Risk factors for rupture of abdominal aortic aneurysm based on three-dimensional study

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Purpose: **Factors influencing the development or rupture of abdominal aortic aneurysms (AAAs) have not yet been confirmed. This study delineated the risk factors for rupture of AAAs as evaluated by means of a combination of three-dimensional (3D) reconstruction and clinical data analysis.**

Methods: **The study population comprised Japanese patients in whom an atherosclerotic AAA had been diagnosed between January 1980 and December 1997. We obtained 3D-based data by means of computer-aided 3D reconstruction from computed tomography studies of AAAs. The data included the tortuosity of the aneurysm, maximum transverse diameter, length of the aneurysm, aneurysmal volume, aneurysmal surface area, largest aneurysmal cross-sectional area, ratio of transverse aneurysmal diameter to the length of the aneurysm (T/L), and amount of mural thrombus. Clinical data were collected from patient files. All data were assessed by means of multivariate analysis for their predictive value for expansion or rupture of AAA.**

Results: **The most efficient predictor of annual expansion rate of maximum transverse diameter (EX-D) was a combination of largest aneurysmal cross-sectional area, tobacco use, and tortuosity. The most efficient predictor of annual expansion rate of aneurysmal volume (EX-V) was a combination of aneurysmal volume and blood urea nitrogen level. The most efficient predictors of aneurysmal rupture was a combination of EX-D, diastolic blood pressure, and T/L.**

Conclusion: **Three-dimensional–based data on aneurysmal morphology, including T/L, largest aneurysmal cross-sectional area, and aneurysmal volume, had strong predictive value for expansion and rupture of AAAs. (J Vasc Surg 2001;33:453-61.)**

Patients with an atherosclerotic abdominal aortic aneurysm (AAA) often have coexistent medical problems because of their advanced age, smoking habits, and generalized atherosclerosis. In such patients, the strategy for treatment should weigh both the risk of rupture of the present AAA and the severity of the coexistent medical problems.[1-6] However, information on the natural history of AAAs is scarce, and risk factors for aneurysmal rupture have not yet been established.[7]

Recent developments in computer technology have made it possible to examine internal organs with three-dimensional (3D) diagnostic techniques such as spiral computed tomography (CT), magnetic resonance imaging, and 3D-digital subtraction angiography.

The purpose of this study was to delineate the risk factors for the rupture of AAAs as evaluated by means of a combination of 3D reconstruction and clinical data analysis.

METHODS

Patients

The study population comprised Japanese patients in whom an atherosclerotic AAA had been diagnosed during an 18-year period between January 1980 and December 1997. Of these patients, those who met the inclusion criteria were selected for the study. The diagnosis of AAA was established when the anteroposterior diameter of the abdominal aorta, measured by means of either ultrasound scanning or CT study, was greater than 50% of the undilated infrarenal aortic diameter, according to the suggested standards recommended by an ad hoc committee.[8]

Inclusion criteria

Three-dimensional–based data were obtained by means of computer-aided 3D reconstruction from CT studies of AAAs. Patients were included in the study according to the inclusion criteria for CT studies. A narrow tomography slice gives a better quality 3D image of an AAA. After evaluating the quality of 3D images reconstructed from original CT studies with various slicing distances, we adopted a CT slicing distance between 5 and 15 mm for the study. For better visualization, contrast enhancement in the original CT studies was necessary to distinguish the aortic wall from surrounding tissues at data input. We determined that a CT study interval of more than 6 months was sufficient to show 3D morphologic change with time.

Three-dimensional reconstruction

Our 3D reconstruction procedure was detailed by Nunokawa et al.[9] We used the commercially available software "OZ" (Rise, Saitama Prefecture, Japan) on a Bild/3D computer system (Computer Built, Tokyo, Japan).

The procedure had three steps. The first step was input of the aneurysmal image from each sequential CT

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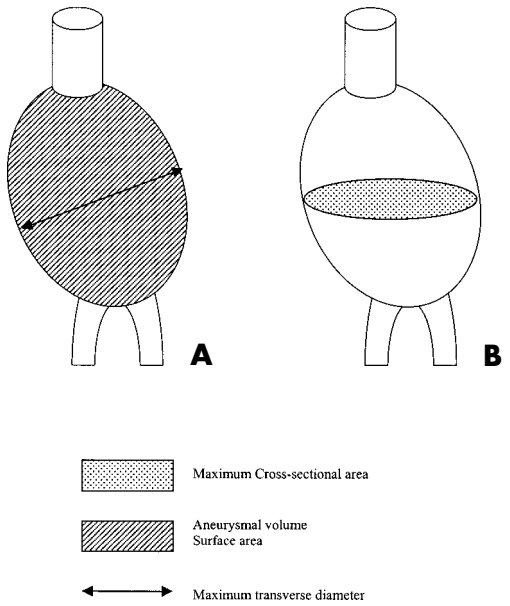

