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REVIEW

STATE-OF-THE-ART PAPER

Thoracic Aortic Calcification

Diagnostic, Prognostic, and Management Considerations

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CME/MOC Objective for This Article: Upon completion, the reader should be able to: 1) recognize how distinct patterns of thoracic aortic calcification may be related to the underlying pathobiology of specific diseases; 2) differentiate which imaging findings related to thoracic aortic calcification provide independent prognostic value, clarify a diagnosis, and/or result in changes in downstream management; 3) define strengths and limitations of an echocardiographic assessment of the thoracic aorta in patients who have had an embolic event; and 4) describe variations in the assessment and analysis of thoracic aortic calcification on computed tomography that may explain discordant results.

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ABSTRACT

Thoracic aortic calcification (TAC) is associated with adverse cardiovascular outcomes, and for the cardiovascular imager, is predominantly encountered in 4 settings: 1) incidentally, for example, during a coronary artery calcium scan; 2) as part of dedicated screening; 3) in the evaluation of an embolic event; or 4) in procedural planning. This review focuses on TAC in these contexts. Within atherosclerosis, TAC is common, variable in extent, and begins in the intima with a patchy distribution. In metabolic disorders, aortitis, and radiation-associated cardiovascular disease, calcification preferentially involves the media and is often more concentric. As an incidental finding, atherosclerotic TAC provides limited incremental discriminative value, and current data do not support screening. After an embolic event, the demonstration of thoracic atheroma provides diagnostic clarity, but has limited treatment implications. Before any procedure, the plan often changes if the most severe form of TAC, a porcelain aorta, is discovered. (J Am Coll Cardiol Img 2018;11:1012–26)

A common imaging finding, thoracic aortic calcification (TAC) reflects systemic atherosclerosis and its attendant cardiovascular morbidity and mortality risks. Typically, TAC is encountered in 4 contexts: 1) incidentally, for example, as part of a coronary artery calcium (CAC) scan or any chest computed tomography (CT) study; 2) as part of a dedicated screening assessment in an asymptomatic patient; 3) in the evaluation of a patient with an embolic event; or 4) as a preprocedural assessment in a patient with severe coronary or valvular heart disease. Because these assessments are often performed with CT or echocardiography, this review primarily discusses these modalities. Thoracic aortic pathology is also frequently assessed with magnetic resonance imaging (MRI), but due to signal void, calcification is not imaged. In addition, although regression of thoracic atheroma can be measured with MRI, the technique remains a research application, and is discussed elsewhere (1–3). Historically, fluoroscopy and chest x-rays have also been used for diagnosis and prognosis, but because of limited accuracy and lack of treatment implications, neither is recommended for evaluating TAC (4–8).

In contemporary practice, the value of any imaging test is framed within a hierarchical context, beginning with technical considerations and diagnostic accuracy and culminating with changes in therapy and improved outcomes (9,10). With TAC, considerations vary based on the indication for the test and include incremental prognostic value, alteration in risk with management changes, diagnostic clarity with downstream treatment modifications, and alterations to a procedural plan. In this review, we address the settings in which the cardiovascular imager will encounter TAC and focus on the significance of TAC within these specific contexts (**Central Illustration**).

PATHOBIOLOGY OF AORTIC CALCIFICATION

A detailed discussion of the regulatory mechanisms of vascular calcification is beyond the scope of this review, but several important concepts warrant emphasis (11,12). First, aortic calcification likely functions as both a consequence and a cause of cardiovascular disease. Atherosclerosis leads not only to calcification through cellular osteogenic differentiation, but can also stiffen the aorta, and depending on the relationship to neighboring lipid pools, may increase local wall stress and contribute to plaque ulceration (13). Second, within atherosclerotic lesions, calcium is common but highly variable (14). Histologically, atherosclerosis is graded as mild, moderate, or severe based on the degree of fibrosis and medial loss (15). Any grade may have superimposed calcified plaque or thrombus (15) (**Figure 1**). Third, calcification occurs in 2 distinct locations within the vessel wall, the intima and the media. Intimal, or neointimal, calcification has a patchy distribution within atherosclerotic lesions and is most commonly amorphous without distinct architecture. As atherosclerotic

ABBREVIATIONS AND ACRONYMS

AS = aortic stenosis

CABG = coronary artery bypass grafting

CAC = coronary artery calcium

CAD = coronary artery disease

CI = confidence interval

CT = computed tomography

ECC = extra-coronary calcium

MI = myocardial infarction

MRI = magnetic resonance imaging

OR = odds ratio

RACD = radiation-associated cardiac disease

TAC = thoracic aortic calcification

TEE = transesophageal echocardiography

TTE = transthoracic echocardiography

calcification progresses, it can also involve the media. Conversely, calcification associated with chronic kidney disease, diabetes mellitus, systemic inflammatory disease, and radiation-associated cardiac disease (RACD) often begins in medial smooth muscle, is concentric, and has a diffuse distribution (16–19).

Currently, noninvasive imaging of the thoracic aorta cannot distinguish the intima from the media, but the pattern of calcification may alert the clinician to an underlying diagnosis (20, 21). Although there is overlap in the pathology of neointimal and medial calcification (21,22), patchy calcification may indicate more typical atherosclerosis. Alternatively, medial calcification may appear more circumferential, with a differential diagnosis that includes arteritis and RACD (**Figure 2**).

DIAGNOSTIC AND PROGNOSTIC IMPLICATIONS OF THORACIC AORTIC ATHEROMA ON ECHOCARDIOGRAPHY

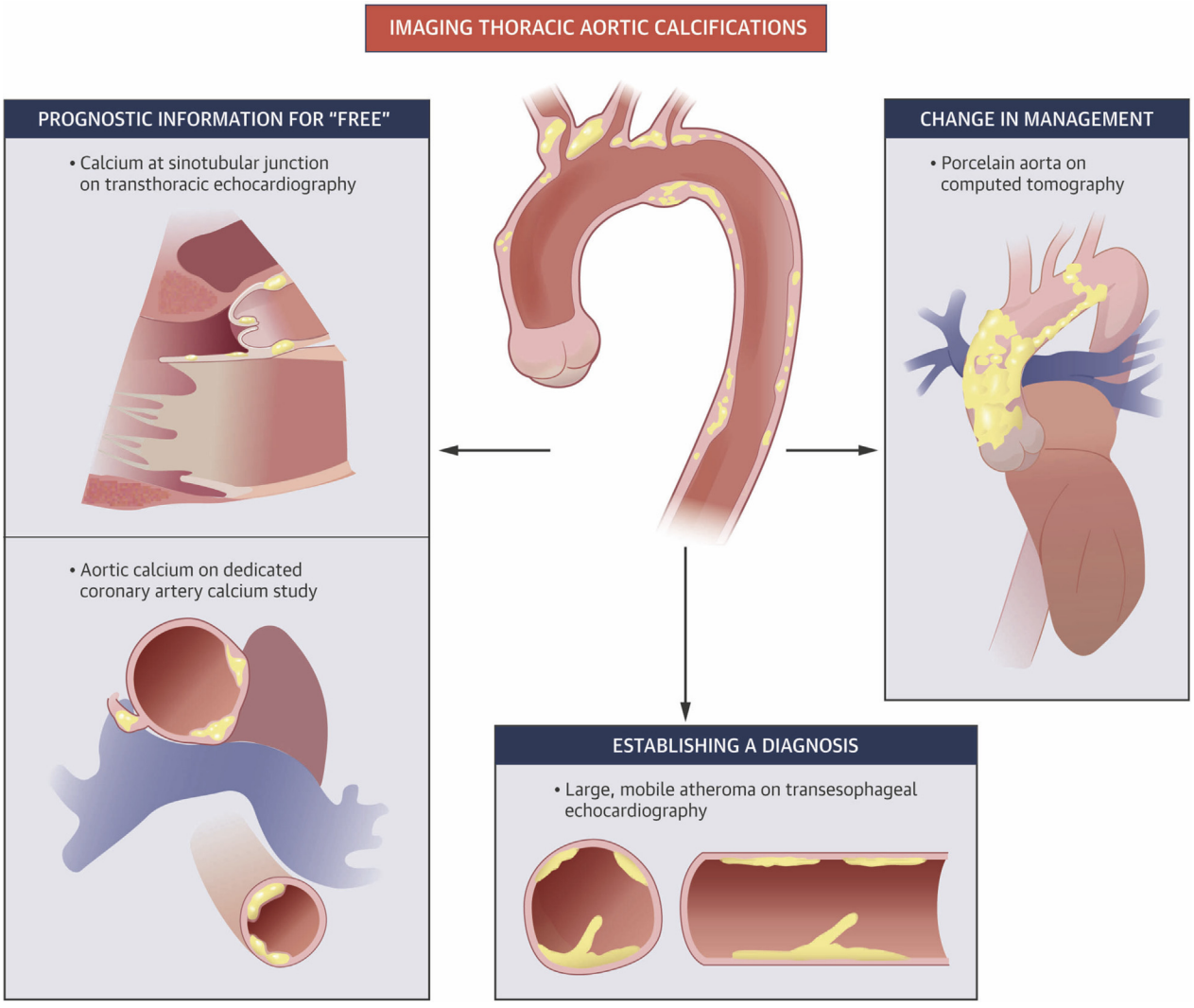
For decades, the location, characteristics, and associated risks of thoracic aortic plaques have been described with echocardiography (23). Compared with CT, the advantages of echocardiography include the lack of ionizing radiation and improved temporal resolution, which facilitates assessment of plaque mobility. In addition, unlike noncontrast CT, echocardiography more readily visualizes noncalcified and calcified atheroma. However, CT often has improved spatial resolution, and severe calcification can also confound the echocardiographic assessment due to reverberation artifact and acoustic shadowing (24). Moreover, in distinction to the Agatston method for CT (8), ultrasound physics are not readily amenable to quantification of calcification. Standard echocardiography also does not image the entire thoracic aorta. Despite these limitations, echocardiographic assessment for thoracic aortic atheroma has been used to aid in the diagnosis of stroke, inform the probability of concomitant coronary artery disease (CAD), and risk stratify for cardiovascular events (25–27).

