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Wall Stress Analysis in Small Asymptomatic, Symptomatic and Ruptured Abdominal Aortic Aneurysms

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Objectives. *To evaluate the potential of wall stress analysis for the identification of abdominal aortic aneurysm (AAA) at elevated risk of rupture in spite of small diameter.*
Materials and methods. *Thirty patients with small AAA, 10 asymptomatic, 10 symptomatic and 10 ruptured, were included. Demographic data and results from physical examinations were recorded in a retrospective fashion. After CT-evaluation and the creation of a patient specific 3D model, wall stress was calculated using the finite element method.*
Results. *No differences were observed in diameter between asymptomatic, symptomatic or ruptured aneurysms (5.1 ± 0.2 cm vs. 5.1 ± 0.2 cm vs. 5.3 ± 0.2 cm respectively; $p = 0.57$). Peak aortic wall stress at maximal systolic blood pressure is significantly higher in ruptured than asymptomatic aneurysms (51.7 ± 2.4 N/cm^2 vs. 39.7 ± 3.3 N/cm^2 respectively; $p = 0.04$). Wall stress analysis at uniform blood pressure, performed to correct for higher blood pressure in the symptomatic and rupture group did not result in significant differences in peak wall stress (asymptomatic 31.7 ± 2.3 N/cm^2; symptomatic 30.5 ± 1.3 N/cm^2; rupture 36.7 ± 4.0 N/cm^2; $p = 0.26$).*
Conclusions. *Wall stress analysis at maximal systolic blood pressure is a promising technique to detect aneurysms at elevated aneurysm rupture risk. Since no significant differences were found at uniform blood pressure, the need for adequate blood pressure control in aneurysm patients is reiterated.*

Keywords: Aortic aneurysm, abdominal; Aneurysm, ruptured; Stress, mechanical; Tomography, spiral computed; Imaging, three-dimensional.

Introduction

The prevalence of Abdominal Aortic Aneurysm (AAA) is estimated between 4.1% and 14.2% in men over the age of 60.[1] Although prevalent, 88–99% of all aneurysms detected at screening are small (<5.5 cm).[2–5] The clinical relevance and optimal management strategy for these small aortic aneurysms have long been the subject of controversy. Results from two randomized controlled trials have shown the safety of surveillance in AAA smaller than 5.5 cm.[6–8] These results were supported by the low annual rupture risk (0.6–1%) observed in the surveillance groups. However, since these low rupture rates are partly explained by meticulous follow-up and a high surgical intervention rate (over 60%), they do not seem to reflect the natural history of small abdominal aneurysms.[9] Results from autopsy studies show that 10–24% of all ruptured AAA have an aneurysm diameter less than 5.5 cm.[10,11] The simple observation, that even small aneurysms rupture questions the use of maximum diameter as the single threshold for surgical intervention and focuses the attention to other patient, or aneurysm specific variables likely to affect aneurysm rupture risk.

AAA rupture occurs when the stress (force per unit area) on the aneurysm wall exceeds wall strength. The first studies on aortic wall stress could not demonstrate local variations in wall stress due to the use of hypothetical, symmetrical geometrical models.[12–14] Recent advancements in Computed Tomography (CT) and image post-processing allow the reconstruction of patient specific three dimensional aneurysm

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models. The use of these models and the availability of a sophisticated mathematical technique, Finite Element Analysis (FEA), have improved wall stress analysis and its clinical applicability.[15] With the use of this technique, Fillinger and others have shown that peak wall stress is significantly higher in ruptured than electively repaired AAA.[16,17] In another series, 103 patients under observation had wall stress analysis. All 22 patients presenting with symptoms of pending aneurysm rupture or actual rupture, including 5 patients with small AAA (<5.5 cm), were found to have significantly higher peak wall stresses well in advance of aneurysm rupture. The authors therefore postulated that wall stress analysis has the potential to detect small aneurysms that cannot be observed safely because of high rupture risk.[18] The purpose of this study is to evaluate the potential of wall stress analysis to detect patients at elevated risk of aneurysm rupture in spite of small aneurysm diameter.