Fig 1. Aneurysmal volume and surface area were measured for segment with the transverse diameter that was 3 mm or more longer than the undilated infrarenal aorta. **A,** Largest cross-sectional area was defined as the one at the level of the maximum transverse diameter of aneurysm in the axial plane **(B)**.

slice on a digitizing pad. A frame of each slice was fixed to the frame marked on the digitizer pad to secure the precise continuity of each slice image of the aneurysm. The second step was data processing, in which the software calculated the coordinates to build the whole 3D image. The number of coordinates settled on was approximately 1 or 2 per 1 mm of mouse tracing. The X- and Y-coordinates were calculated from the input aneurysmal slice image. The Z-coordinates were calculated from the input slicing distance and the order of each slice. The last step was 3D image presentation on the display. The reconstructed 3D image can be shown with a scale and rotated in any direction on the computer display.

The 3D-reconstruction software OZ can calculate the volume, surface area, or cross-sectional area of the reconstructed 3D image. When necessary, the inner structure of the 3D image can be shown in fluoroscopic mode. The mural thrombus of AAAs was shown by means of this mode, and the amount of it was also calculated.

Study design

Clinical data. The patient record was retrospectively reviewed to collect information on age; sex; height; weight; tobacco use; systolic and diastolic blood pressure; pulse pressure; coexistent diseases such as hypertension (systolic blood pressure > 140 mm Hg or diastolic blood pressure > 90 mm Hg or controlled with a drug), diabetes mellitus (controlled by means of diet, oral drug, or insulin), coronary artery diseases (history of angina, myocardial infarction, or coronary artery bypass graft), arteriosclerosis obliterans (diagnosed by means of pedal pulses and symptoms), cerebrovascular disease (history of transient or complete stroke), chronic obstructive pulmonary disease (forced expiratory volume 1.0% < 50%), renal failure (serum creatinine level > 3.0 mg/dL), and liver disease; earlier laparotomy; laboratory data, including cholesterol level, triglyceride level, urea nitrogen level, creatinine level, fibrinogen level, fibrin degradation product level, platelet count, and prothrombin time; symptoms of AAA; rupture; and familial AAA.

Three-dimensional–based morphology data. The maximum aneurysmal transverse diameter and length of the aneurysm were measured on the reconstructed 3D image by the use of the scale on the display. Because the 3D figures used for this purpose were not drawn in perspective, it was possible and valid to measure the diameter and length with the scale on it. The length of the aneurysm was defined as the length of a straight line drawn through the center of the most proximal and most distal cross section, in which the transverse aortic diameter was at least 3 mm larger than the normal infrarenal aortic diameter. Aneurysmal volume, aneurysmal surface area, and largest aneurysmal cross-sectional area were calculated by means of the OZ software. A volume value physically measured was compared with the one calculated by means of the software in 20 clay models of aneurysms to validate the accuracy of volume measurement with this software. As shown in Fig 1, *A*, the volume was defined as that of the segment with the transverse diameter that was 3 mm or more longer than the undilated infrarenal aorta. Surface area was defined as the outer area of the segment, which was defined as the aforementioned. The largest cross-sectional area was defined as the one at the level of the maximum transverse diameter of the aneurysm in the axial plane (Fig 1, *B*).

In the 3D figures of AAA, tortuosities of the aorta and the aneurysm were classified into three types while they were examined, with a 3D image of the vessel axis drawn by means of a function of the OZ software. A type of tortuosity was defined as (1) a bent type when the axis of the aorta and the aneurysm was on the same plane without any element of spiral form, (2) a clockwise type when the axis looked like a clockwise whorl when the aorta was viewed from the cranial to caudal direction, or (3) a counterclockwise type when the axis was opposite to the clockwise type (Fig 2).

We also measured the ratio of the transverse aneurysmal diameter to the length of the aneurysm (T/L; Fig 3). The ratio of the maximum transverse diameter to the normal infrarenal aortic diameter (ROD) was also measured on the 3D image.

