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Aortic Wall Thickeness as a Predictor of Acute Aortic Dissection

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Methods: Aortic wall cohesion of 496 patients divided into two groups according to AWT was analyzed using the Dissectometer, a device mimicking transverse shear stress. Correlation of cohesion testing (P7,P8,P9), histology as well as diameter of the ascending aorta with AWT were analyzed.

Results: AWT > 2.28mm was associated with decreased aortic cohesion (P7:131.7±66.3 vs.153.7±89.5 p=0.02; P8:2.95±1.55 vs.3.78±1,90 p<0.01; P9:4.22±1.75 vs.4.94±2.12 p<0.01) and increased media degeneration (45.8%vs.15.8% p<0.01) compared to AWT ≤ 2.28mm. Diameter of ascending aorta did not correlate with AWT (p=0.20). Majority of patients with AD presented with normal aortic diameter (13/18, 72.2%) and had an AWT > 2.28mm (15/18, 83.3%).

Keywords: *aortic wall, dissection, aortic wall cohesion testing.*

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Conclusions: Aortic wall thickness>2.28mm appears to correlate with decreased aortic cohesion as well as histological signs of aortic wall instability irrespective of aortic diameter.

Keywords: aortic wall, dissection, aortic wall cohesion testing.

I. Introduction

Acute aortic dissection (AD) is a serious disease with high morbidity and mortality, regularly presenting without any prognostic symptoms, but nevertheless being associated with underlying aortic wall pathology [1,2].

In addition to spontaneous dissection without triggering injury, acute aortic complications occur in approximately 0.16% - 0.35% of patients undergoing cardiac surgery as well as following other interventions involving aortic manipulation including intra-aortic balloon pump (IABP) insertion [3]. Currently, prediction of individual risk for future aortic dissection is mainly based on aortic diameter as well as history of connective tissue diseases (e.g. Marfan syndrome) or aortic valve abnormalities (e.g. bicuspid valve) [2]. Although it has been established that aneurysm size has a profound impact on risk of rupture, dissection and death, large studies have shown that a significant proportion of patients developing acute aortic dissection have a normal or only marginally enlarged aortic diameter [3]. Our current means of risk stratification for aortic dissection or rupture are therefore suboptimal, and prophylactic aortic replacement based solely on aortic diameter appears to be an insufficient strategy [4,5]. There is therefore a need to develop further diagnostic tools to predict the risk of future aortic complications. Aortic enlargement with resultant wall thinning was believed to be an important factor increasing wall stress and leading to aortic rupture or dissection. Therefore, it was the aim of this study to test the hypothesis that the aortic wall thickness (AWT) correlates to histological or clinical signs of aortic wall instability and cohesion as assessed by the Dissectometer device [6].