Materials and Methods

Patient population

The study included thirty patients with small AAA, 10 asymptomatic, 10 symptomatic and 10 ruptured. After a review of medical records from two institutions 20 patients with a CT scan evaluation of a small ruptured ($n = 10$) or symptomatic aneurysm ($n = 10$) were identified. Two patients in both the rupture and symptomatic group had a CT evaluation while asymptomatic but experienced rupture or symptoms (e.g. severe back pain) within the following 6 months. All patients evaluated at the time of acute aneurysm related symptoms or aneurysm rupture had stable conditions.

Patients in the asymptomatic group ($n = 10$) had a CT evaluation for elective aneurysm repair and remained asymptomatic for at least 6 months; until the end of the study in march 2006 or until elective repair (3/10). All CT scans were performed during routine care between January 2003 and March 2006 and no CT scan was obtained for the purpose of performing stress analysis.

Patients medical records were reviewed for demographics, medical history and blood pressure data. For all patients in the asymptomatic group and most patients in the ruptured and symptomatic group blood pressure was recorded from the year prior to CT evaluation. For 3 patients in the ruptured group and 2 patients in the symptomatic group this data was unavailable and blood pressure was recorded at the time of CT evaluation. All data acquisition was performed after approval by the local ethics committee.

AAA morphology and patient body habitus

Overall indices of patient body habitus (e.g. body mass index) are recorded from medical records. Possible or known indices of AAA geometry related to aneurysm rupture, like AAA diameter, diameter asymmetry and the diameter of the body of vertebra L3 are measured and calculated from CT data. Aneurysm diameter asymmetry is defined as the difference between major and minor axis diameter.

Wall stress analysis

In vivo aneurysm wall stress is computed by using the Finite Element Method (FEM). The basic approach of the finite element method is to divide a complex geometrical structure (AAA) into smaller pieces or elements. These elements are connected by nodes. The entire network of elements and nodes is called a mesh. Wall stress is determined by predicting movement of the nodes which are influenced by the material properties of the aneurysm wall (e.g. stiff or elastic) and preset boundary conditions (e.g. blood pressure). Wall stress analysis therefore requires three main components: the in vivo three dimensional aneurysm model to create the mesh, the boundary conditions and a material model that describes the mechanical properties of the aneurysm wall.

AAA geometry is derived from contrast enhanced spiral CT data. For all patients, including those who experienced rupture, the aneurysm outer wall could be identified and depicted by image post-processing (segmentation of bloodflow, thrombus and calcified plaques). This resulted in a patient specific three dimensional in vivo aneurysm model. From these models the mesh is created by using a previously described semi-automated process.[17,18] In brief; dividing the original model into large amounts of elements (the mesh), results in highly accurate stress analysis. However, by using large amounts of elements computation time is long. Our typical meshes currently contain approximately 35 000−45 000 elements. This results in accurate measurements with acceptable computation time (up to 60 minutes). Increasing the number of elements beyond this number changes computed wall stress less than 2%, but significantly augments computational time. Besides the number of elements the element shape is important. Odd shaped elements (e.g. severe angles) might result in computational errors. To eliminate these elements

a refinement algorithm, based upon multiple iterations, was designed. Ultimately, this refinement was incorporated into the initial mesh generation, and this new method was validated using prior datasets before using it in this study. Finally, the refined mesh is entered into a commercially available software program for finite element analysis (ABAQUS v.6.5, Hibbit, Karlsson and Sorensen, Inc, Pawtucket, RI).

The material model is a set of equations that characterize the relation of aneurysm wall movement and the forces acting on the wall. Forces acting on the aneurysm wall result in large deformations or strains. To account for these large strains we use a previously described and validated isotropic hyperelastic nonlinear model. This model is based upon the mechanical properties of abdominal aortic aneurysms in a series of 69 patients.[19]

The FEM boundary conditions consist of the mechanical load on the wall (e.g. blood pressure) and possible physiological constraints (e.g. iliac and renal arteries) to wall movement. Bloodflow creates shear stress on the lumen surface, these shear stresses are however negligible compared to the stresses due to blood pressure. Therefore peak wall stresses reported in this study are all at systolic blood pressure. An additional analysis at uniform blood pressure (120 mmHg) is performed to rule out possible differences in maximal systolic blood pressure between groups and to evaluate the effects of blood pressure on wall stress analysis.