An outstanding feature observed in the 3D images was the varying amount of mural thrombus. To evaluate the amount, we calculated the volume of mural thrombus with the same 3D-reconstructing software, OZ. The amount was classified as "much" when the amount of mural thrombus was more than 50% of aneurysmal vol-

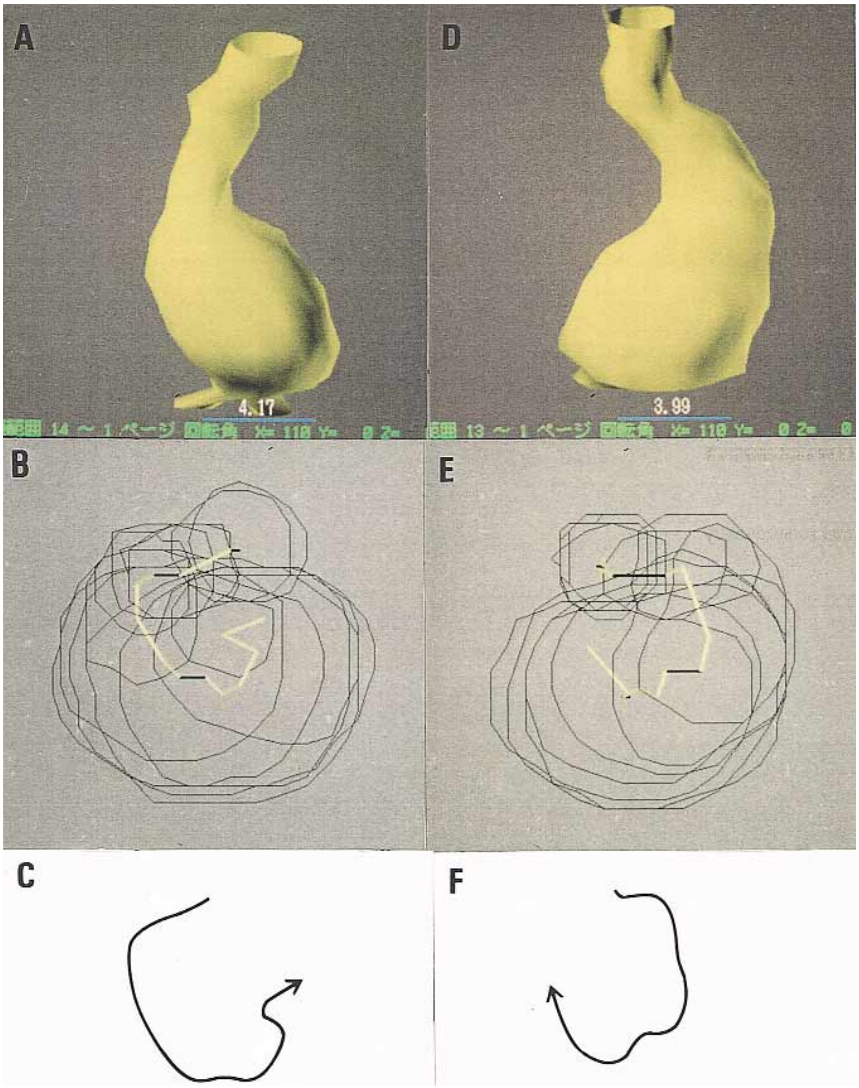

Fig 2. Spiral tortuosity of AAA. Spiral tortuosity seen in AAAs consisted of two types, counterclockwise clockwise **(A, B, C)** and clockwise **(D, E, F)**. **B** and **E** are cranial-to-caudal views of 3D images **A** and **D,** respectively. **C** and **F** show their axes, which are counterclockwise and clockwise, respectively.

ume, "medium" when it was from 10% to 50% of aneurysmal volume, and "slight" when it was less than 10% of aneurysmal volume.

To measure the change in an AAA with time, we calculated the annual expansion rate of the maximum transverse diameter of the aneurysm (EX-D) and that of the aneurysmal volume(EX-V).

Statistical analysis

Both the clinical and 3D morphology-related data were analyzed with the software "Lotus 123" and "123 Add In Statistics" (Social Information, Tokyo, Japan). These data and either EX-D or EX-V were statistically analyzed as a means of assessing factors associated with aneurysmal expansion. Risk factors for rupture of AAA were assessed by analyzing clinical data, 3D morphology-related data, EX-D, and EX-V.