TRANSESOPHAGEAL ECHOCARDIOGRAPHY, AORTIC ATHEROMA, AND STROKE. Transesophageal echocardiography (TEE) has an established role in the investigation for an embolic source of stroke due to 3 important observations (28). First, in an autopsy study of 500 patients, ulcerated plaques were more common in the aortic arch in patients without an alternative cause for cerebral infarction (29). Presumably, thrombi form on these plaques and embolize. Second, observational studies have shown that 20% to 30% of patients with embolic strokes have aortic arch atheroma on TEE (25,30). Finally, case–control studies have demonstrated an association between thoracic aortic atheroma and stroke. In a study of 122 patients, protruding atheromas (≥5 mm) were associated with stroke after adjustment for traditional risk factors, and only cases had atheromas with mobile components (**Figure 3**, Online Videos 1 and 2) (31). Another study of 250 patients also showed that, after adjustment for clinical risk, atheroma (≥4 mm) was associated with stroke (25).

Despite these observations to support association, the evidence to support causation between aortic atheroma and stroke is less compelling. In a prospective cohort study, aortic atheroma was not associated with cerebrovascular events after multivariable adjustment, although only 41 events occurred after a median follow-up of 5 years (32). Moreover, the implications for management are unclear. An observational study of 129 patients with thoracic aortic atheroma demonstrated an increased unadjusted risk for future embolic events if patients were not treated with anticoagulation (33). However, larger observational studies with adjustment for confounding and randomized controlled trials are lacking. Therefore, in comprehensive TEE, to evaluate for an embolic source of stroke, the severity and mobility of thoracic aortic atheroma provides diagnostic insights, but may not alter standard secondary preventative therapies.

TAC ON ECHOCARDIOGRAPHY AND CONCOMITANT CAD. Although assessment of aortic calcification is not a primary indication for transthoracic echocardiography (TTE), adjunctive information may be clinically relevant and is acquired noninvasively without ionizing radiation. This rationale has prompted investigations focused on concomitant CAD. In a study of 338 patients 65 years old or younger with chest pain, aortic root calcium was weakly associated with an abnormal myocardial perfusion study (54.9% vs. 41.5%; $p < 0.05$) (26). However, when calcifications involving the aortic valve leaflets and mitral annulus were also considered, the association strengthened (odds ratio [OR]: 2.12; 95% confidence interval [CI]: 1.18 to 3.78 for calcium at ≥2 sites). Even after adjustment for clinical risk, multiple calcium deposits remained associated with an abnormal myocardial perfusion study (OR: 2.08; 95% CI 1.27 to 3.41) (26).

CENTRAL ILLUSTRATION **The Value of Imaging Thoracic Aortic Calcifications**

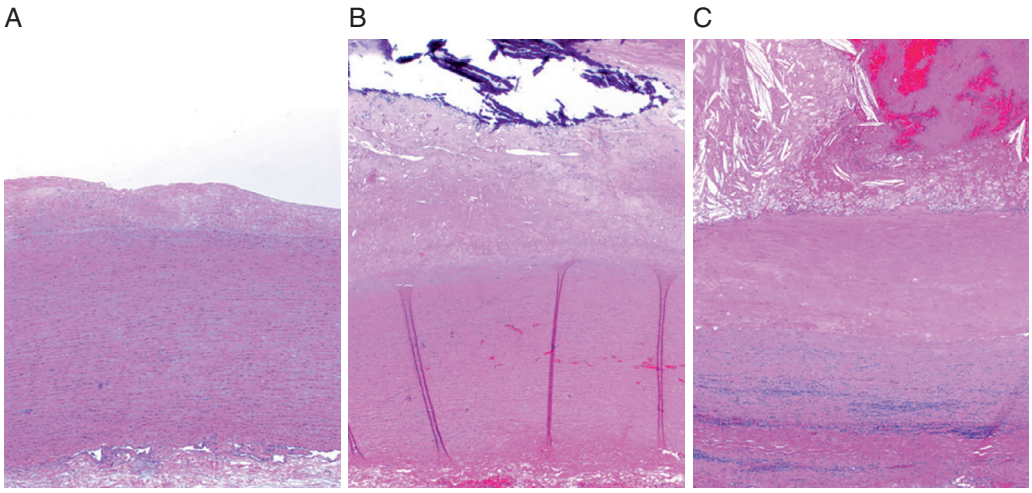


Desai, M.Y. et al. J Am Coll Cardiol Img. 2018;11(7):1012-26.

The imaging evaluation of thoracic aortic calcifications can provide insights regarding prognosis, diagnosis, and management. Specifically, when an echocardiogram or coronary artery calcium study is performed, the presence of thoracic aortic calcification combined with other noncoronary sites of calcification has ancillary prognostic information. In a patient with an unknown cause for stroke, the extent and mobility of thoracic aortic atheroma may establish a source for embolus. For a patient considering a cardiovascular intervention, the presence of a porcelain aorta may alter the procedural approach.

Subsequent studies have used similar echocardiographic calcium scores, incorporating calcium at the aortic root, at the aortic and mitral valve leaflets, and at the mitral annulus (**Figure 4**, Online Videos 3, 4, and 5). Unfortunately, variations in scoring methodologies and different assessments for CAD limits comparison. However, in general, calcium deposits at multiple sites have been associated with coronary atherosclerosis defined on invasive angiography, as a CAC score >400, or >50% narrowing on coronary CT angiography (34–36). Likely, these associations reflect the systemic nature of atherosclerosis and may alert the clinician to the possible presence of CAD. Still, previous studies have made these observations in patients in whom assessments for CAD were already planned, and current data do not support an

FIGURE 1 Grading of Atherosclerosis

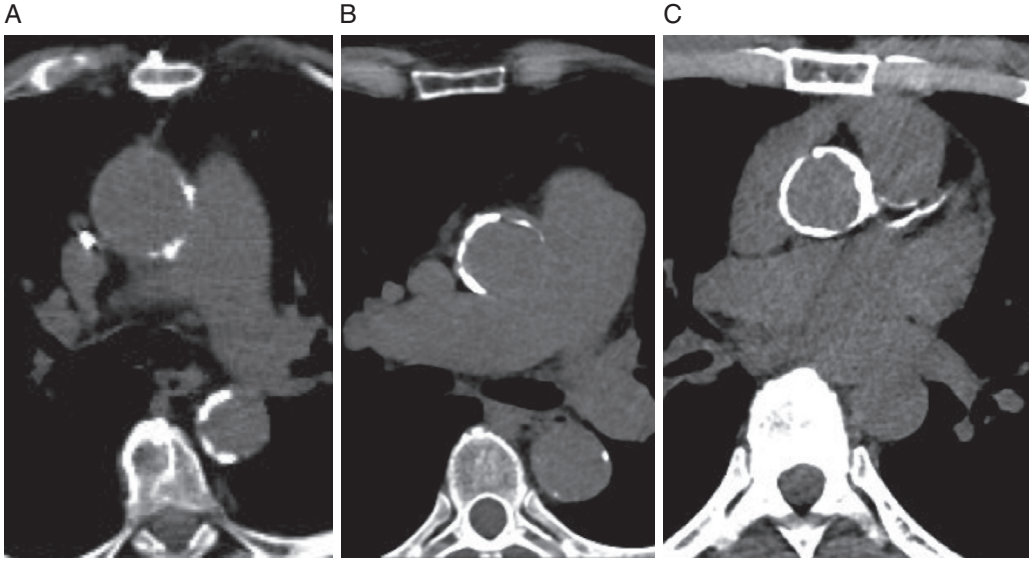


(A) Mild atherosclerosis is characterized by extracellular lipid deposits without fibrosis. **(B)** Moderate atherosclerosis has fibrosis and less than one-third loss of the media thickness, and **(C)** severe atherosclerosis has greater than one-third loss of media thickness. Any grade of atherosclerosis can have **(B)** superimposed calcification or **(C)** thrombus. Adapted with permission from Stone et al. (15).

investigation for anatomic CAD based solely on multiple sites of echocardiographic calcification.