Statistical analysis

The three groups (asymptomatic, symptomatic and ruptured) were compared by using SPSS v12.0.1, Chicago, ill. Continuous variables were compared with analysis of variance (ANOVA) with post hoc analysis. Nominal data was analyzed using contingency table analysis and Chi-square or Fischer exact tests. All continuous data is reported as mean $\pm$ SE.

Results

Demographics

No statistical significant differences were found between the three groups with respect to age, hypertension (treated or >140 mmHg systolic), diabetes (treated with insulin, oral medication or diet adjustment), chronic obstructive pulmonary disease (treated or not), stroke (TIA or CVA), use of cardiovascular and anti-inflammatory medication, peripheral vascular disease or current smoking (Table 1). The only two variables that reached significance are the use of statin for hypercholesterolemia (asymptomatic 7/10, symptomatic 1/10 and rupture 2/10; $p = 0.01$) and the use of diuretics; less frequent in the symptomatic group (asymptomatic 7/10, symptomatic 1/10 and rupture 6/10; $p = 0.02$). There was a trend towards more ischemic heart disease in the asymptomatic group but this did not result in statistically significant differences ($p = 0.054$).

Gender differences were noted but did not reach significance between groups nor after combining the ruptured and symptomatic group (asymptomatic 1/10 female vs. rupture/symptomatic 6/20 female; $p = 0.23$).

AAA morphology and patient body habitus

Maximal (major axis) aortic diameter is not significantly different between the three groups (asymptomatic 5.1 ± 0.2 cm; symptomatic 5.1 ± 0.2 cm; rupture 5.3 ± 0.2 cm; $p = 0.57$). The only variable of body habitus and aneurysm dimension reaching marginal significance is aneurysm diameter asymmetry showing more asymmetry in the asymptomatic group. Variables of aneurysm and patient morphology are listed in Table 2.

Peak wall stress and blood pressure

Peak aortic wall stress at maximum systolic blood pressure is significantly different between the

Table 1. Demographics and use of medication

	Asymptomatic $N = 10$	Symptomatic $N = 10$	Rupture $N = 10$	P value
Age (years)	72 ± 2	75 ± 3	70 ± 2	.44
Gender (M/F)	9/1	7/3	7/3	.48
CAD[a]	7	2	3	.05
CHF[b]	1	2	2	.79
Hypertension	7	10	8	.19
Diabetes	1	3	1	.38
COPD[c]	3	4	2	.62
Stroke	2	0	2	.32
ABI[d]	4	2	2	.51
Smoking (current)	4	3	4	.87
Aspirin use	5	4	4	.87
Betablocker use	6	5	2	.17
Calcium antagonist use	0	1	2	.33
Diuretics use	7	1	6	.02
Statin use	7	1	2	.01
Steroid use	1	0	2	.33

[a] CAD, Coronary Artery Disease.
[b] CHF, Chronic Heart Failure.
[c] COPD, Chronic Obstructive Pulmonary Disease.
[d] ABI, Ankle Brachial Index (<1.0).

Table 2. AAA morphology and patient habitus

	Asymptomatic $N=10$	Symptomatic $N=10$	Rupture $N=10$	P value
AAA diameter (cm)	5.1 ± 0.2	5.1 ± 0.2	5.3 ± 0.2	.57
Asymmetry[a]	0.6 ± 0.1	0.5 ± 0.1	0.3 ± 0.1*	.09
AAA/TC[b]	1.8 ± 0.1	1.7 ± 0.1	1.9 ± 0.1	.15
AAA/L3[c]	0.9 ± 0.8	0.9 ± 0.8	1.1 ± 0.9	.16
BMI[d]	23.0 ± 1.5	24.6 ± 0.7	23.9 ± 0.9	.61