Laboratory data were categorized as either normal or abnormal. They were statistically analyzed as both original and categorized data. All data were quantified for regression analyses. A single correlation coefficient for all clinical and morphology-related data was calculated for either EX-D or EX-V, and data that showed higher coefficient value were selected. For the prevention of multicolinearity, variables that had a high correlation coefficient for each other were treated as one group, and more than two variables from each group were not used in the multiple regression analysis. Thus, variables that had a higher single correlation coefficient for EX-D or EX-V and had little correlation with each other were adopted for multiple regression analysis.

Table I. Characteristics of 39 patients with an AAA who were involved in the study

Characteristic	*Value*
Age (y)	75.8 ± 1.0
Sex	
Men (%)	87.2
Women (%)	12.8
Height (cm)	157.4 ± 1.15
Body weight (kg)	52.7 ± 1.2
Ever smoked regularly (%)	47.1
Tobacco use (packs/day)	7.38 ± 1.44
Systolic blood pressure (mm Hg)	145.6 ± 3.0
Diastolic blood pressure (mm Hg)	81.0 ± 1.9
Pulse pressure (mm Hg)	64.6 ± 2.4
Hypertension (%)	64.7
Diabetes mellitus (%)	14.7
Coronary artery disease (%)	20.6
Arteriosclerosis obliterans (%)	44.1
Cerebrovascular disease (%)	20.6
Chronic obstructive pulmonary disease (%)	23.5
Renal failure (%)	23.5
Liver disease (%)	2.9
Serum cholesterol (mg/dL)	202.9 ± 6.71
Triglyceride (mg/dL)	134.2 ± 13.3
Urea nitrogen (mg/dL)	24.6 ± 2.63
Serum creatinine level (mg/dL)	1.85 ± 0.40
Fibrinogen (mg/dL)	261.4 ± 7.87
Platelet count (1000/mm^3)	21.9 ± 0.82
Prothrombin time (%)	89.8 ± 1.68
Maximum transverse diameter (cm)	5.00 ± 1.43
Length of aneurysm (cm)	7.40 ± 2.83
Largest cross-sectional area (cm^2)	19.1 ± 12.1
Aneurysmal surface area (cm^2)	104.0 ± 58.0
Aneurysmal volume (cm^3)	110.2 ± 92.2
Ratio of transverse to length of diameter (T/L)	0.72 ± 0.22
Tortuosity	
Clockwise (%)	35.3
Counterclockwise (%)	38.2
Bent (%)	2.9
Ratio of maximal aneurysmal to infrarenal diameter (ROD)	1.90 ± 0.51
Infrarenal aortic diameter (cm)	2.67 ± 0.61
Symptomatic (%)	26.5
Earlier laparotomy (%)	52.9
Familial AAA (%)	2.9
Growth rate of maximal transverse diameter (cm/y)	0.311 ± 0.047
Growth rate of aneurysmal volume (cm^3/y)	25.01 ± 4.67
Aneurysmal rupture (%)	20.6

Clinical data, morphology-related data, EX-D, and EX-V were assessed with a single correlation coefficient for aneurysmal rupture. Again, data were selected to prevent multicolinearity multivariate analysis. Risk factors for aneurysmal rupture were analyzed in discriminant analysis. Accuracy in this analysis meant the coincidence rate of actual results, ruptured or nonruptured, and expected results from the analysis. The correlation ratio was the r^2 value in the discriminant analysis.

RESULTS

Thirty-nine patients with an atherosclerotic AAA met the inclusion criteria and were included in the study (Table I). The mean ± the SD values for the length of the follow-up period was 31.8 ± 24.5 months and for the number of CT studies was 2.9 ± 1.4 times. Eighteen patients were operated on for an AAA, and seven patients had ruptured AAAs. None of the 39 patients was lost to follow-up. Neither clinical nor morphology-related data were significantly different from those of our whole patient population. The mean calculated volume value of clay models of aneurysms was not significantly different from the mean physically measured value (103.6 ± 5.3 cm^3 vs 100.0 ± 0.0 cm^3). The mean ± SD values for EX-D and EX-V were 0.31 ± 0.29 cm/y and 25.0 ± 28.7 cm^3/y, respectively.