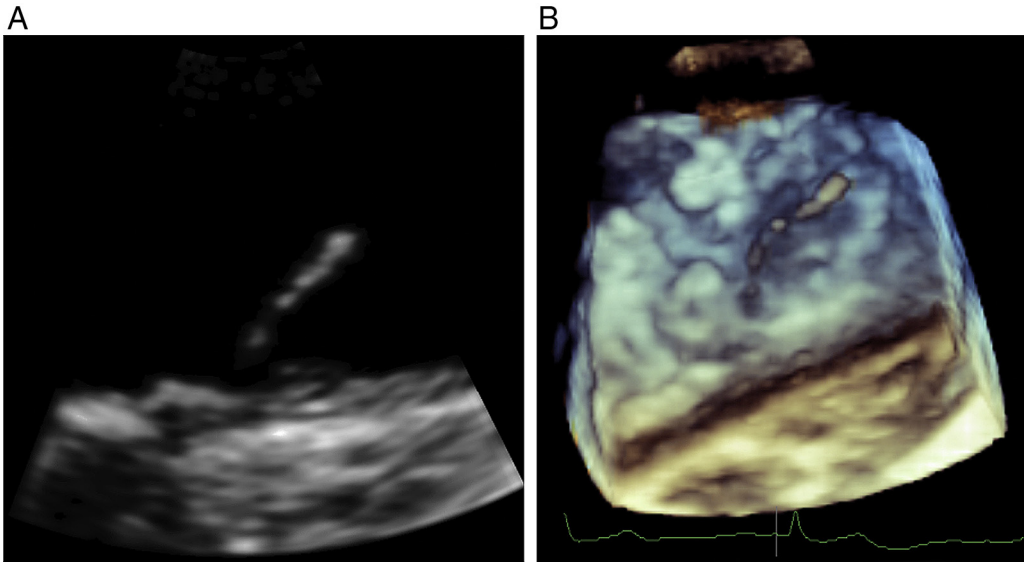
ECHOCARDIOGRAPHIC CALCIFICATION AND PROGNOSIS. Likewise, studies have investigated the relationship between echocardiographic calcium scores and outcomes, including all-cause death, myocardial infarction, and stroke. Among 443 patients without known cardiac disease, after adjustment

FIGURE 2 Patterns of TAC



In these electrocardiographically-gated, noncontrast-enhanced computed tomography (CT) scans, there is **(A)** patchy calcification typical of atherosclerosis, **(B)** more confluent calcification in a patient with a remote history of aortitis, and **(C)** circumferential calcification in a patient with radiation-associated cardiovascular disease. TAC = thoracic aortic calcification.

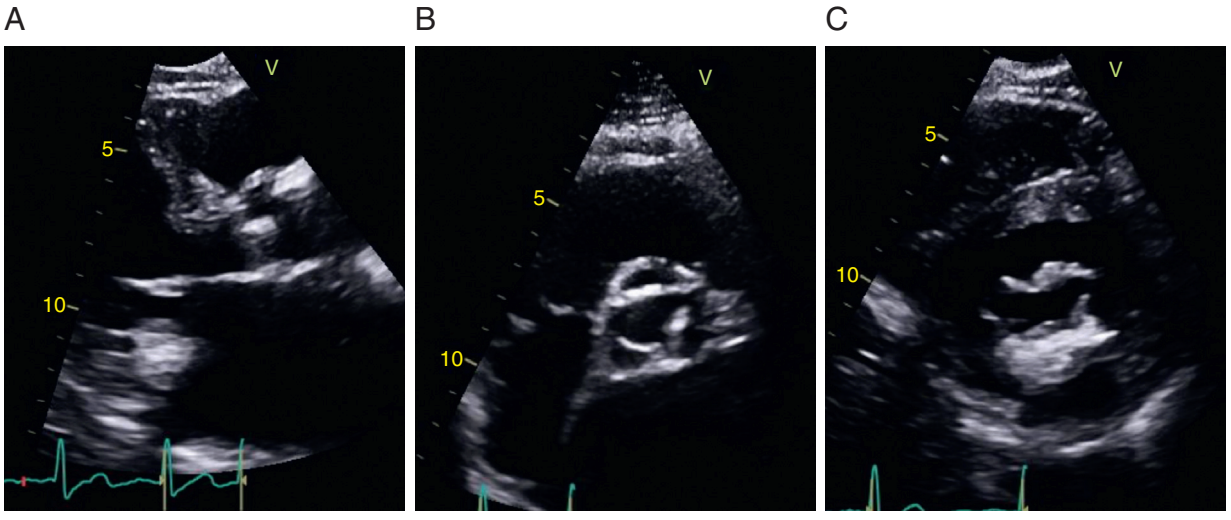
FIGURE 3 Mobile Aortic Arch Atheroma



In this patient with a history of an embolic stroke, **(A)** a long (1.0 cm) mobile aortic arch atheroma is visualized, and **(B)** the extent of atheroma is appreciated on 3-dimensional reconstruction. Severe plaque thickness (≥4 mm) and mobile atheroma are associated with stroke. See Online Videos 1 and 2.

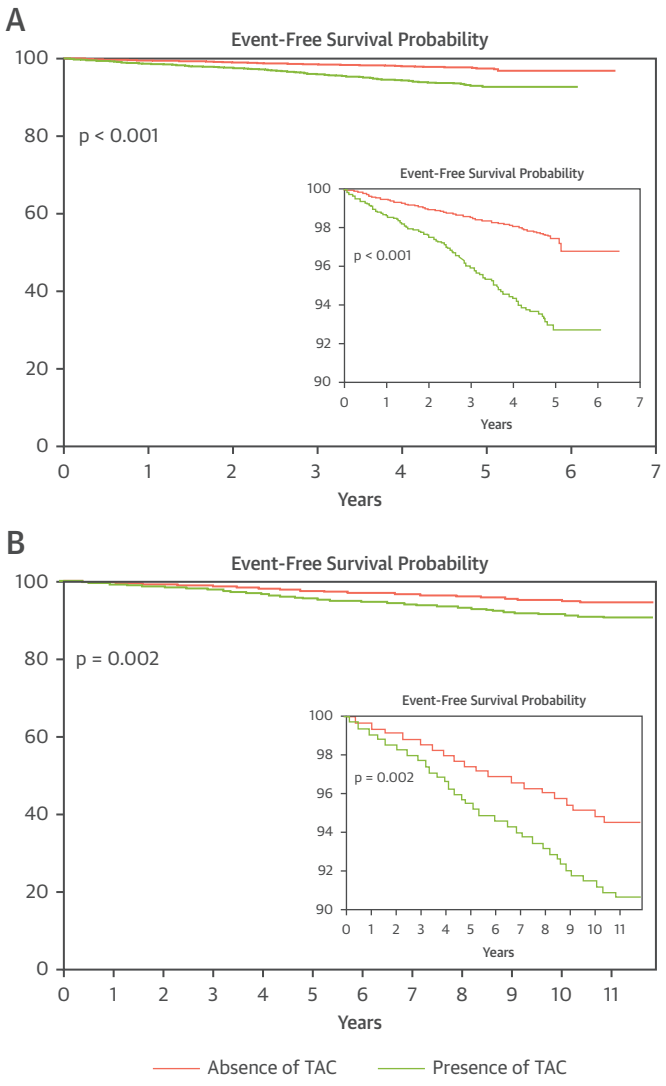
for clinical risk, the echocardiographic calcium score was associated with total mortality and stroke (37). In another study of 1,303 patients with stress echocardiography and no known CAD, an echocardiographic calcium score was also associated with all-cause death and myocardial infarction (27). However, after assessing for stress-induced wall motion abnormalities and

FIGURE 4 Echocardiographic Calcium Assessment



In this patient with shortness of breath, **(A)** calcification is noted at the sinotubular junction, **(B)** the aortic valve leaflets are also thickened and calcified, and **(C)** there is prominent mitral annular calcification **(A)**. As a secondary assessment, the extent of echocardiographic calcium has prognostic value, although it is limited. See Online Videos 3, 4, and 5.

