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Increased Peak Wall Stress, but Not Maximum Diameter, Is Associated with Symptomatic Abdominal Aortic Aneurysm

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WHAT THIS PAPER ADDS

Because the presence of symptoms is strongly associated with the risk of rupture, this fact was used to assess the differences between the most commonly used parameter, the AAA maximum diameter, and finite element analysis calculated PWS in identifying symptomatic patients. This study indicates that maximum diameter and PWS are greater in symptomatic than in asymptomatic AAA. However considering patients with a maximum diameter $\geq$ 65 mm alone, only PWS was useful in differentiating symptomatic from asymptomatic AAA.

Objective: Maximum diameter (MD) is the established rupture predictor for abdominal aortic aneurysm (AAA). However, biomechanical markers from finite element analysis (FEA) could be more accurate predictors for these patients. In this study, the association between peak wall stress (PWS) and MD with symptoms of AAA was evaluated.

Methods: Patients diagnosed with infrarenal non-ruptured AAA at the centre between 2009 and 2015 were included. Clinical data, morphological variables (including MD), and the biomechanical variables PWS and diameter normalised PWS (dnPWS) in symptomatic (sAAA) and asymptomatic AAA patients (aAAA) were included.

Results: A total of 170 patients were analysed, 153 aAAA and 17 sAAA. MD was significantly greater in sAAA patients than in aAAA patients (70.4 mm, 95% CI 66.4–86.0 vs. 59.1 mm, 95% CI 53.7–67.8, respectively; $p = .002$). PWS was also significantly higher in the sAAA group (324.6 kPa, 95% CI 217.4–399.5 vs. 199.2 kPa, 95% CI 165.6–239.5; $p < .01$). No differences in MD were found in patients with an AAA $\geq$ 65 mm (43 aAAA and 14 sAAA); however, both PWS (327.4 kPa, 95% CI 239.0–473.3 vs. 229.4 kPa, 95% CI 210.0 to 289.4; $p = .020$) and dnPWS (4.3, 95% CI 3.17–4.67 vs. 3.03, 95% CI 2.8–3.49; $p = .004$) were higher in sAAA than in aAAA.

Conclusions: This study suggests that MD and the biomechanical parameters obtained by finite element analysis are greater in sAAA than in aAAA. However, considering patients with MD $\geq$ 65 mm alone, only PWS, and particularly dnPWS, were able to differentiate sAAA from aAAA.

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INTRODUCTION

An abdominal aortic aneurysm (AAA) is a progressive focal dilatation and weakening of the abdominal aorta and it is the most common type of arterial aneurysm. In adults, an aortic diameter $>$ 3.0 cm is generally considered aneurysmal. The disease is progressive, with growth and rupture.[1,2] A ruptured AAA is life threatening with a high mortality rate and requires immediate repair.[3] Open surgery or endovascular repair are the only treatments currently available for AAA.

Although AAA are usually asymptomatic (aAAA), between 5 and 22% of patients manifest clinical symptoms such as abdominal or back pain, and are termed symptomatic AAA (sAAA).[4] The presence of a symptomatic abdominal aortic aneurysm is generally a harbinger of rupture, and sAAA patients require urgent AAA repair.[5] Regarding aAAA, the decision to proceed with surgical repair is generally determined by assessing the maximum AAA diameter (MD), which is routinely monitored by medical imaging.[6] Elective repair is usually considered when the MD is greater than 55 mm. However, rupture of aneurysms less than 55 mm

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Table 1. AAA demographics.

	All patients			Patients with MD ≥ 65 mm		
	aAAA	sAAA	*p*	aAAA	sAAA	*p*
n	153	17		43	14	
Age	74 (68–79)[a]	77 (69–81)	.29	75 (67–80)	78 (72–83)	.17
Weight (Kg)	75 (70–85)	70 (61–80)	.047	73 (67–82)	67 (60–80)	.082
Height (m)	1.7 (1.65–1.74)	1.7 (1.66–1.71)	.69	1.70 (1.67–1.73)	1.7 (1.66–1.71)	.42
Sex, % (women)	3.9	5.9	.80	2.3	7.1	.99
CSBP (mmHg)	140 (130–146)	140 (138–141)	.92	140 (130–140)	140 (130–140)	.68
CDBP (mmHg)	80 (70–80)	70 (70–80)	.35	70 (70–80)	70 (70–80)	.61
Dyslipidaemia (%)	54.9	41.2	.41	55.8	35.7	.32
HTN (%)	76.5	76.5	.76	69.8	71.4	.82
Diabetes (%)	21.6	17.6	.94	23.3	14.3	.73
Smokers (%)	25.5	18.8	.78	2.9	15.4	.97
PAD (%)	37.9	38.5	.80	46.5	4.0	.99
BVD (%)	7.2	11.8	.85	7.0	14.3	.77
IHD (%)	26.8	35.3	.65	16.3	35.7	.24
COPD (%)	15.7	17.6	.88	18.6	21.4	.87