Factors associated with aneurysmal expansion of maximum transverse diameter. Variables for which the single correlation coefficient (*r* value) for EX-D was more than 0.5 included aneurysmal volume, maximum cross-sectional area, aneurysmal surface area, maximum transverse diameter,

Table II. Single correlation coefficient for EX-D, EX-V, and aneurysmal rupture

Variables	*EX-D*	*EX-V*	*Aneurysmal rupture*
Age	0.118	–0.024	0.3555
Sex	–0.009	0.208	0.0468
Height (cm)	–0.365	0.037	–0.3520
Weight (kg)	–0.181	0.004	–0.2290
Smoker/nonsmoker	0.572*	0.415	0.0374
Tobacco use (packs/day)	0.404	0.352	0.0064
Systolic blood pressure	0.022	0.231	0.2037
Diastolic blood pressure	0.242	0.346	0.4701
Pulse pressure	–0.168	0.023	–0.1214
Hypertension	0.183	0.329	0.3750
Diabetes mellitus	0.216	–0.166	0.0546
Coronary artery disease	0.416	–0.102	0.2500
Arteriosclerosis obliterans	0.067	–0.234	–0.0403
Cerebrovascular disease	–0.174	– 0.221	–0.2500
COPD	0.127	–0.158	–0.0468
Renal failure	0.140	0.265	0.1873
Liver disease	–0.226	–0.090	–0.1021
Cholesterol	–0.174	–0.240	–0.2374
Triglyceride	0.340	–0.010	0.2848
Urea nitrogen	0.373	0.594*	0.2139
Creatinine level	0.220	0.534†	0.0122
Fibrinogen	–0.099	0.186	0.0225
Platelet count	–0.196	–0.142	0.0315
Prothrombin time	–0.230	–0.218	–0.0577
Maximum transverse diameter	0.657*	0.783*	0.4231†
Longitudinal diameter	0.620*	0.761*	0.0936
Largest cross-sectional area	0.634*	0.806*	0.5600*
Aneurysmal surface area	0.622*	0.836*	0.2316
Aneurysmal volume	0.667*	0.910*	0.4055†
T/L	–0.235	–0.195	0.3675*
Tortuosity	0.514	0.031	–0.1217
ROD	0.361	0.517	0.3302
Infrarenal aortic diameter	0.422	0.447	0.2379
Rupture	0.540	0.301	0.2406
Previous laparotomy	–0.184	0.189	0.0801
Familial AAA	–0.224	–0.175	–0.0933
EX-D			0.5403†
EX-V			0.3008

*$P < .01$.
†$P < .05$.
COPD, Chronic obstructive pulmonary disease; *T/L,* ratio of transverse diameter to longitudinal diameter; *ROD,* ratio of the maximum transverse diameter to the normal infrarenal aortic diameter; *EX-D,* expansion rate of maximal transverse diameter; *EX-V,* expansion rate of aneurysmal volume.

length of the aneurysm, tobacco use, and tortuosity (Table II). Aneurysmal volume, maximum cross-sectional area, aneurysmal surface area, maximum transverse diameter, and length of the aneurysm were treated as one group, because they had higher single correlation coefficients for each other. Multiple regression analysis was carried out among any single variable of this group, tobacco use, or tortuosity (Table III). As shown in equation A, the most efficient predictors for EX-D were maximum cross-sectional area (X cm^2), tobacco use (Y = 1, smoker; Y = 0, nonsmoker), and tortuosity (Z = 1, counterclockwise; Z = 0, clockwise), with 0.72 of the decision coefficient (r^2 value; $P = .0004$).

$$\text{(A)} \qquad \text{EX-D} = 0.012X + 0.185Y + 0.184Z - 0.163$$

Factors associated with aneurysmal expansion of aneurysmal volume. Variables for which the single correlation coefficient for EX-V was more than 0.5 included urea nitrogen level, serum creatinine level, ROD, maximum cross-sectional area, aneurysmal length of the aneurysm, maximum transverse diameter, aneurysmal surface area, and aneurysmal volume (Table II). Urea nitrogen and serum creatinine levels were treated as one group, because they had higher single correlation coefficients for each other. Also treated as one group were ROD, maximum cross-sectional area, aneurysmal length of the aneurysm, maximum transverse diameter, aneurysmal surface area, and aneurysmal volume. Multiple regression analysis was carried out by the use of any single variable from either group or any two variables from each of these two groups (Table IV). As shown in equation B, the most efficient predictors for EX-V were aneurysmal volume (X cm^3) and urea nitrogen level (mg/dL) with 0.71 of the decision coefficient (r^2 value; $P = .0004$).