FIGURE 5 TAC on CT and Adverse Events in MESA and HNR



(A) In a study of 6,807 subjects from MESA (Multi-Ethnic Study of Atherosclerosis) with 232 events, unadjusted Kaplan-Meier curves show a significant association between survival and the presence of TAC, although on multivariable adjustment for clinical risk factors and coronary artery calcium (CAC), TAC was associated with coronary heart disease* events only in women (Budoff et al. [40]). **(B)** In a study of 3,630 subjects from HNR (Heinz Nixdorf Recall Study) with 241 events, the presence of TAC was associated with the composite outcome of myocardial infarction, stroke, and cardiac death, but there was no association after adjustment for clinical risk and CAC (Mahabadi et al. [44]). *CHD events = definite or probable myocardial infarction, resuscitated cardiac arrest, cardiac death, definite angina, and probable angina with revascularization. Abbreviations as in Figure 2.

clinical risk, echocardiographic calcium did not improve discrimination.

In summary, a comment on aortic calcification, when combined with an assessment for calcium elsewhere, likely has prognostic value because it reflects the extent of atherosclerosis. If wall motion abnormalities are considered, however, this prognostic value is attenuated, likely in part due to the identification of patients with CAD. Therefore, echocardiographic calcium may aid in global risk stratification, but discrimination is not robustly improved, and downstream changes in patient management are not defined. On a resting echocardiogram, calcification for prognosis is consequently best relegated as a secondary assessment in a study performed for another indication.

PROGNOSTIC IMPLICATIONS OF TAC ON CT

A wealth of data have emerged regarding TAC and risk stratification, principally from additional analyses of primary prevention cohorts that focused on CAC (Figure 5). As is typical for investigations that study diverse populations with different methodologies and slight variations in outcomes, results have been mixed. With studies of TAC, notable differences apply to 3 domains.

The first relates to patient characteristics, such as background risk and symptoms. The second applies to outcomes, which vary from narrowly defined coronary events, to more inclusive cardiovascular events, to the least biased outcome, all-cause mortality. Third, and of particular interest, TAC has been disparately defined. For example, dedicated CTs for CAC do not include the aortic arch and proximal descending aorta, which are common sources of calcification. Moreover, TAC can be expressed as binary, or as a continuous variable with an Agatston score, extrapolating a methodology developed for CAC. In addition, most studies have evaluated noncontrast CTs, which will miss or underestimate the extent of noncalcified atherosclerosis. Finally, the relevance of TAC can be investigated independently or can be incorporated as part of an extra-coronary calcification (ECC) score, which is similar to previous discussions regarding echocardiographic calcifications. All of these distinctions warrant emphasis in determining whether the clinician should ascribe any prognostic value to TAC, and if so, whether its value is simply as an incidental finding or instead warrants a dedicated evaluation for risk stratification (Table 1).

DIFFERENCES IN COHORTS. In the MESA (Multi-Ethnic Study of Atherosclerosis) trial, in an initial population of 6,814 participants from 4 ethnic groups, aortic wall calcification was present in 28.0%, and approximately one-half had CAC (38,39). Traditional cardiovascular risk factors were associated with aortic calcification, although hypertension and current

TABLE 1 Differences in Design and Analysis Among Studies Assessing the Value of TAC for Risk Stratification

First Author (Ref. #)	Cohort	Outcomes	Events	Include Aortic Arch and Proximal Descending Aorta	Assessment of TAC	TAC as Part of an ECC Score	Contrast	Prognostic Value After Adjustment for Clinical Risk Factors and CAC	Improved Discrimination by C-Statistic or NRI
Budoff et al. (40)	6,807 subjects from MESA	CHD*	232	No	Agatston	No	No	Only in women	Not reported
Yeboah et al. (47)	5,745 nondiabetics from MESA	CHD, stroke, other cardiac death	346	No	Agatston	No	No	For CHD and death after clinical risk (not adjusted for CAC)	No
Tison et al. (54)	5,903 nondiabetics from MESA	CHD, other cardiac death, all-cause mortality	348 CHD events, 572 deaths	No	Binary	Yes	No	Yes	Yes, for all-cause mortality with ECC score
Hoffmann et al. (41)	3,486 subjects from FHS	MI, ischemic stroke, cardiac death, all-cause mortality	255	No	Agatston	No	No	Only for mortality	No (although analyses not performed for all-cause mortality)
Mahabadi et al. (44)	3,630 subjects from HNR	MI, stroke, cardiovascular death	241	No	Agatston	No	No	No	No
Wong et al. (45)	2,303 subjects from EISNER	MI, stroke, late revascularization, cardiac death	47	No	Agatston	No	No	No	No
Bos et al. (51)	2,408 subjects from Rotterdam Study	All-cause mortality	283	Yes	Agatston	No	No	Yes	Not reported
Santos et al. (48)	8,401 subjects from a single-center	All-cause mortality	124	No	Binary	No	No	Yes	Not reported
Kurra et al. (53)	862 patients before cardiac surgery	All-cause mortality	119	Yes	Plaque thickness and circumferential extent	No	Yes	Only after clinical risk (CAC not assessed)	Not reported

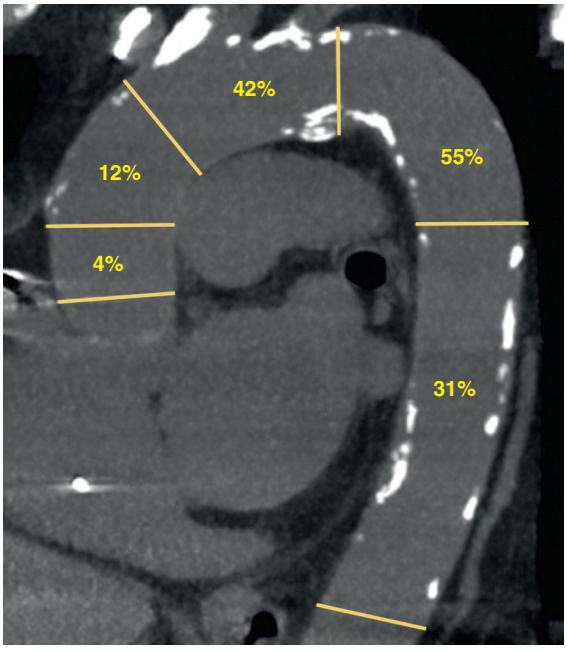
*CHD events = definite or probable MI, resuscitated cardiac arrest, cardiac death, definite angina, and probable angina with revascularization.

CAC = coronary artery calcium; CHD = coronary heart disease; ECC = extra-coronary calcium; EISNER = Early Identification of Subclinical Atherosclerosis Using Non-Invasive Imaging Research; FHS = Framingham Heart Study; HNR = Heinz Nixdorf Recall study; MESA = Multi-Ethnic Study of Atherosclerosis; NRI = net reclassification improvement; TAC = thoracic aortic calcification.

smoking had the strongest associations (38). At a mean follow-up of 4.5 years, only 1.9% had a myocardial infarction (MI), resuscitated cardiac arrest, or cardiac death (40). Similarly, in 3,217 participants from the imaging cohort of the FHS (Framingham Heart Study) trial, 42.5% had CAC, and 20.8% had TAC. During a mean follow-up of 8 years, the event rate was also low; 1.7% had a nonfatal MI or cardiac death (41). By contrast, despite a similar enrollment period as MESA, the Heinz Nixdorf Recall (HNR) study had a prevalence of TAC of 63.1% and a prevalence of CAC of 67.9% (42), which was likely related to higher baseline cardiovascular risk (43). Accordingly, after a follow-up of nearly 10 years, event rates were higher at 6.6% (44).

Because of these differences, discrepant results might be expected, but overall, conclusions regarding hard cardiac events (nonfatal MI, resuscitated cardiac arrest, cardiac death) are generally consistent. In MESA and FHS, after adjusting for clinical risk factors and CAC, TAC was not independently associated with cardiac events (40,41). Likewise, in a report from the EISNER (Early Identification of Subclinical Atherosclerosis by Noninvasive Imaging Research) study, TAC did not improve discrimination for cardiac events after adjustment for the Framingham risk score and CAC (45). The higher risk HNR study, which defined cardiovascular events as stroke, MI, and cardiac death, found a trend toward a higher event rate (hazard ratio: 1.33; 95% CI: 0.87 to 1.81) after multivariable adjustment (44). In a model that included measurements of left atrial size, epicardial adipose and TAC, a statistically detectable improvement in the C statistic was demonstrated compared with CAC and clinical risk factors. Although an important risk factor may have little influence on the C statistic in a well-developed model, this minimal change, coupled with the lack of an independent association with cardiovascular events, argued that TAC had little impact on prognosis in this cohort (46). Therefore, among diverse primary prevention cohorts, TAC has not reliably demonstrated prognostic value for hard

FIGURE 6 Distribution of TAC



In a single-center study of 970 patients referred to a cardiovascular prevention unit, percentages of patients with calcifications at different segments of the thoracic aorta are shown. Calcifications were most commonly visualized in the aortic arch and proximal descending aorta, which are segments not typically included in a CAC scan. Reproduced from data in Craiem et al. (50). Abbreviations as in **Figures 2 and 5**.

cardiac events, independent of clinical risk factors and CAC.

