38 (90.5)	33 (58.9)	**0.001**[c]
Female	4 (9.5)	23 (41.1)	
Age	65 (57.50-72.25)	64 (55-71.50)	0.628[m]
Smoking	25 (59.5)	20 (35.7)	**0.033**[c]
Hypertension	23 (54.8)	24 (42.9)	0.335[c]
Medical treatment	26 (61.9)[a]	47 (83.9)[b]	**<0.001**[c]
AA graft replacement	0 (0)[a]	8 (14.3)[b]	**<0.001**[c]
EVAR	9 (21.4)[a]	0 (0)[b]	**<0.001**[c]
AAA graft replacement	7 (16.7)[a]	1 (1.8)[b]	**<0.001**[c]
Exitus	1 (2.4)	1 (1.8)	1.000[c]
Enlargement rate			
≤1	34 (81)	53 (94.6)	0.051[c]
>1	8 (19)	3 (5.4)	
WBC	8.07 (5.61-10.44)	8.01 (6.17-9.71)	0.926[m]
Hb	13.40 (11.23-14.58)	13.35 (11.43-14.78)	0.994[m]
MCH	28.80 (27.98-30.53)	29 (27.50-29.88)	0.497[m]
RBC	4.67 (4.01-5.25)	4.78 (4.04-5.26)	0.796[m]
PLTx1000	189.50 (169.75-245.25)	233.50 (197.75-284.75)	**0.010**[m]
MPV	10.70 (10.05-11.13)	10.65 (9.93-11.65)	0.951[m]
NLR	3.13 (1.79-5.88)	2.19 (1.51-3.76)	**0.044**[m]
AST	17.30 (14.75-19.40)	19.40 (16-26)	**0.016**[m]
ALT	15.50 (12.75-18)	16 (13-23.75)	0.585[m]
T blr	0.47 (0.35-0.64)	0.49 (0.30-0.66)	0.928[m]
D blr	0.21 (0.16-0.33)	0.21 (0.15-0.27)	0.595[m]
HDL	45 (40.25-56.05)	53 (39.25-65)	0.269[m]
LDL	99.14±27.27	110.73±35.25	0.080[s]
Tgl	111.50 (73.88-145.50)	119 (89.25-186.50)	0.539[m]

[c]: Chi-Square test, [s]: Independent samples t-test, [m]: Mann Whitney U test. Descriptive statistics are expressed as mean ± standard deviation, median (25th-75th percentiles), or frequency (percentage). The same letters in the same row indicate the similarity between column percentages, and different letters indicate the difference between column percentages. TAA: Thoracic aortic aneurysm, AAA: Abdominal aortic aneurysm, AA: Aortic aneurysm, EVAR: Endovascular aortic repairment, WBC (white blood cell), Hb (hemoglobin), MCH (mean cell hemoglobin), RBC (red blood cell), plt (platelet counts), MPV (mean platelet volume), NLR (neutrophile–lymphocyte ratio), AST (aspartate aminotransferase), ALT (alanine aminotransferase), t blr (total bilirubin), d blr (direct bilirubin), HDL (high- density lipoprotein), LDL (low-density lipoprotein), tgl (triglyceride).

Mortality was reported for two patients in the AA group. The first of these patients died after an AAA repair with graft interposition. He was 82 years old, and during the postoperative period he could not be weaned from ventilatory support due to his comorbid chronical obstructive pulmonary disease; he died on the 15th postoperative day. The second patient was a 68-year-old male who died due to catheter infection and sepsis on the on the 20th postoperative day after TAA repair with supra-coronary graft interposition.

Discussion

The current study describes the postoperative outcomes of our included patients and indicates factors associated with AA. When we compared TAA and AAA with regard to possible related etiologic features of AA, we found that smoking, enlargement rate>1 cm/year, and higher NLR were significantly higher in the AAA group; in contrast, higher AST values, higher platelet counts are significantly higher in TAA group. HDL, WBC, D1 and D2 values of AA had a significant effect on enlargement rate>1 cm/year. WBC, D1 and D2 values of AA increase the risk of an enlargement rate >1 cm.