Table III. Multiple regression analysis for EX-D

Combination of variables	r^2 *value*	P *value*
T,H,C	0.693	.0001
T,H,R	0.675	.0002
T,H,V	0.638	.0005
H,C	0.611	.0002
H,R	0.599	.0003
T,H,S	0.575	.0019
H,V	0.562	.0006
T,H,L	0.540	.0036
T,R	0.499	.0020
T,C	0.494	.0022
H,S	0.481	.0027
T,H	0.471	.0032
T,V	0.471	.0032
H,L	0.450	.0046
T,S	0.423	.0071
T,L	0.407	.0091
R	0.336	.0059
H	0.314	.0082
V	0.312	.0085
C	0.307	.0092
T	0.291	.0117
L	0.262	.0178
S	0.248	.0215

C, Maximum cross-sectional area; *H,* tortuosity; *L,* aneurysmal longitudinal diameter; *R,* maximum transverse diameter; *S,* aneurysmal surface area; *T,* tobacco use; *V,* aneurysmal volume.

Table IV. Multiple regression analysis for EX-V

Combination of variables	r^2 *value*	P *value*
b,V	0.8713	.00000001
c,V	0.8702	.00000001
V	0.8226	.00000001
b,S	0.7885	.00000085
c,C	0.7821	.00000111
c,S	0.7596	.00000268
b,C	0.7434	.00000482
c,R	0.7138	.00001287
b,R	0.6960	.00002217
S	0.6847	.00000370
b,L	0.6357	.00011303
C	0.6347	.00001546
R	0.5981	.00003934
c,L	0.5932	.00030522
L	0.5598	.00009597
b,r	0.5016	.00189643
c,r	0.4991	.00198563
b	0.3531	.00450025
c	0.2850	.01268805
r	0.2353	.02582899

b, Urea nitrogen level; *c,* serum creatinine level; *r,* ROD; *C,* maximum cross-sectional area; *L,* aneurysmal longitudinal diameter; *R,* maximum transverse diameter; *S,* aneurysmal surface area; *V,* aneurysmal volume.

$$\text{(B)} \qquad \text{EX-V} = 0.203X + 267Y - 8.08$$

Factors associated with aneurysmal rupture. Variables that had a higher single correlation coefficient for aneurysmal rupture were age, diastolic blood pressure, T/L, maximum cross-sectional area, and EX-D (Table II). Maximum cross-sectional area and EX-D were treated as one group, because they had higher single correlation coefficients for each other. Discriminant analysis was made with any single variable of this group, age, diastolic blood pressure, or T/L (Table V). As shown in equation C, the most efficient predictors for aneurysmal rupture were diastolic blood pressure (X mm Hg), EX-D (Y cm/y), and T/L (Z) with 95.8% of accuracy and 0.632 of the correlation ratio (r^2 value).

Table V. Discriminant analysis for aneurysmal rupture

Combination of variables	r^2 *value*	P *value*
b,t,E	0.6589	.0003
t,E	0.5762	.0004
a,b,t,E	0.6630	.0010
a,t,E	0.5790	.0017
t,C	0.4560	.0042
b,t,C	0.5016	.0069
t	0.3009	.0100
a,t,C	0.4628	.0126
b,t	0.3846	.0127
a,b,E	0.4509	.0150
a,b,t,C	0.5133	.0161
a,b,t	0.4347	.0189
b,E	0.3531	.0198
a,t	0.3464	.0218
C	0.2309	.0275
b,C	0.3214	.0305
a,b	0.3212	.0306
a,b,C	0.3928	.0333
a,C	0.2974	.0417
a,E	0.2931	.0441
a	0.1769	.0577
E	0.1767	.0577
b	0.1637	.0689

a, Age; *b,* diastolic blood pressure; *t,* T/L; *C,* maximum cross-sectional area; *E,* EX-D.

$$\text{(C)} \qquad R = 0.151X + 14.6Y + 17.2Z - 30.8$$
$$(R > 0, \text{ rupture; } R < 0, \text{ nonrupture})$$

DISCUSSION

The results of this study revealed that aneurysmal rupture and expansion in diameter or volume could be predicted more precisely by means of a combination of the factors than by means of any single factor. For aneurysmal rupture, the factors included EX-D, diastolic blood pressure, and T/L. For expansion rate in diameter (EX-D), the factors included cross-sectional area, tobacco use, and tortuosity. For expansion rate in volume (EX-V), the factors included aneurysmal volume and blood urea nitrogen level. Some of the factors were known to be significant for aneurysmal expansion or rupture, but some of them were not.