BEYOND RISK STRATIFICATION FOR CORONARY EVENTS. If the outcome is coronary-artery related events, the lack of substantial incremental prognostic value for TAC beyond CAC is not surprising. In low-risk primary prevention patients, calcified atherosclerosis in the coronary arteries should be more closely aligned with incident coronary events compared with calcified atherosclerosis elsewhere. However, because of the predilection of atherosclerosis for different vascular beds, an additional question is whether aortic calcification is associated with more broadly defined cardiovascular events and all-cause mortality.

In an analysis from MESA that included 5,745 patients without diabetes with 9 years of follow-up, 251 participants had cardiac events, 346 had cardiovascular events, and 321 died (47). Although TAC was associated with coronary events and all-cause death in a multivariable model, TAC did not improve discrimination, as assessed by C-statistics and net reclassification improvement, when added to the Framingham risk score and CAC. In results from the FHS, after adjusting for CAC, TAC was associated with all-cause mortality (32).

Other cohorts have similarly demonstrated a statistically detectable association between TAC and all-cause mortality. In a single-center study of 8,401 asymptomatic individuals followed for a median of 5 years with 124 deaths, TAC was associated with higher mortality after adjustment for clinical risk factors and CAC (48). Another single center study of 4,554 patients with 163 deaths also demonstrated an association between TAC and all-cause mortality in a multivariable model (49). Therefore, although TAC is not independently associated with coronary-related deaths for patients with CAC scanning, TAC may be associated with all-cause mortality.

EXTENT OF THORACIC AORTA VISUALIZED. In primary prevention, CT scans for calcification have included different fields of view, and the most important distinction is whether the aortic arch and proximal descending thoracic aorta have been included. In a single center study of 970 asymptomatic patients, the aortic arch and proximal descending thoracic aorta had 60% of all TACs (**Figure 6**) (50). In comparison to a typical scan range for a CAC study, which excludes the aortic arch and proximal descending thoracic aorta, the prevalence of TAC doubled with this extended measurement (50).

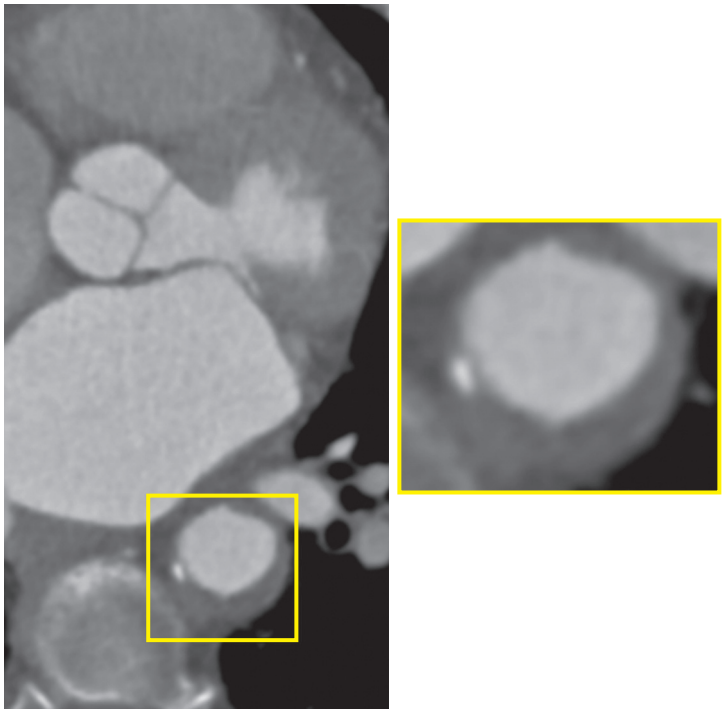
As has been highlighted in analyses from the FHS and MESA, TAC has not emerged as a robust independent risk factor when evaluated with traditional CAC scans (41,47). However, among 2,408 patients from the Rotterdam Study, aortic arch calcification was associated with cardiovascular mortality, independent of calcification in other vascular beds (51). Moreover, in 1,000 patients with cardiac scans that included the aortic arch, TAC was associated with a history of noncardiovascular events, although a notable limitation of this study was the lack of prospective adjudication of outcomes (52). Nonetheless, these data provoke a question of whether TAC would be more prognostically relevant in CAC scanning if the field of view were expanded.

MEASUREMENT OF TAC. In addition to variations in the extent of the ascending thoracic aorta included, different methods for measuring TAC have been reported. The simplest method is to consider TAC as binary. Alternatively, TAC can be expressed

continuously via the Agatston method, similar to a CAC score. Although the Agatston score was initially developed and validated for CAC with electron beam CT (39), application of this methodology to the thoracic aorta allows for quantification of the severity based on the density and area of calcification. However, established CAC strata (0, 1 to 9, 10 to 99, 100 to 399, and ≥400) are likely not applicable to TAC.

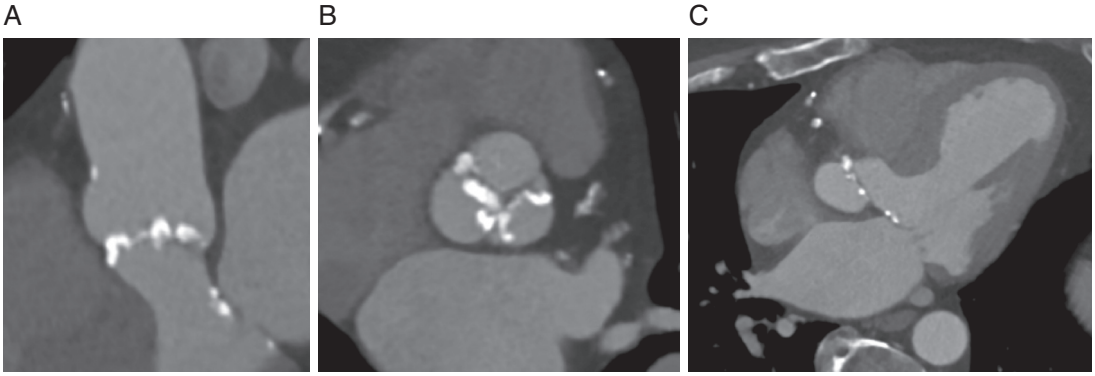
As discussed, when assessed as a categorical variable in patients with CAC scans, the prognostic value of TAC has been generally underwhelming (41). However, in an analysis from MESA with Agatston scoring, TAC was associated with cardiac events in women, but not in men, even after adjustment for CAC and clinical risk (40). Conversely, in a separate analysis from MESA restricted to patients without diabetes, but also using TAC as a continuous variable, TAC was associated with cardiac events in a multivariable model, although the model did not improve discrimination (47). In an analysis from the EISNER study, TAC was assessed continuously and with the same scoring categories as those commonly used for CAC. Although limited by few events, TAC was not associated with cardiovascular outcomes (45). In summary, the expression of TAC with an Agatston score may occasionally yield a statistically significant association in a multivariable model, but substantial improvement in discriminating cardiovascular events has not been demonstrated.