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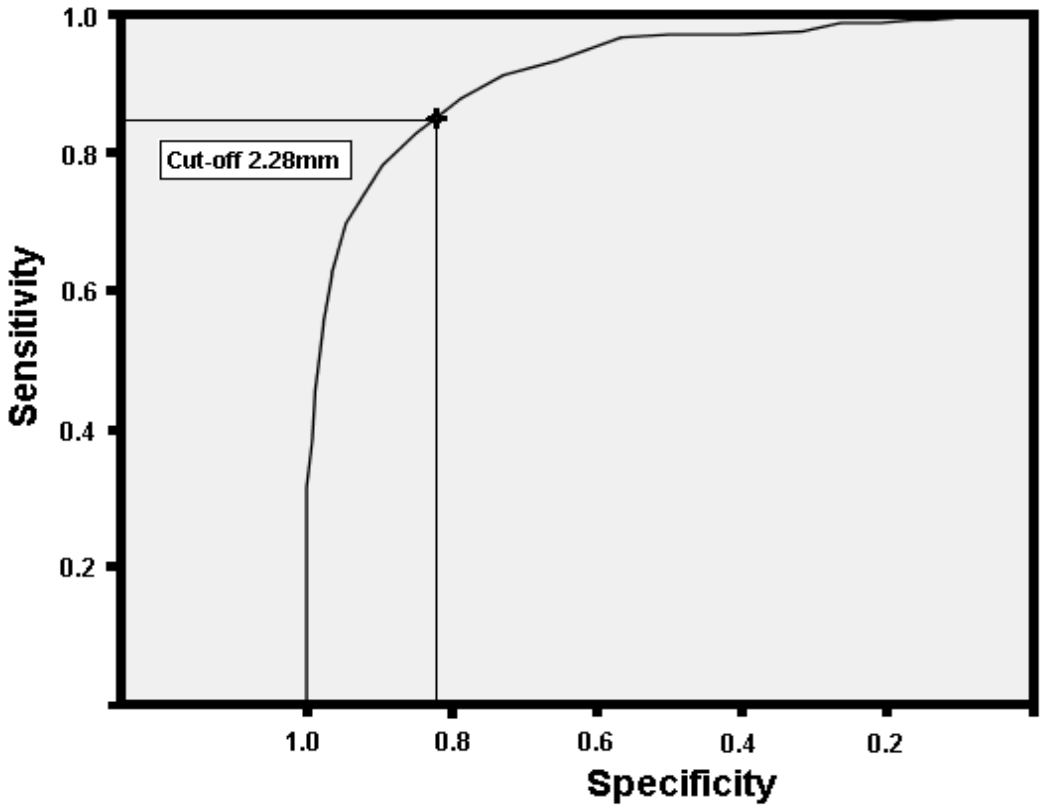
II. Materials and Methods

a) Study design

The study was approved by the Institutional Review Board and patients' written informed consent was obtained. This single-center, non-randomized study enrolled 496 consecutive patients undergoing surgery for aortic valve stenosis (AS) or regurgitation (AR), aortic aneurysms (AA) and coronary artery bypass grafting (CABG) (including concomitant procedures) at the West-German Heart Center Essen between March 2010 and December 2013. Patients with acute aortic dissection (ADD) were only included in this study if a piece of the aortic wall could be resected for histological and cohesion testing that was clearly unaffected by dissection.

The study population was divided into two groups according to aortic wall thickness: Group 1: AWT ≤ 2,28mm (n=260) and group 2: AWT > 2,28mm (n=236). The optimal cutoff level was defined by the largest sum of sensitivity and specificity of the AWT for histological signs of aortic wall instability using ROC analysis (Cut-off 2,28 mm: sensitivity 83% and specificity 85% see Fig. 1).

Figure 1



ROC curve - Sensitivity and specificity for cut-off 2.28mm

b) Sample collection

Surgery was carried out through a median sternotomy using cardiopulmonary bypass with ascending aortic cannulation. In patients with aortic valve disease or aortic aneurysm, a sample of the aortic wall was harvested from the edge of the aortic incision site (~ 3cm above the aortic valve), as previously described [6]. In patients undergoing isolated CABG, the sample of aortic wall was harvested using a rounded scalpel. The resulting hole was then used as the insertion point for a vein graft. In patients with acute type A dissection, only those patients in whom sufficient specimens of non-dissected aorta allowing for measurement of thickness and cohesion were included in the study.

The aortic sample was immediately placed in cold saline until the cohesion test was performed (within 2 hours of collection). Aortic wall thickness was measured immediately before cohesion testing using a micrometer (Kometex B.V./Hogetex, Netherlands).

c) Intraoperative echocardiography

TOE was performed with a multiplane 2.9–6.7 MHz (6T-RS) phased-array probe (Vivid i, GE Healthcare, Milwaukee, WI, USA) using a standardized protocol prior to cardiopulmonary bypass in all patients. The following aortic dimensions were measured: diameter of the aortic annulus, aortic sinuses, sinotubular junction and ascending aorta.