T/L is a novel factor that represent the longitudinal expansion (Fig 3). When two aneurysms have the same transverse diameters but different lengths, the one with a higher T/L value can be said to have a more longitudinally

stretched aneurysmal wall and, consequently, a more two-dimensionally stretched wall than the one with a lower T/L value. Since the time when Szilagyi et al reported that diameter had an important bearing on the decision of operability, there have been many articles on the close relation between size and rupture of AAAs.[1,5,10-14] For the evaluation of AAAs, maximal transverse diameter has been the gold standard, whereas longitudinal expansion has hardly been considered. The results of our study demonstrated that the factor of longitudinal expansion represented as T/L is significantly associated with aneurysmal rupture. Ouriel et al[15] demonstrated that "the saccular index" that represented the ratio of the transverse diameter to the length of AAA had no correlation to aneurysmal rupture. The important difference between Ouriel's study and ours is the measurement of aneurysmal length with or without 3D reconstruction. We think this method is the most reliable means of measuring various values of AAA, such as maximum transverse diameter, length, cross-sectional area, or volume, because AAAs were so tortuous in 80% of cases in this study that these values could be misinterpreted on the planes of conventional imaging studies.

The growth rate of the transverse diameter of an AAA (EX-D) was also one of three clues in predicting the rupture of an AAA. Since Bernstein et al[10] first introduced the growth rate of AAA as a powerful tool for making decisions about surgery, there have been numerous discussions focusing on the subject. In their subsequent report, surgical resection was recommended for patients with smaller AAAs who underwent follow-up ultrasonography when the aneurysm grew rapidly (> 5 mm in 3 months) or symptoms developed.[1] Gadowski et al[16] studied the expansion rate of AAA in patients prospectively monitored by means of serial ultrasound scanning examination. They found that the mean expansion rate was significantly greater in patients with a ruptured AAA than in patients with an unruptured AAA (0.82 ± 0.74 cm/y vs 0.42 ± 0.41 cm/y; $P = .04$). Bengtsson et al[17] studied the characteristics of the aneurysm that influenced the rate of rupture by means of the Cox proportional hazard method. They found that diameter, length, and expansion rate of diameter had relative risks of 2.9, 3.4, and 2.3, respectively.

Diastolic blood pressure was another factor for predicting aneurysmal rupture in the study. The same results were described by some authors. Cronenwett et al[11] studied risk factors associated with the rupture of small AAAs in patients initially selected for nonoperative treatment. They identified diastolic blood pressure, initial anteroposterior diameter of the aneurysm, and degree of obstructive pulmonary disease as being independently predictive of rupture. In a retrospective study of patients with an AAA, Foster et al[3] found that rupture of the aneurysm was the cause of death in 72% of patients with diastolic hypertension and an untreated abdominal aneurysm; however, they did not conclude that diastolic hypertension had directly contributed to the rupture.

Our results were comparable with these reports, which demonstrated that a growth rate of an aneurysm or diastolic blood pressure was a risk factor for aneurysmal rupture. However, our study results indicate the combination of three factors were the most powerful means of predicting aneurysmal rupture.

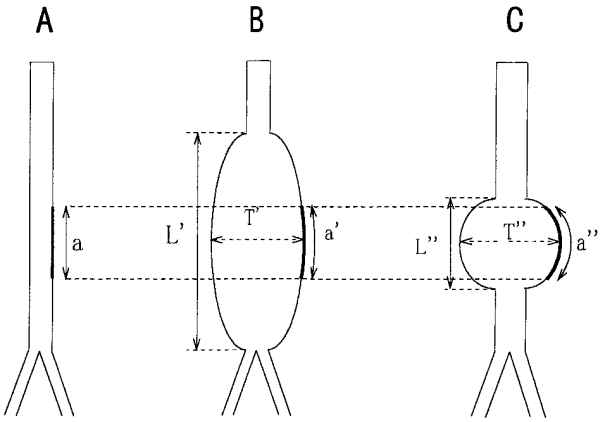

Fig 3. The ratio transverse diameter to length of aneurysm (T/L). The original aortoiliac segment **(A)** is shown with two types of aneurysms, spindle **(B)** and spheric **(C)**. The maximum transverse diameter is identical, and circumferential tension is the same between the two aneurysms. Focusing on the longitudinal tension, segment *a"* is more stretched than segment *a'* when compared with the original segment *a*. This difference in longitudinal tension is easy to evaluate by comparing the ratio T/L (T'/L' < T"/L"). The spheric type has advanced more in longitudinal expansion than the spindle one, if the two aneurysms have same transverse diameter.