FIGURE 7 Calcified and Noncalcified Thoracic Aortic Atheroma



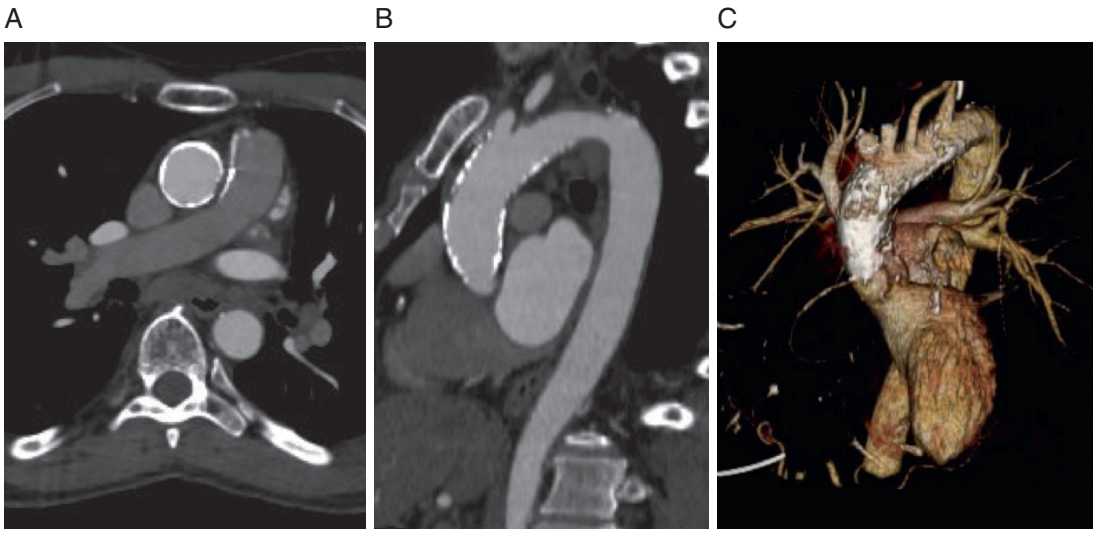
Calcification can have a patchy distribution within atheroma. The extent of calcified and noncalcified atheroma, as defined by thickness and circumference, is better appreciated with a contrast-enhanced CT. Abbreviation as in Figure 2.

FIGURE 8 Extracoronary Calcium on CT



In this electrocardiographically gated, contrast-enhanced CT scan, calcification is seen at the **(A)** sinotubular junction, **(A to C)** aortic valve leaflets, **(A and C)** mitral valve leaflets, and the **(B and C)** coronary arteries. An extracoronary calcium assessment may have limited discriminative value beyond clinical risk factors and CAC. Abbreviations as in Figures 2 and 5.

FIGURE 9 CT Appearance of a Porcelain Aorta



In this patient with a history of radiation-associated cardiovascular disease, there is **(A)** circumferential calcification involving the **(B)** entire ascending aorta, the extent of which is appreciated on the **(C)** 3-dimensional volume–rendered image. Safe aortic cross-clamping is not possible, and also note the **(A)** calcium involving the pulmonary artery.

CALCIFIED AND NONCALCIFIED ATHEROSCLEROSIS. In primary prevention, CT scanning for risk stratification is obtained without intravenous contrast, in part because of the risks of contrast, which include allergic reactions and nephropathy. Moreover, the increased attenuation from contrast confounds assessment of the CAC score. However, the lack of contrast also precludes visualization of noncalcified atheroma, which may be especially prominent in the thoracic aorta (**Figure 7**). In a single-center study of 862 patients with electrocardiographically gated, contrast-enhanced CT scans of the chest before cardiac surgery, the thickness and extent of aortic atheroma was measured in a semiquantitative fashion (53). Over a mean follow-up of 25 months, 119 patients died, and thoracic aortic atheroma was independently associated with all-cause mortality (53). Although contrast-enhanced studies in diverse cohorts are lacking, these results suggest that calcifications may not completely encompass cardiovascular risk related to thoracic aortic atherosclerosis.

TAC AS PART OF AN EXTRACORONARY CALCIUM ASSESSMENT. Because of the systemic nature of atherosclerosis, an additional line of inquiry has focused on whether TAC improves risk stratification as a component of an ECC assessment. Other sites with a predilection for calcification include the aortic valve leaflets, mitral valve leaflets and annulus, and the aortic root, especially the sinotubular junction (**Figure 8**). From the MESA study, investigators studied whether an ordinal ECC score based on calcification at these locations had prognostic value (54). Due to the lack of standardization of the Agatston method at noncoronary sites as well as over-representation of TAC because absolute calcium scores are higher at this location, the investigators chose a binary approach to these 4 sites. In 5,903 patients without diabetes, increasing ECC had a graded association with higher cardiac events and mortality. The ECC score also marginally improved the area under the receiver-operating curve, although the model with traditional risk factors and CAC was already well-developed (54). Overall, these results are consistent with previous studies that suggested TAC at best modestly improves discrimination, primarily for noncoronary events.

CLINICAL IMPLICATIONS. A clinically relevant risk factor should not only provide independent prognostic value, but should also reclassify risk such that subsequent treatment changes. Screening with a primary purpose to assess for TAC has not met this high standard and should not be recommended. Likewise, in the context of CAC scanning, extending the field of view to include the aortic arch and proximal descending thoracic aorta should not be endorsed.

Calcification at these locations may identify a patient at higher risk for noncoronary events, but the extent of reclassification and implications for management are unclear. In addition, although contrast may refine risk by delineating noncalcified atheroma, the relevance in a primary prevention population is undefined. Finally, expressing TAC as a continuous variable with an Agatston score may increase the likelihood of observing a statistically significant result in a multivariable model, but whether this is clinically meaningful is still uncertain. However, with a CAC scan, most of the thoracic aorta is included without any additional radiation exposure. As an ancillary finding, a comment on TAC in conjunction with other noncoronary calcium deposits is reasonable, accepting that the prognostic value is almost entirely encapsulated with clinical risk factors and CAC.

DEFINITION, PREVALENCE, AND PROCEDURAL IMPLICATIONS OF A PORCELAIN AORTA

DEFINING A PORCELAIN AORTA. In severe cases of TAC, patients may be labeled as having a porcelain aorta, which is defined practically as severe calcification that prevents safe aortic cross-clamping or cannulation (55). Because of a lack of standardization, the term has been used inconsistently, and traditionally, various assessments aided in diagnosis included chest x-ray, fluoroscopy, and manual palpation (56). More recently, CT has been used for pre-procedural planning and has facilitated a more standard definition by delineating the location and circumferential extent of atherosclerosis (**Figure 9**) (57). Clinical trials in aortic stenosis (AS) have also been instrumental in this standardization. According to the Valve Academic Research Consortium-2 consensus, a porcelain aorta is defined as "heavy circumferential calcification or severe atheromatous plaque of the entire ascending aorta such that cross-clamping is not feasible" (58).

PREVALENCE OF A PORCELAIN AORTA. The true prevalence of a porcelain aorta in an asymptomatic primary prevention population is unknown, but rare. Because a porcelain aorta implies a modification to the standard surgical approach, a more relevant question relates to the prevalence in populations being evaluated for cardiac surgery. Specifically, investigators have focused on patients undergoing coronary artery bypass grafting (CABG), patients with severe AS, and patients with RACD.

In patients undergoing first-time isolated CABG, a porcelain aorta is uncommon. Of >1,800 consecutive patients with CABG at a single center, only 23 had a porcelain aorta (1.2%) (59). However, these patients were diagnosed without CT scanning. The actual prevalence is possibly higher, assuming that CT increases sensitivity beyond chest x-ray, fluoroscopy, and manual palpation. Of note, epiaortic ultrasound can also increase sensitivity for severe atheromatous plaque and has been used to modify an approach at the time of surgery (60).

A porcelain aorta is more common in patients with severe symptomatic AS. In the inoperable PARTNER (Placement of Aortic Transcatheter Valve) trial cohort, 15.1% of patients had a porcelain aorta, similar to the prevalence in a Canadian registry of high or prohibitive surgical risk patients with AS (18.0%) (61,62). The prevalence expectedly decreases in a population of AS patients with broader risk profiles and was 7.5% in 1 single-center study of 240 consecutive AS patients (63).

Finally, patients with RACD are also at high risk for a porcelain aorta. In a study of 117 patients with surgery for RACD, 59% had ascending aortic calcification, and 13% had severe circumferential calcification (64). In conclusion, because of the likelihood of a porcelain aorta in patients with severe AS and RACD, as well as the subsequent change in management, preprocedural CT is indicated. Moreover, although CT often identifies high-risk findings in patients with previous CABG and a plan for repeat cardiac surgery (65), the prevalence of a porcelain aorta in patients with a first-time CABG is not well-defined, but appears low. Further data are therefore needed before pre-operative chest CT can be more universally recommended.

RISKS AND MANAGEMENT OF A PORCELAIN AORTA. Patients with a porcelain aorta are at high risk for embolic stroke due to manipulation of aortic atheroma during surgery (66). For patients undergoing isolated CABG, a "no touch" approach is typically used, often with off-pump techniques, arterial grafting, and a radial graft as a side Y or T graft, if needed (67). With surgical aortic valve replacement, however, cardiopulmonary bypass and aortic manipulation are necessary. Many techniques have been described, and all add complexity to the surgery (68,69). Consequently, in most patients with severe AS and a porcelain aorta, transcatheter aortic valve replacement has become the preferred treatment option. Among the inoperable PARTNER cohort, a porcelain aorta was the most common reason for technical inoperability, and procedural outcomes were similar in these patients (70). In addition, transaortic

transcatheter aortic valve replacement may be possible in certain patients with a porcelain aorta with no significant calcium at the anterior and lateral aspect of the distal ascending aorta (71).