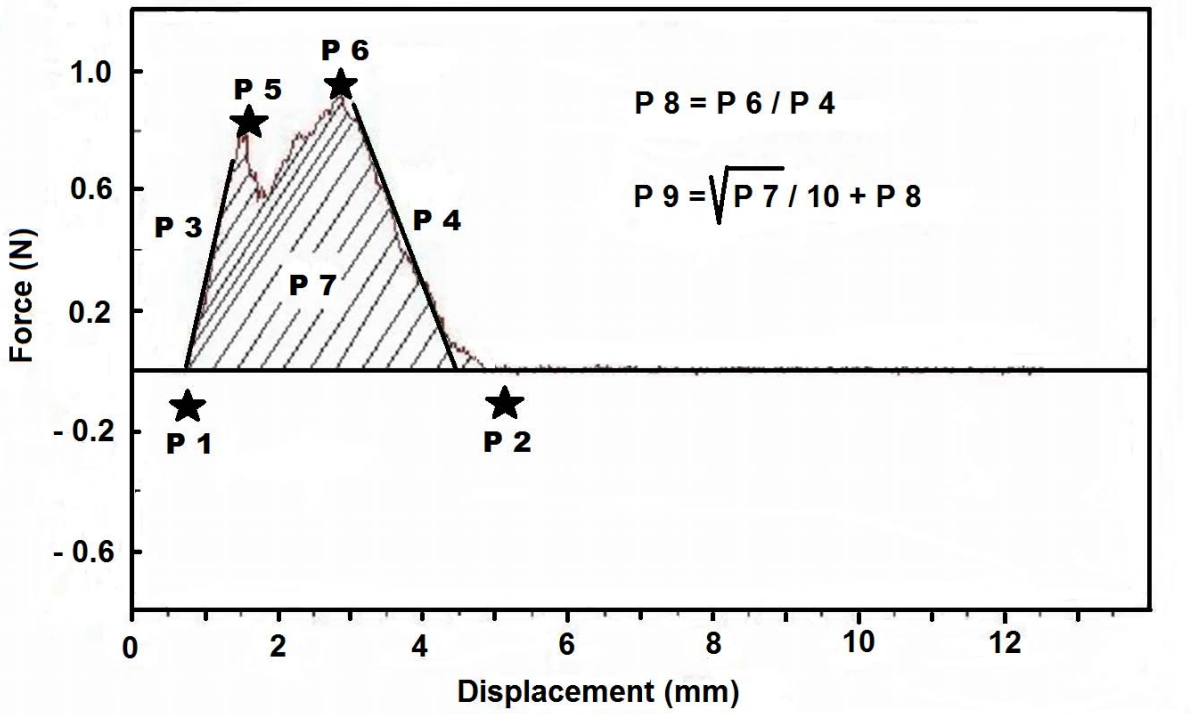
d) Aortic wall cohesion testing

Aortic wall cohesion testing was performed using the Dissectometer, a device mimicking transverse shear stress (simulating the acute dissection process), as previously described [6]. Results of the dissection were visualized as tensile strain curves (TSC), which were subsequently converted to numerical parameters.

P1, P2, P5 and P6 correspond to points on the curve. P1 (mm) is the beginning of the positive deviation – the point when the Dissectometer registers the tension in the sample. P2 (mm) is the point of the dissection and the power has a value of zero. P5 (N) is the first power maximum (at this point the power has decreased temporarily). After this point the aortic wall sample is damaged irreversibly. P6 (N) represents the "dissection limit" after which the power necessary to disrupt the aorta decreases. P3 ($N.mm^{-1}$) is the angle of the line between P1 and P5. This characteristic describes the elasticity of the aortic wall – the sharper the angle, the greater the elasticity of the aorta. P4 ($N.mm^{-1}$) is the angle of the power decrease, which characterizes the cohesion of the aortic wall. P7 (N.mm) represents the area under the TSC, which describes the total cohesion of the aorta. These seven parameters were used to mathematically derive the next two parameters, P8 and P9. P8 is described as the "dissection tendency" (calculated as the maximal force divided by the downward angle) and P9 as the "dissection potential"

(calculated as the sum of P8 and the square root of P7 divided by ten). The parameters with the highest sensitivity and specificity for discriminating between histologically stable and unstable aortic wall identified in a previous study (P7, P8 and P9) were analyzed in the present study (Fig. 2) [7]. One observer blinded to all patient data performed all cohesion tests.