BVD = brain vascular disease; CDBP = chronic diastolic blood pressure; COPD = chronic obstructive pulmonary disease; CSBP = chronic systolic blood pressure; HTN = chronic hypertension; IHD = ischaemic heart disease; PAD = peripheral artery disease.

[a] Non-normally distributed quantitative absolute data are expressed as median (25–75%).

diameter has been reported, suggesting that the risk of aneurysm rupture is not determined by MD alone.[7,8]

Mechanisms leading to AAA rupture remain unclear. Many studies report that the difference between complicated AAA (sAAA and ruptured AAA) and aAAA is primarily biomechanical wall stress. Peak wall stress (PWS) evaluated using computer modelling through finite element analysis (FEA) is a useful parameter for predicting the risk of rupture,[9–12] with PWS being greater in symptomatic or ruptured AAA than in asymptomatic intact AAA.

Since symptomatic aneurysms harbour an increased risk of rupture, the aim of this study was to compare the ability of biomechanical parameters from FEA and MD to differentiate between symptomatic and asymptomatic AAA patients.

METHODS

Patients

Between 2009 and 2015, consecutive patients diagnosed with infrarenal AAA were included in the study. The diagnosis of AAA was confirmed by computed tomography (CT) scan. Exclusion criteria included unsuitable computed tomography angiography (CTA) for FEA analysis, juxtarenal aneurysms (since the presence of adjacent visceral arteries on the imaging studies can complicate FEA analysis), mycotic aneurysms, and ruptured aneurysms (confirmed by the presence of free blood in the abdominal space on CTA). Patients with symptoms were included in the sAAA non-ruptured group. sAAA was considered when the patient had an intact AAA on CT scanning and current onset back, abdominal, or groin pain not identified to be from other causes.[5] To exclude other causes of pain, blood analysis, radiological, or ultrasound examinations were obtained when needed. An emergency physician also confirmed the differential diagnosis.

Only strictly necessary clinical data from patients were used, obtained from the informatics database of the hospital. All data were collected by the same investigator, and were stored on a computer using a personal key. The data included the clinical history of various comorbidities, including diabetes mellitus (DM, all types), systolic and diastolic blood pressure, dyslipidaemia (total cholesterol >200 mg/dL), cerebrovascular disease (history of stroke, transient ischaemic attack, or major neurological deficit), heart disease (history of myocardial infarction, angina pectoris, or previous coronary intervention), lung disease (chronic obstructive pulmonary disease), smoking (during the last year), and peripheral artery disease. Each patient's weight and height were also recorded (see Table 1).

The protocol was approved by the institution's review board (protocol code, IIBS-FIN-2013-89). As this was a retrospective case series analysis, informed consent was not deemed necessary.

Finite element analysis

FEA was performed on the CTA of all patients, using A4clinics-Research Edition software (VASCOPS Vascular Diagnosis Company, Graz, Austria). The analysis was performed by a single member of the group (B.S.) to avoid inter-observer errors.

The three dimensional AAA geometry was acquired from routine CTA imaging data. The lumen, intraluminal thrombus, and external wall data were acquired separately and semi-automatically. The program includes a manual correction feature if some special point is found, such as a penetrating ulcer or some other unusual anatomy. The resultant geometry is subdivided into multiple contiguous elements that form a fine mesh. The AAA is ready for wall stress computation after the appropriate material properties of the AAA wall and components have been specified by using the computational software. The end result is aneurysm specific wall stress distribution.[9,12]

In all cases, the segment from the infrarenal aorta to the iliac bifurcation was analysed. The morphological variables

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