

Predicting the Risk for Acute Type B Aortic Dissection in Hypertensive Patients Using Anatomic Variables

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OBJECTIVES This study sought to identify possible anatomic predictors of acute type B aortic dissection (AAD) in hypertensive patients using multidetector computed tomography angiography (CTA).

BACKGROUND Although hypertension remains one of the most significant risk factors for AAD development, it is unlikely to be the only risk factor for AAD. Few studies have assessed anatomical predictors of AAD development.

METHODS CTA of normotensive patients without AAD (group 1, n = 35), hypertensive patients without AAD (group 2, n = 37), and hypertensive patients with AAD (group 3, n = 37) were compared. The length, diameter, volume, and tortuosity of the aorta as well as arch vessel angulation were measured for each patient and normalized to group 1 averages. Stepwise logistic regression identified significant anatomical associations; the model was validated based on 1,000 bootstrapped samples.

RESULTS The demographics of the groups were similar. The length of the proximal and entire aorta, the diameters in the proximal ascending aorta and aortic arch, and the aortic volumes were all greater ($p < 0.0001$, $p = 0.0064$ for ascending aortic diameter) in group 3 than in groups 1 and 2, as was entire aortic tortuosity ($p < 0.0001$). An AAD risk model was developed based on aortic arch diameter, length from the aortic root to the iliac bifurcation, and angulation of the brachiocephalic artery origin from the aorta. The bootstrap estimate of the area under the receiver operating curve was 0.974.

CONCLUSIONS Enlargement of the ascending aorta and aortic arch and increased aortic tortuosity reflect an aortopathy which enhances the probability of AAD. A model based on 3 anatomical variables demonstrates significant associations with AAD: it may allow identification by aortic imaging of the hypertensive patient most at risk, and permit implementation of aggressive medical management and consideration of pre-emptive surgery to prevent dissection. (J Am Coll Cardiol Img 2013;6:349–57)

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Acute type B aortic dissection (AAD) is a highly feared thoracic aortic pathology encountered in hospital settings. Although many type B AADs can be managed medically, most patients with this pathology suffer significant morbidity and mortality (1). Even after optimal medical and surgical therapy, the long-term prognosis of type B AAD is dismal, with 5- and 10-year survival rates of 60% and 35%, respectively (2,3).

Hypertension remains one of the most significant risk factors for type B AAD development, with nearly 75% of patients having a history of hypertension (4). Nevertheless, although nearly 75 million adults in the United States are hypertensive, the incidence of AAD is only 2.9 to 3.5 per 100,000 person-years (5,6). Given the high mortality rate and dismal long-term prognosis of type B AAD, it seems logical to try to identify additional characteristics that might predispose an individual with hypertension to development of type B AAD. This study is an attempt to provide an anatomic aortic profile to identify those hypertensive patients most at risk, who may need more aggressive medical management or even pre-emptive surgery to prevent dissection.

ABBREVIATIONS AND ACRONYMS

AAD = acute aortic dissection

CTA = multidetector computed tomography angiography

ROC = receiver-operating curve

METHODS

Study design. A review of our institutional database disclosed 56 patients with AAD who presented to Mount Sinai Medical Center (MSMC) from February 2002 to July 2010. A multidetector computed tomography angiography (CTA) within 2 weeks of presentation and symptom onset was required to diagnose AAD: patients with type B AAD were excluded from the study if CTA was not carried out as part of the diagnostic examination, if the study was done at an outside institution, if the dissection was secondary to a traumatic event, or evidence of congenital or connective tissue disease was found, such as bicuspid aortic valve or Marfan's syndrome. These criteria resulted in inclusion of 37 type B AAD patients. Data from these patients were compared with those for 37 hypertensive patients without AAD and those for 35 normotensive patients without AAD who underwent CTA.

The institutional review board approved this retrospective research and waived the need for individual patient consent. Patient information was obtained from medical records of patients presenting to the MSMC Emergency Department (ED)

with AAD or who were transferred to our institution after confirmation of diagnosis. Normotensive and hypertensive adult patients who presented to the ED with episodes of chest, abdominal, or back pain described as sharp, pressure-like, or aching and who underwent CTA to determine the cause of the presenting symptoms were included in the study. ED medical records of these patients were then assessed for patients' presenting blood pressure and antihypertensive medication history.

Computed tomography. Electrocardiographic (ECG)-gated CTA of the thoracic aorta was carried out with injection of 100 ml of contrast with 50 ml of saline chaser, threshold 80 HU; rotation speed: 330 ms; collimation: 64×0.6 mm; pitch: 0.2; voltage: 140 kV; current: 700 to 850 mAs. Datasets were analyzed on a cardiovascular workstation (AquariusWS version 3.7.0.13, TeraRecon, San Mateo, California) using dedicated vascular analysis software. The software enables real-time diagnostic review of 2-, 3-, and 4-dimensional images for managing large thin-slice computed tomography and magnetic resonance scans, and includes workflow tools which simplify the interpretation by automatically presenting the 3-dimensional volume based on the study type selected by the operator.

The length of the ascending aorta from the aortic root to the left subclavian artery, and the entire aorta from the aortic root to the iliac bifurcation were measured (Fig. 1). Additional measurements included the volumes of the ascending aorta and the aortic arch, as well as the maximal diameters of the aortic root, the ascending aorta at the right pulmonary artery, and the aortic arch. Tortuosity of the aorta, defined as the length of the midline within the aorta divided by the linear distance between the aortic root and the iliac bifurcation, was also calculated. The tortuosity of the ascending aorta, defined as the measured length of the ascending aorta divided by the linear distance between the aortic root and left subclavian artery, was similarly calculated for each patient (Figs. 2A and 2B). The angle of the origin of the brachiocephalic and of the left subclavian arteries from the aortic arch was measured. The volume and diameter measurements of the descending and abdominal aorta were likely confounded by the presence of type B AAD, and were not compared between the groups. Analysis was therefore restricted to the ascending aorta and aortic arch proximal to the left subclavian artery.

Statistical analysis. For group comparisons, we used chi-square tests or Fisher exact tests for categorical variables, analysis of variance (ANOVA) allowing

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