Figure 2



Tensile strain curve – Localization of the parameters P1 - P7; mathematical formula for P8 and P9.

e) Histological examination

All samples were collected in 4% buffered Formalin, embedded in paraffin and cut to micrometer sections. These sections where stained with Hematoxilin and Eosin, and Elastica van Giessen. Histological examination was performed by an independent, blinded pathologist. The aortic wall was categorized using an integrated approach, by evaluating the media according to presence of vacuolization and texture disturbances as "pathological/media degeneration", or "intact /minimal changes". Media degeneration was defined as fibrosis of the media with fragmentation and/or loss of elastic fibers and increased deposition of proteoglycans. Media disruption was defined as disruption of elastic fibers in the media of the aorta.

f) Statistics

Descriptive statistics are summarized for categorical variables as frequencies (%). Pearson's χ^2 or Fisher´s exact tests were used for comparisons between groups. Continuous variables are reported as mean ± standard deviation and were compared using the Student´s *t*-test or Mann-Whitney U test. *P*-value of < 0.05 was considered to indicate statistical significance. All statistical analyses were performed using the SPSS System®, version 19.0 (IBM Corp., Armonk, NY, USA).

III. Results

Out of 496 patients, 260 had an ≤ AWT 2,28mm, while 236 patients presented with an AWT > 2,28mm. There were no difference in demographics and prevalence of comorbidities (age, gender, diabetes mellitus, chronic kidney insufficiency, hypercholesterolemia and chronic obstructive pulmonary disease) between the two groups (Table 1). Type of surgery and proportion of histology positive for aortic wall instability are summarized in table 2. A total of 109 patients underwent replacement of the aortic valve due to aortic stenosis (slightly more than in Group 2, $p = 0.07$). Coronary artery revascularization was performed in 285 patients ($p = 0.87$), in 134 cases as isolated procedure. 18 patients underwent surgery for aortic dissection (significantly more in Group 2, $p < 0.01$). While only 41 (15.8%) patients in Group 1 showed histological signs of aortic wall instability, the aortic wall of 108 (45.8%) patients in Group 2 was classified as histologically unstable ($p < 0.01$).

Table 1 : Demographics

n=496	Group1 (n=136)	Group 2 (n=360)	*P*-value*
Age (years)	65.0±12.9	65.8±12.7	0.69
Female	48 (35.3)	96 (26.7)	0.06
Hypertension	110 (80.9)	315 (87.5)	0.06
DM	28 (20.6)	66 (18.3)	0.57
Renal insufficiency	19 (14.0)	33 (9.2)	0.12
Hypercholesterolemia	67 (49.3)	190 (52.8)	0.49
COPD	14 (10.3)	37 (10.3)	1.00

*Data are presented as mean±SD or number (%); DM, Diabetes mellitus; COPD, Chronic obstructive pulmonary disease; *, group 1 versus group 2.*

Table 2 : Operation diagnoses and positive histology

n=496	Group 1 (n=136)	Group 2 (n=360)	OR	*P*-value*
AS	62 (45.6)	115 (31.9)	0.77	<0.01
AR	31 (22.8)	101 (28.1)	1.2	0.24
AA	23 (16.9)	87 (24.2)	1.3	0.08
CAD	74 (54.4)	211 (58.6)	1.0	0.40
Dissection	1 (0.7)	17 (4.7)	6.2	0.03
Positive Histology	24 (17.7)	161 (44.7)	2.1	<0.01