As for the cross-sectional area of aneurysm, Cronenwett et al[18] evaluated the elliptic cross-sectional area as a indicator of aneurysmal size in patients with small AAAs who were selected for nonoperative treatment and followed up with sequential ultrasound scanning size measurements. They found that a larger initial size, longer follow-up, and widened pulse pressure predicted larger aneurysmal size at the final evaluation of the study. Wolf et al[19] studied the association between rapid expansion of AAAs and CT scanning variables, including aneurysmal cross-sectional area. The strongest predictor of the rapid expansion was not aneurysmal cross-sectional area but an increased thrombosed load.

Smoking is a well-evidenced risk factor for AAA or aneurysmal expansion. In patients with small AAAs, MacSweeney et al[20] studied growth rates by means of serial ultrasound scanning. They found that growth rates were higher in patients who continued to smoke (0.16 vs 0.09 cm/y in patients who no longer smoked, $P = .038$). They concluded that the cessation of smoking could reduce the growth rate of small AAAs.[20] Cannon and Read[21] studied granular elastolytic activity in serum and leukocytes in patients with and without atherosclerosis. In smokers with AAAs, the activity was significantly increased, whereas serum antiproteolytic capacity was reduced compared with control nonsmokers and patients

with Leriche's syndrome. They concluded that the development of AAAs in patients who smoke correlates with an abnormal homeostasis between proteolytic and antiproteolytic activity. Blue and Janof[22] reported that human polymorphonuclear leukocytes released elastase when incubated with cigarette smoke condensate and that the enzyme remained active in the presence of condensate. Cohen et al[23] studied aortic elastase in rabbits divided into cigarette-exposed and control groups. They found that aortic elastase was significantly higher in the smoke-exposed group than in both cage-control and machine-control groups. They implied that patients with an AAA who smoke may have an accelerated rate of aneurysm growth, earlier rupture, or both. Auerbach and Garfinkel[24] reported that in autopsy series aneurysms were found eight times more frequently among people smoking one to two packets of cigarettes a day than in patients who did not smoke. They stressed the association of cigarette smoking with AAA. Hammond and Garfinkel[25] did a prospective epidemiologic study of more than 1 million men and women. They found that death rates from aortic aneurysm were low among patients who did not smoke and increased greatly with the amount of cigarette smoking. The Aneurysm Detection and Measurement (ADAM) study demonstrated that smoking was the risk factor most strongly associated with AAAs; the odds ratio for AAAs of 4.0 cm or larger compared with normal aortas was 5.57.[26] In summary, smoking may have not only an initiating effect, but also a promoting one. Our results point to the latter effect of smoking.

We could find no data that supported the association between tortuosity and aneurysmal enlargement. Information on tortuosity is scarce, with the exception of the suggestion of its correspondence with atherosclerosis.[27] More information pertaining to the value of 3D tortuosity may emerge from the development of a 3D study–based diagnosis.

Factors that efficiently predicted EX-V included aneurysmal volume and blood urea nitrogen level. This result indicates that the initial aneurysmal size in volume was an important factor; however, volume expansion as a percentage of the original volume did not have better correlation with the factors studied in this study. This might be because another important factor, blood urea nitrogen, has a significant role to predict aneurysmal expansion in volume.

Balm et al[28] studied the efficacy of measurement of aortic lumen volume as a means of evaluating the results of management of transfemoral endovascular aneurysm repair. They advocated that the maximum aneurysm diameter measurement, change in aortic lumen volume, and the thrombus volume might be more appropriate means of discriminating successful from failed exclusion. Again, other information on aneurysmal volume was not found. Data may emerge from future 3D-based studies.

The urea nitrogen level is elevated when protein catabolism increases, such as in critically ill patients.[29,30] An increased level of blood urea nitrogen may be associated with increased protein catabolism in aortic degeneration caused by the development of end-stage atherosclerosis, but our results show the need for a thorough exploration of the possibility. There is no evidence for the hypothesis.

In conclusion, this study demonstrated that the rupture of AAAs could be precisely predicted by a combination of three factors, T/L, EX-D, and diastolic blood pressure. T/L is a novel factor that represents the rate of longitudinal aneurysmal expansion. EX-D could be correctly predicted by means of a combination of three factors, cross-sectional area, tobacco use, and tortuosity. Factors associated with aneurysmal expansion are not identical to those associated with aneurysmal rupture. This might indicate that transverse diameter is no longer the single important value for determining surgical indication. There is well-known evidence that small AAAs can rupture.[6] We think that further studies with 3D reconstruction will reveal the important factor of aneurysmal rupture, such as a significant bleb-like change on the aneurysmal surface.

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