* AAA in rupture group less asymmetric ($p = 0.05$).
[a] Asymmetry, major axis diameter minus minor axis diameter.
[b] AAA/TC, major axis AAA diameter/aorta diameter at level of Truncus Coeliacus.
[c] AAA/L3, major axis AAA diameter/diameter corpus vertebra L3.
[d] BMI, Body Mass Index; Weight (kg)/Length^2 (cm).

asymptomatic and rupture group (39.7 ± 3.3 N/cm^2 vs. 51.7 ± 2.4 N/cm^2 $p = 0.04$). Peak wall stress in the symptomatic group was also higher compared to the asymptomatic group although this did not reach significance (Table 3). Fig. 1 shows the result from wall stress analysis for a ruptured aneurysm. Note the resemblance in location of aneurysm rupture and peak wall stress.

Maximum systolic blood pressure is significantly higher in the symptomatic group (asymptomatic 147 ± 5 mmHg; symptomatic 181 ± 7 mmHg; rupture 165 ± 7 mmHg; $p = 0.004$). The additional analysis of wall stress at uniform blood pressure (120 mmHg), performed to compensate for these difference in maximal systolic blood pressure, did not result in statistical significant differences in aneurysm wall stress between the three groups. (asymptomatic 31.7 ± 2.3 N/cm^2; symptomatic 30.5 ± 1.3 N/cm^2; rupture 36.7 ± 4.0 N/cm^2; $p = 0.26$).

Table 3. Wall stress and blood pressure

	Asymptomatic $N=10$	Symptomatic $N=10$	Rupture $N=10$	P value
Peak Stress (N/cm^2) max. systolic BP[b]	39.7 ± 3.3	47.6 ± 2.7	51.7 ± 2.4[a]	.11
Stress (120 mmHg)	31.7 ± 2.3	30.5 ± 1.3	36.7 ± 4.0	.26[c]
Max. systolic BP	147 ± 5	181 ± 7[d]	165 ± 7	.004

[a] Peak wall stress in ruptured AAA significantly higher than in asymptomatic AAA ($p = 0.04$).
[b] BP, Blood Pressure.
[c] For all three groups ($p = 0.26$). Asymptomatic vs. ruptured ($p = 0.21$).
[d] Max. systolic BP significantly higher for symptomatic vs. asymptomatic patients ($p = 0.001$).

Discussion

Observation of small asymptomatic (<5.5 cm) infrarenal aortic aneurysms is considered safe in the general population. Some small aneurysms however do rupture. Wall stress analysis has been proposed to identify patients with elevated risk of aneurysm rupture in spite of small diameter. This is the first study directly comparing aortic wall stress in small asymptomatic, symptomatic and ruptured aneurysms. Results from this study show that peak wall stress at maximal systolic blood pressure is different for asymptomatic and ruptured small aortic aneurysms. These results therefore seem to confirm the observation by Fillinger, that the use of peak aortic wall stress could improve the selection of patients for aneurysm repair and the timing of surgical intervention.[17,18] Although this is an important finding, there is a remarkable difference between the previous studies on wall stress analysis and the present work. In contrast with previous work, significant differences in peak wall stress did not persist after analysis at uniform blood pressure. This has important consequences and requires explanation.

First, it stresses the importance of blood pressure control. Hypertension is a well known risk factor for aneurysm rupture and seems equally important in wall stress analysis. Eight patients in both the symptomatic and rupture group had a CT evaluation while experiencing rupture or symptoms of pending aneurysm rupture. Calculating wall stress using blood pressure data from the time of CT evaluation could therefore lead to falsely lower wall stress in the rupture group due to hemodynamic impairment. Conversely, wall stress calculations using elevated blood pressure at the time of symptoms increases wall stress due to pain and distress. To correct for this selection bias we used blood pressure data from the year prior to CT evaluation. Since some patients presenting with adverse outcome were never evaluated before, this data was unavailable for 3 patients in the ruptured and 2 patients in the symptomatic group. For these patients wall stress was calculated using blood pressure from the time of CT evaluation. The effect of this bias seems however limited as wall stress is elevated for ruptured AAA and not significantly higher for patients experiencing symptoms.