Data are presented as number (%); OR, Odds ratio; AS, aortic stenosis; AR, aortic regurgitation; AA, ascending aneurysm; CAD, Coronary artery disease;, group 1 versus group 2.*

Echocardiographic and Dissectometer-derived results are summarized in table 3, showing that aortic diameter as assessed by TOE (i.e. the annulus, aortic sinuses, sinotubular junction and ascending aorta) did not differ between the two groups. We observed statistically significant differences in aortic wall cohesion between the Group 1 and Group 2 as demonstrated by Dissectometer testing (P7: 153.7 ± 89.5 vs. 131.7 ± 66.3, $p < 0.02$; P8: 3.78 ± 1.90 vs. 2.95 ± 1.55, $p < 0.01$; P9: 4.94 ± 2.12 vs. 4.22 ± 1.75, $p < 0.01$), indicating a more stable aortic wall in patients with a thin aortic wall.

Table 3 : Transesophageal dimensions and TSC results

n=496	Group 1 (n=136)	Group 2 (n=360)	*P*-value*
Aortic annulus (mm)	24.5±2.2	24.5±2.4	0.74
Aortic Sinuses (mm)	33.5±6.3	34.4±7.7	0.62
Sino-tubular junction (mm)	30.8±7.2	31.5±8.2	0.70
Ascending aorta (mm)	35.1±9.0	37.0±10.2	0.09
P7	165.3±103.3	132.7±84.9	<0.01
P8	4.52±2.17	2.53±1.14	<0.01
P9	5.74±2.37	3.62±1.35	<0.01

A significant correlation (CC) was found between aortic wall thickness and the parameters P7 (CC 0.13; $p = 0.04$), P8 (CC 0.29; $p < 0.01$) and P9 (CC 0.27; $p < 0.01$) in cohesion testing, presence of acute dissection (CC 0.17; $p < 0.01$) and positive histological changes in aortic media (CC 0.55; $p < 0.01$). Diameter of ascending aorta did not correlate with AWT (CC 0.06 $p = 0.20$).

Of 18 patients presenting with acute type A aortic dissection, 13 (72.2%) had an aortic diameter of less than 45 mm and the majority of these patients presented with an AWT > 2,28 mm (15/18; 83.3%).

III. Discussion

Acute aortic dissection is a serious disease with significant associated morbidity and mortality, which often occurs spontaneously in individuals with no significant comorbidities, but is also observed as a rare complication of cardiac surgery as a result of aortic wall injury from cannulation, cross-clamping, aortic incisions

or central anastomoses of bypass grafts [3]. The mechanisms leading to high susceptibility for further injury and development of localized subadventitial hematoma or widespread acute dissection in some patients are only poorly understood. Luk et al [8] described histological changes including intimal thickening, cystic media necrosis and disruption of the media in excised aortic samples of patients undergoing surgery for AD as a post-operative complication after primary cardiac surgery. The majority of changes were located near the cannulation sites, aortic incisions or cross-clamping sites and near to stitch holes or knots. In addition, non-dissected samples of aortic wall still showed changes in vessel architecture. Williams et colleagues [3] published data from the Society of thoracic Surgeons (STS) database analyzing prevalence and risk factors for intraoperative AD in more than 2 million patients undergoing elective surgery. 1294 patients suffered from intraoperative AD (0.06%). Patients with intraoperative dissection were more likely to be older, female and have a history of previous cardiac surgery, compared to patients without intraoperative dissection.

Another retrospective single-center study including mainly patients undergoing CABG reported an incidence of 0.12% of intraoperative or early postoperative (8-32 days) AD [9]. In all cases of intraoperative AD, the primary tear was located at the cannulation site. Histological examination of the aortic wall revealed cystic media necrosis in four cases, atherosclerosis in three cases, but no pathological changes in two cases. Aortic diameter did not predict the development of AD.