CONCLUSIONS

Calcification occurs in 2 sites of the vessel wall, the intima and the media. Despite overlapping pathologies, neointimal calcification is more often patchy and associated with typical atherosclerosis, whereas medial calcification is diffuse, concentric, and associated with diabetes mellitus, chronic kidney disease, systemic arteritis, and RACD. Like any imaging test, the assessment for TAC should have diagnostic, prognostic, and management implications. In a patient with an unknown cause for stroke, the severity and mobility of aortic atheroma on TEE aids in diagnosis, although it may not change standard secondary preventative therapies. With TTE, the extent of calcification may marginally improve global risk stratification, but the additive discriminative value is limited. Similarly, in routine CAC scanning, risk is almost entirely defined with clinical data and the coronary Agatston score, although discrimination may be slightly improved if TAC is incorporated with other noncoronary sites of calcification. Finally, CT has helped standardize the definition of a porcelain aorta as severe circumferential, or near circumferential, atherosclerosis that prevents safe cross-clamping. In patients with severe symptomatic AS or RACD, the presence of a porcelain aorta directly affects the procedural plan. For AS, TAVR is preferred because of the complexity involved with surgical aortic valve replacement and a porcelain aorta.

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REFERENCES

1. Corti R, Fuster V, Fayad ZA, et al. Lipid lowering by simvastatin induces regression of human atherosclerotic lesions: two years' follow-up by high-resolution noninvasive magnetic resonance imaging. Circulation 2002;106:2884–7.

2. Helft G, Worthley SG, Fuster V, et al. Progression and regression of atherosclerotic lesions: monitoring with serial noninvasive magnetic resonance imaging. Circulation 2002;105:993–8.

3. Lima JA, Desai MY, Steen H, Warren WP, Gautam S, Lai S. Statin-induced cholesterol lowering and plaque regression after 6 months of magnetic resonance imaging-monitored therapy. Circulation 2004;110:2336–41.

4. Marschall K, Kanchuger M, Kessler K, et al. Superiority of transesophageal echocardiography in detecting aortic arch atheromatous disease: identification of patients at increased risk of stroke during cardiac surgery. J Cardiothorac Vasc Anesth 1994;8:5–13.

5. Witteman JC, Kok FJ, van Saase JL, Valkenburg HA. Aortic calcification as a predictor of cardiovascular mortality. Lancet 1986;2:1120–2.

6. Danielsen R, Sigvaldason H, Thorgeirsson G, Sigfusson N. Predominance of aortic calcification as an atherosclerotic manifestation in women: the Reykjavik study. J Clin Epidemiol 1996;49:383–7.

7. Iijima K, Hashimoto H, Hashimoto M, et al. Aortic arch calcification detectable on chest x-ray is a strong independent predictor of cardiovascular events beyond traditional risk factors. Atherosclerosis 2010;210:137–44.

8. Agatston AS, Janowitz WR, Hildner FJ, Zusmer NR, Viamonte M Jr., Detrano R. Quantification of coronary artery calcium using ultrafast computed tomography. J Am Coll Cardiol 1990;15:827–32.

9. Fryback DG, Thornbury JR. The efficacy of diagnostic imaging. Med Decis Making 1991;11:88–94.

10. Fordyce CB, Douglas PS. Outcomes-based CV imaging research endpoints and trial design: from pixels to patient satisfaction. J Am Coll Cardiol Img 2017;10:253–63.

11. Sage AP, Tintut Y, Demer LL. Regulatory mechanisms in vascular calcification. Nat Rev Cardiol 2010;7:528–36.

12. Johnson RC, Leopold JA, Loscalzo J. Vascular calcification: pathobiological mechanisms and clinical implications. Circ Res 2006;99:1044–59.

13. Hoshino T, Chow LA, Hsu JJ, et al. Mechanical stress analysis of a rigid inclusion in distensible material: a model of atherosclerotic calcification and plaque vulnerability. Am J Physiol Heart Circ Physiol 2009;297:H802–10.

14. Stary HC, Chandler AB, Dinsmore RE, et al. A definition of advanced types of atherosclerotic lesions and a histological classification of atherosclerosis. A report from the Committee on Vascular Lesions of the Council on Arteriosclerosis, American Heart Association. Circulation 1995;92:1355–74.

15. Stone JR, Bruneval P, Angelini A, et al. Consensus statement on surgical pathology of the aorta from the Society for Cardiovascular Pathology and the Association for European Cardiovascular Pathology: I. Inflammatory diseases. Cardiovasc Pathol 2015;24:267–78.

16. Moe SM, Chen NX. Mechanisms of vascular calcification in chronic kidney disease. J Am Soc Nephrol 2008;19:213–6.

17. Shao JS, Cheng SL, Sadhu J, Towler DA. Inflammation and the osteogenic regulation of vascular calcification: a review and perspective. Hypertension 2010;55:579–92.

18. Amann K. Media calcification and intima calcification are distinct entities in chronic kidney disease. Clin J Am Soc Nephrol 2008;3:1599–605.

19. Paccou J, Brazier M, Mentaverri R, Kamel S, Fardellone P, Massy ZA. Vascular calcification in rheumatoid arthritis: prevalence, pathophysiological aspects and potential targets. Atherosclerosis 2012;224:283–90.

20. Hjortnaes J, New SE, Aikawa E. Visualizing novel concepts of cardiovascular calcification. Trends Cardiovasc Med 2013;23:71–9.

21. Lanzer P, Boehm M, Sorribas V, et al. Medial vascular calcification revisited: review and perspectives. Eur Heart J 2014;35:1515–25.

22. Vattikuti R, Towler DA. Osteogenic regulation of vascular calcification: an early perspective. Am J Physiol Endocrinol Metab 2004;286:E686–96.

23. Tunick PA, Kronzon I. Atheromas of the thoracic aorta: clinical and therapeutic update. J Am Coll Cardiol 2000;35:545–54.

24. Bertrand PB, Levine RA, Isselbacher EM, Vandervoort PM. Fact or artifact in two-dimensional echocardiography: avoiding misdiagnosis and missed diagnosis. J Am Soc Echocardiogr 2016;29:381–91.

25. Amarenco P, Cohen A, Tzourio C, et al. Atherosclerotic disease of the aortic arch and the risk of ischemic stroke. N Engl J Med 1994;331:1474–9.

26. Jeon DS, Atar S, Brasch AV, et al. Association of mitral annulus calcification, aortic valve sclerosis and aortic root calcification with abnormal myocardial perfusion single photon emission tomography in subjects age < or =65 years old. J Am Coll Cardiol 2001;38:1988–93.

27. Gaibazzi N, Porter TR, Agricola E, et al. Prognostic value of echocardiographic calcium score in patients with a clinical indication for stress echocardiography. J Am Coll Cardiol Img 2015;8:389–96.

28. Saric M, Armour AC, Arnaout MS, et al. Guidelines for the use of echocardiography in the evaluation of a cardiac source of embolism. J Am Soc Echocardiogr 2016;29:1–42.

29. Amarenco P, Duyckaerts C, Tzourio C, Henin D, Bousser MG, Hauw JJ. The prevalence of ulcerated plaques in the aortic arch in patients with stroke. N Engl J Med 1992;326:221–5.

30. Jones EF, Kalman JM, Calafiore P, Tonkin AM, Donnan GA. Proximal aortic atheroma. An independent risk factor for cerebral ischemia. Stroke 1995;26:218–24.

31. Tunick PA, Perez JL, Kronzon I. Protruding atheromas in the thoracic aorta and systemic embolization. Ann Intern Med 1991;115:423–7.

32. Meissner I, Khandheria BK, Sheps SG, et al. Atherosclerosis of the aorta: risk factor, risk marker, or innocent bystander? A prospective population-based transesophageal echocardiography study. J Am Coll Cardiol 2004;44:1018–24.

33. Ferrari E, Vidal R, Chevallier T, Baudouy M. Atherosclerosis of the thoracic aorta and aortic debris as a marker of poor prognosis: benefit of oral anticoagulants. J Am Coll Cardiol 1999;33:1317–22.

34. Corciu AI, Siciliano V, Poggianti E, Petersen C, Venneri L, Picano E. Cardiac calcification by transthoracic echocardiography in patients with known or suspected coronary artery disease. Int J Cardiol 2010;142:288–95.

35. Pressman GS, Crudu V, Parameswaran-Chandrika A, Romero-Corral A, Purushottam B, Figueredo VM. Can total cardiac calcium predict the coronary calcium score? Int J Cardiol 2011;146:202–6.