More complex is the effect of this blood pressure related bias on wall stress analysis at uniform (120 mmHg) blood pressure. Including patients with ruptured AAA and subsequent low blood pressure (<120 mmHg) could lead to an increase of wall stress computed at uniform blood pressure, increasing the probability of finding significant differences between

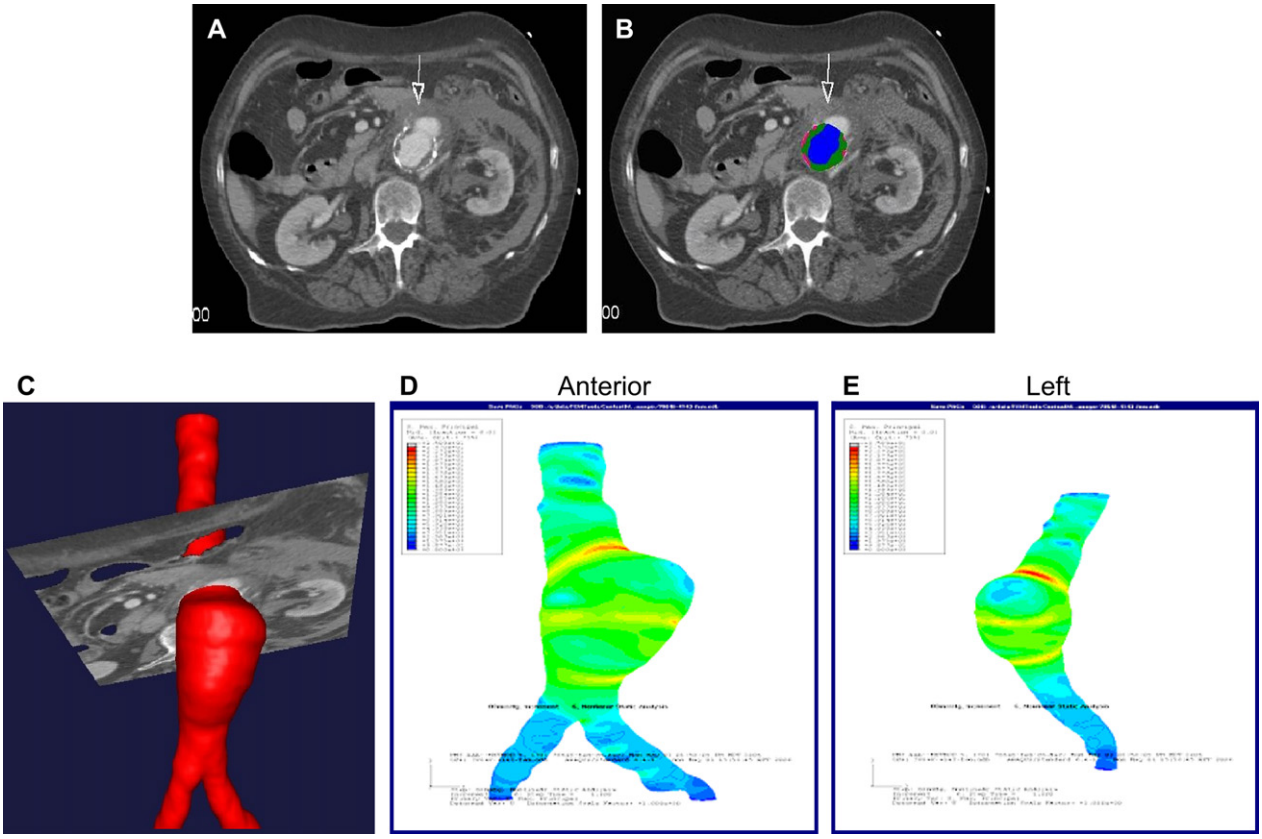

Fig. 1. A. Rupture located left anterior. B. Result from segmentation process, note the hematoma is not included in the segmentation. C. Corresponding slice within 3D model. D and E. Red area location of maximal wall stress.