AAs may develop in all aortic segments. They can produce life-threatening complications such as aortic dissection and aortic rupture, which represent difficult clinical challenges to cardiothoracic surgery teams (4).

The normal aortic wall is composed of three layers: the intima, media, and adventitia. During embryological development, the increase in medial thickness is related to an increase in the number of lamellar units (from 35 to 56), whereas there is only a minor increase in their thickness (from 12 to 17 μm) (5). However, medial growth in the abdominal aortic segment is associated with an increase in thickness of the lamellar units (from 12 to 26 μm) that is related to smooth muscle cell proliferation, but there is a minimal increase with regard to their number (from 25 to 28) (5). Therefore, the tension on lamellar units in adults is greater in the abdominal aorta than in the thoracic aorta (5). In addition, the amount of elastic tissue diminishes from the thoracic to the abdominal aorta (6). In light of this knowledge, it is believed that the thoracic aorta is better protected from aneurysm formation compared to the abdominal aorta.

Table 3. Comparative statistics of subgroups of AA according to enlargement rate.

Variables	**Enlargement rate** Total n=98		p
	≤1 *cm/year* (n=87)	>1 *cm/year* (n=11)	
Gender			
Male	61 (70.1)	10 (90.9)	0.281[c]
Female	26 (29.9)	1 (9.1)	
Age	62.64±13.48	64.82±14.63	0.619[s]
AAA	34 (39.1)	8 (72.7)	0.051[c]
TAA	53 (60.9)	3 (27.3)	
Smoking	38 (43.7)	7 (63.6)	0.352[c]
Hypertension	39 (44.8)	8 (72.7)	0.154[c]
Medical treatment	70 (80.5)[a]	3 (27.3)[b]	**0.004**[c]
AA graft replacement	6 (6.9)[a]	2 (18.2)[a]	**0.004**[c]
EVAR	6 (6.9)[a]	3 (27.3)[b]	**0.004**[c]
AAA graft replacement	5 (5.7)[a]	3 (27.3)[b]	**0.004**[c]
Exitus	2 (2.3)	0 (0)	>0.999[c]
WBC	8.16±2.86	10.68±6.07	0.201[s]
Hb	13.40 (11.50-14.70)	12.40 (10.80-15.90)	0.547[m]
MCH	29 (27.60-30.20)	28.60 (27.80-29.90)	0.657[m]
RBC	4.78 (4.02-5.27)	4.24 (3.88-5.22)	0.344[m]
PLTx1000	221 (177-259)	184 (169-295)	0.613[m]
MPV	10.86±1.22	10.36±0.73	0.181[s]
NLR	2.29 (1.61-3.94)	3.35 (1.74-8.18)	0.151[m]
AST	18 (16-23)	18 (16-26)	0.731[m]
ALT	16 (13-22)	17 (13-36.10)	0.573[m]
T blr	0.47 (0.33-0.63)	0.52 (0.23-1.08)	0.800[m]
D blr	0.21 (0.16-0.27)	0.20 (0.13-0.33)	0.804[m]
HDL	53 (41-64)	45 (36-51.10)	0.180[m]
LDL	106.10 (86-126)	91 (85.60-113)	0.434[m]
Tgl	120 (90-173)	99 (65-135.20)	0.098[m]

[c]: Chi-Square test, [s]: Independent samples t-test, [m]: Mann Whitney U test. Descriptive statistics are expressed as mean±standard deviation, median (25^{th}-75^{th} percentiles), or frequency (percentage). The same letters in the same row indicate the similarity between column percentages, and different letters indicate the difference between column percentages. AAA: Abdominal aortic aneurysm, TAA: Thoracic aortic aneurysm, AA: Aortic aneurysm, EVAR: Endovascular aortic repairment, WBC (white blood cell), Hb (hemoglobin), MCH (mean cell hemoglobin), RBC (red blood cell), plt (platelet counts), MPV (mean platelet volume), NLR (neutrophile–lymphocyte ratio), AST (aspartate aminotransferase), ALT (alanine aminotransferase), t blr (total bilirubin), d blr (direct bilirubin), HDL (high- density lipoprotein), LDL (low-density lipoprotein), tgl (triglyceride).