36. Nucifora G, Schuijf JD, van Werkhoven JM, et al. Usefulness of echocardiographic assessment of cardiac and ascending aorta calcific deposits to predict coronary artery calcium and presence and severity of obstructive coronary artery disease. Am J Cardiol 2009;103:1045–50.

37. Lu ML, Gupta S, Romero-Corral A, et al. Cardiac calcifications on echocardiography are associated with mortality and stroke. J Am Soc Echocardiogr 2016;29:1171–8.

38. Takasu J, Katz R, Nasir K, et al. Relationships of thoracic aortic wall calcification to cardiovascular risk factors: the Multi-Ethnic Study of Atherosclerosis (MESA). Am Heart J 2008;155:765–71.

39. Bild DE, Detrano R, Peterson D, et al. Ethnic differences in coronary calcification: the Multi-Ethnic Study of Atherosclerosis (MESA). Circulation 2005;111:1313–20.

40. Budoff MJ, Nasir K, Katz R, et al. Thoracic aortic calcification and coronary heart disease events: the Multi-Ethnic Study of Atherosclerosis (MESA). Atherosclerosis 2011;215:196–202.

41. Hoffmann U, Massaro JM, D'Agostino RB Sr., Kathiresan S, Fox CS, O'Donnell CJ. Cardiovascular event prediction and risk reclassification by coronary, aortic, and valvular calcification in the Framingham Heart Study. J Am Heart Assoc 2016;5:e003144.

42. Kalsch H, Lehmann N, Mohlenkamp S, et al. Prevalence of thoracic aortic calcification and its relationship to cardiovascular risk factors and coronary calcification in an unselected population-based cohort: the Heinz Nixdorf Recall Study. Int J Cardiovasc Imaging 2013;29:207–16.

43. Erbel R, Delaney JA, Lehmann N, et al. Signs of subclinical coronary atherosclerosis in relation to risk factor distribution in the Multi-Ethnic Study of Atherosclerosis (MESA) and the Heinz Nixdorf Recall Study (HNR). Eur Heart J 2008;29:2782–91.

44. Mahabadi AA, Lehmann N, Mohlenkamp S, et al. Noncoronary measures enhance the predictive value of cardiac CT above traditional risk factors and CAC score in the general population. J Am Coll Cardiol Img 2016;9:1177–85.

45. Wong ND, Gransar H, Shaw L, et al. Thoracic aortic calcium versus coronary artery calcium for the prediction of coronary heart disease and cardiovascular disease events. J Am Coll Cardiol Img 2009;2:319–26.

46. Wong ND. Is there more to the calcium scan than just coronary calcium? J Am Coll Cardiol Img 2016;9:1186–7.

47. Yeboah J, Carr JJ, Terry JG, et al. Computed tomography-derived cardiovascular risk markers, incident cardiovascular events, and all-cause mortality in nondiabetics: the Multi-Ethnic Study of Atherosclerosis. Eur J Prev Cardiol 2014;21:1233–41.

48. Santos RD, Rumberger JA, Budoff MJ, et al. Thoracic aorta calcification detected by electron beam tomography predicts all-cause mortality. Atherosclerosis 2010;209:131–5.

49. Allison MA, Hsi S, Wassel CL, et al. Calcified atherosclerosis in different vascular beds and the risk of mortality. Arterioscler Thromb Vasc Biol 2012;32:140–6.

50. Craiem D, Chironi G, Casciaro ME, Graf S, Simon A. Calcifications of the thoracic aorta on extended non-contrast-enhanced cardiac CT. PLoS One 2014;9:e109584.

51. Bos D, Leening MJ, Kavousi M, et al. Comparison of atherosclerotic calcification in major vessel beds on the risk of all-cause and cause-specific mortality: the Rotterdam Study. Circ Cardiovasc Imaging 2015;8:e003843.

52. Craiem D, Chironi DG, Casciaro ME, Sirieix ME, Mousseaux E, Simon A. Association of thoracic aorta calcium and noncardiac vascular events in cardiac disease-free individuals. Atherosclerosis 2016;245:22–7.

53. Kurra V, Lieber ML, Sola S, et al. Extent of thoracic aortic atheroma burden and long-term mortality after cardiothoracic surgery: a computed tomography study. J Am Coll Cardiol Img 2010;3:1020–9.

54. Tison GH, Guo M, Blaha MJ, et al. Multisite extracoronary calcification indicates increased risk of coronary heart disease and all-cause mortality: The Multi-Ethnic Study of Atherosclerosis. J Cardiovasc Comput Tomogr 2015;9:406–14.

55. Coselli JS, Crawford ES. Aortic valve replacement in the patient with extensive calcification of the ascending aorta (the porcelain aorta). J Thorac Cardiovasc Surg 1986;91:184–7.

56. Abramowitz Y, Jilaihawi H, Chakravarty T, Mack MJ, Makkar RR. Porcelain aorta: a comprehensive review. Circulation 2015;131:827–36.

57. Amorim PA, Penov K, Lehmkuhl L, Haensig M, Mohr FW, Rastan AJ. Not all porcelain is the same: classification of circular aortic calcifications (porcelain aorta) according to the impact on therapeutic approach. Thorac Cardiovasc Surg 2013;61:559–63.

58. Kappetein AP, Head SJ, Genereux P, et al. Updated standardized endpoint definitions for transcatheter aortic valve implantation: the Valve Academic Research Consortium-2 consensus document. J Am Coll Cardiol 2012;60:1438–54.

59. Leyh RG, Bartels C, Notzold A, Sievers HH. Management of porcelain aorta during coronary artery bypass grafting. Ann Thorac Surg 1999;67:986–8.

60. Davila-Roman VG, Barzilai B, Wareing TH, Murphy SF, Kouchoukos NT. Intraoperative ultrasonographic evaluation of the ascending aorta in 100 consecutive patients undergoing cardiac surgery. Circulation 1991;84 Suppl:III47–53.

61. Leon MB, Smith CR, Mack M, et al. Transcatheter aortic-valve implantation for aortic stenosis in patients who cannot undergo surgery. N Engl J Med 2010;363:1597–607.

62. Rodés-Cabau J, Webb JG, Cheung A, et al. Transcatheter aortic valve implantation for the treatment of severe symptomatic aortic stenosis in patients at very high or prohibitive surgical risk: acute and late outcomes of the multicenter Canadian experience. J Am Coll Cardiol 2010;55:1080–90.

63. Faggiano P, Frattini S, Zilioli V, et al. Prevalence of comorbidities and associated cardiac diseases in patients with valve aortic stenosis. Potential implications for the decision-making process. Int J Cardiol 2012;159:94–9.

64. Desai MY, Karunakaravel K, Wu W, et al. Pulmonary fibrosis on multidetector computed tomography and mortality in patients with radiation-associated cardiac disease undergoing cardiac surgery. J Thorac Cardiovasc Surg 2014;148:475–81.e3.

65. Kamdar AR, Meadows TA, Roselli EE, et al. Multidetector computed tomographic angiography in planning of reoperative cardiothoracic surgery. Ann Thorac Surg 2008;85:1239–45.

66. Davila-Roman VG, Murphy SF, Nickerson NJ, Kouchoukos NT, Schechtman KB, Barzilai B. Atherosclerosis of the ascending aorta is an

independent predictor of long-term neurologic events and mortality. J Am Coll Cardiol 1999;33:1308–16.

67. Sirin G, Sarkislali K, Konakci M, Demirsoy E. Extraanatomical coronary artery bypass grafting in patients with severely atherosclerotic (porcelain) aorta. J Cardiothorac Surg 2013;8:86.

68. Gillinov AM, Lytle BW, Hoang V, et al. The atherosclerotic aorta at aortic valve replacement: surgical strategies and results. J Thorac Cardiovasc Surg 2000;120:957–63.

69. Svensson LG, Sun J, Cruz HA, Shahian DM. Endarterectomy for calcified porcelain aorta associated with aortic valve stenosis. Ann Thorac Surg 1996;61:149–52.

70. Makkar RR, Jilaihawi H, Mack M, et al. Stratification of outcomes after transcatheter aortic valve replacement according to surgical inoperability for technical versus clinical reasons. J Am Coll Cardiol 2014;63:901–11.

71. Bapat VN, Attia RQ, Thomas M. Distribution of calcium in the ascending aorta in patients undergoing transcatheter aortic valve implantation and its relevance to the transaortic approach. J Am Coll Cardiol Intv 2012;5:470–6.

KEY WORDS computed tomography, echocardiography, thoracic aortic calcification

APPENDIX For supplemental videos, please see the online version of this paper.

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