ruptured and non-ruptured AAAs. However, in the current analysis all patients with ruptured AAA presented with blood pressures over 120 mmHg. This does not explain the difference between the present and previous studies on wall stress as Fillinger also analyzed patients in stable conditions only, but it does reemphasize the importance of blood pressure in wall stress analysis.[17]

Second, if blood pressure is uniform, differences in computed wall stress are based upon variations in aneurysm geometry. Since no significant differences in wall stress were found between asymptomatic, symptomatic and ruptured aneurysms at uniform blood pressure, AAA geometry has to be relatively uniform. This might be due to the fact that we studied small aortic aneurysms. During aortic dilatation the aneurysm is remodelled, elastin fibres are degraded, and collagen is synthesized. If dilatation is more prominent (larger aneurysms) this remodelling might cause more dramatic shape changes possibly leading to larger differences in peak wall stress. Previously, diameter asymmetry has been associated with elevated rupture risk and increased wall stress.[20,21] Remarkably, our ruptured aneurysms showed less diameter asymmetry. A possible explanation for this finding is the high portion of ruptured aneurysms (8/10) at the time of CT-evaluation. Aneurysm rupture could result in the formation of hematoma, distorting the original geometry (asymmetry) and obscuring subtle anatomical characteristics known to affect aneurysm rupture risk, like blebs and blisters.[22]

Besides these limitations related to study design, several opportunities exist to refine the material model. Currently, the model is based upon several assumptions (material isotropy and homogeneity) and does not account for intra-luminal thrombus (ILT) or calcification. The effect of ILT on aneurysm rupture and wall weakening is controversial.[23] Some investigators have observed focal aneurysm wall hypoxia and subsequent wall weakening in the presence of ILT.[24] Others have suggested a reduction in aneurysm rupture risk related to a possible ILT 'cushioning effect' decreasing pressure transmission.[25,26] Schurink however, measured in vivo mean arterial and pulse pressure near the aneurysm wall and found no reduction in pressure transmission to the aneurysm wall in the presence of thrombus.[27] Therefore, the effects of thrombus on wall stress seem limited and the main

limitations of the current material model are the assumptions made at initial development; aneurysm wall isotropy and material homogeneity.[19]

Materials are called isotropic when the physical properties are identical for any given direction. This is probably not true for the aneurysm wall as recent experiments have shown preferential stiffening in the circumferential direction (anisotropy).[28,29] Although the introduction of aneurysm wall anisotropy could result in more accurate stress reports the effect of this new finding on the finite element assessment of aneurysm rupture risk remains to be investigated.

The assumption of material homogeneity includes, uniform wall thickness and identical material properties for every aneurysm. Several authors have addressed this phenomenon and investigated the effect of variations in wall thickness and changed material properties on computed wall stress and aneurysm rupture risk.[30–32] Local alternations in wall thickness could result in increased wall stress and variations in material properties like aneurysm tensile strength (maximal stress before aneurysm rupture).[30]

Since aneurysm rupture occurs when wall stress exceeds wall strength, future material models should include patient specific information on aneurysm wall strength. Although this will substantially refine rupture risk assessment, wall stress analysis using the current material model is already superior to diameter in differentiating patients at elevated risk of rupture.[18]

Conclusion

Wall stress at maximal systolic blood pressure is significantly higher for ruptured compared to asymptomatic aneurysms. This confirms the potential of wall stress analysis as a promising technique to detect small aneurysms at elevated risk of rupture. Analysis at uniform blood pressure resulted in less pronounced and non-significant differences in wall stress between asymptomatic and ruptured small aneurysms. This reiterates the need for strict blood pressure control to reduce peak aortic wall stress and aneurysm rupture risk. Larger prospective follow-up programmes will be needed to confirm our findings and to investigate the effect of aneurysm growth and blood pressure change over time on aortic wall stress.

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