Another proposition for AA development is that the upregulation of common pathways in terms of reactive oxygen radical production may lead to smooth muscle cell dysfunction, extracellular matrix destruction, and aortic inflammation (7,8).

Many etiologic factors were found to be related to the progression of AA, including advanced age, male sex, smoking, family history of aneurysm, family history of hypertension, and hypercholesterolemia (9,10). We found significant relationships between AAA and male sex, smoking, and familial history of AAA as reported in the literature; however, we did not find the same relationships for TAA.

Table 4. Comparison of aneurysm types according to enlargement rate.

Aneurysm type	**Enlargement rate (n / %)**		p
	≤1 *cm/year* (n=165)	>1 *cm/year* (n=16)	
AAA	54 (32.7)	12 (75)	**0.002**[c]
TAA	111 (67.3)	4 (25)	

[c]: Chi-Square test. Descriptive statistics are expressed as frequency (percentage). AAA: Abdominal aortic aneurysm, TAA: Thoracic aortic aneurysm.

Vuruşkan and Folsom demonstrated that AAA is significantly correlated with higher WBC counts and higher NLR compared with the control patients (3,4). We did not find a strong relationship of either higher WBC counts or higher NLR with AA. However, platelet counts were significantly higher in TAA group compared to AAA group.

In Cho's et al. study, AA has been found to be related to atherosclerosis and hypercholesterolemia (12). However, in the present study we did not find the same result. In our region, the Mediterranean diet is preferred. Perhaps, this habit let the blood lipid (LDL, tgl) level lower. Therefore, our results were not correlated with those in the literature.

Kang and Kawamoto's cohort studies show that increased levels of serum bilirubin protect vessels against atherosclerotic processes in peripheral arterial disease and carotid artery disease (13,14). However, we did not find any relationship between higher bilirubin levels and lower incidence of AA. In contrast, AST values are significantly higher in the TAA group.

Table 5. Univariate and multivariate logistic regression analysis results.

	Univariate logistic regression		Multivariate logistic regression	
	OR [95% CI]	p	OR [95% CI]	p
HDL	0.958 [0.919-0.998]	**0.040**	0.960 [0.919-1.003]	0.065
WBC	1.173 [1.025-1.342]	**0.020**	1.173 [1.015-1.354]	**0.030**
D1	1.529 [1.040-2.248]	**0.031**	1.560 [1.051-2.315]	**0.027**
D2	2.148 [1.469-3.140]	**<0.001**	-	-

OR: Odds ratio, CI: Confidence interval, HDL (high- density lipoprotein), WBC (white blood cell), D1: Initial diameter measurement of AA, D2: Second diameter measurement of AA.

Kirsch and Schmid's studies on TAA have focused on hereditary connective tissue disorders, bicuspid aortic valve association, and smooth muscle cell loss (5,15). In our study, there were no patients diagnosed with either connective tissue disorder or bicuspid aortic valve association.

Medical management up to surgical repair includes the control of risk factors that may prevent growth and rupture of AAs, but at present such medical therapies have limited success. Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, β-blockers, and statins may be utilized to decrease the growth rate of AAs.

Conclusion

In the current study we found that TAA was associated with higher AST levels and higher platelet counts than were in the AAA group, whereas AAA showed strong relationships with male gender and smoking that were not observed for the TAA group.

New opportunities to determine various targets involved in vascular inflammation, cell death, extracellular matrix degradation, intramural thrombosis, and atherosclerosis of the vasa vasorum of the aortic wall are required. Relevant innovative research and studies are important and necessary.

Limitations: There are some limitations of our study. Firstly, its retrospective design may limit the ability to investigate detailed information of the patient population. Secondly, this study was designed as a single-center study; the number of patients can therefore be considered limited, and it needs to be repeated in a larger population sample. Also, no patients with Ehler-Danlos, Loeyz-Dietz or Marfan syndromes were included in this study, and there were also patients who were diagnosed with associated bicuspid aortic valve.

Conflict of interest statement

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