Current guidelines suggest intervention in the general population when the thoracic aorta exceeds 5.5 cm in diameter, as the annual rupture risk outbalances the perioperative mortality. However, several large studies of patients with small aortic aneurysms have revealed heterogeneity in patterns of growth and rupture potential among patients with a moderate dilatation of the aorta. Indeed, the majority of patients with acute type A aortic dissection present with aortic diameters <5.5 cm and thus do not fall within current guidelines for elective ascending aortic replacement [3,10].

Besides aortic diameter, many other properties of the aorta and their potential roles in the pathogenesis of aortic dissection have been discussed. Beller et al [11] demonstrated that the most distinct motion of the ascending aorta can be observed approximately 2 cm above the STJ, which is the most frequent location of intimal tear formation in the process of AD, indicating a potential role for aortic dissection.

As previously mentioned the histological changes observed in patients with acute aortic dissection are heterogeneous and vary from minimal changes, to fragmentation of elastin or fibrosis to complete media necrosis. However, these changes are not specific as they have also been frequently observed in healthy patients [12].

Hypertension is widely believed to be a major triggering factor for the development of AD [13,14,15,16]. Sommer [17] showed that distension of the aorta during systole induces radial movement of the wall layers against each other relative to the distance from the aortic center, as reflected by the diameter of the aorta. Based on this mechanistic approach, increased systolic pressure produces greater expansion of the aortic wall and movement of aortic layers, and might be more harmful than diastolic pressure in conferring dissection risk. Movement of the radial layer caused by systolic pressure might cause a rupture between tunica adventitia and media making the aortic wall susceptible to further injury, leading to dissection. This mechanism could explain the higher dissection risk in those patients with a large aorta or systolic hypertension than patients with a normal aorta or diastolic hypertension.

Bicuspid aortic valve (BAV), the most common congenital heart defect, has long been implicated in the development of severe aortic complications. However, in our previous study comparing the cohesion of the aortic wall in patients presenting with bicuspid and tricuspid aortic valves, we did not detect any difference between these two groups [18]. One explanation for this discrepancy might be the higher prevalence of hypertension in the tricuspid group in this study, possibly modifying aortic wall cohesion of this patient cohort more distinctly.

Another potential factor in the pathogenesis of AD may be the impairment of vasa vasorum flow, as postulated by Angouras et al [19]. Impairment of blood supply to the thoracic aorta in an experimental setting leads to abnormal morphology of collagen and elastin resulting in increased stiffness of the aortic wall. When ischemia of the aortic wall results, even mild traction might cause separation of the aortic layers, with resultant aortic dissection.

Currently, there are only limited data available on the impact of aortic wall thickness on the development of aortic dissection. Fanari et al [20] demonstrated that combined intimal/medial thickness as well as total aortic wall thickness was greater in patients with AD compared to controls. For this reason, the current study focuses on aortic wall thickness, a parameter which can be easily measured preoperatively in routine practice using TOE or CT. We were able to show that aortic wall thickness > 2mm predicts histological pathology, and poorer aortic wall cohesion as measured by Dissectometer. This finding is underlined by the clinical fact that incidence of acute dissection was significantly higher in patients with a thicker aortic wall. At first glance, this seems to be

paradox as aortic enlargement with consecutive wall thinning are believed to be the most important factors increasing wall stress and leading to aortic rupture or dissection.

IV. Conclusion

The current study could showed that patients with AWT of more than 2,28 mm may be at higher risk of aortic wall instability, as measured by Dissectometer examination and histology compared to patients with a thinner aortic wall. However, a larger prospective study with a long-term follow-up is necessary to confirm our findings.

Limitations

There are some limitations of our study. Firstly, our study suffers from the general limitations of a single-center, retrospective investigation. A larger prospective study with a long-term follow-up is necessary to confirm our findings. Although histology is considered to be the standard technique for analyzing aortic wall stability, the predictive value of this method is unknown.

V. Acknowledgments

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Disclosures

The authors had full access to the data and take full responsibility for its integrity. All authors have read and agreed to the manuscript as written. The authors disclose no conflict of interests in regard to the present manuscript.

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