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REVIEW

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Aortic regurgitation: A multimodality approach

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Abstract

Aortic regurgitation (AR) is a common valvular pathology. Multimodality noninvasive cardiovascular imaging is routinely used to assess the mechanism of AR, degree, and its hemodynamic impact on the cardiovascular system. Collecting this information is crucial in establishing the prognosis and in guiding patient management and follow-up. While echocardiography remains the primary test to assess AR, a comprehensive assessment of this valvulopathy can be obtained by combining the information from different techniques. This state-of-the-art review is intended to provide an update ed overview of the applications, strengths, and limits of transthoracic echocardiography, cardiac magnetic resonance, and cardiac computed tomography in patients with AR.

KEYWORDS
aortic regurgitation, cardiac magnetic resonance, echocardiography, computed tomography, multimodality imaging

1 | INTRODUCTION

Aortic regurgitation (AR) is defined as the presence of a regurgitant flow from the aorta to the left ventricle (LV) during diastole. Hemodynamic consequences are different in the acute or chronic setting. In severe acute AR, the volume overload on the LV results in an abrupt

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List of abbreviations: 2D, bi-dimensional; 3D, three-dimensional; 4D, four-dimensional; AR, Aortic regurgitation; BSA, Body surface area; CAD, Coronary artery disease; CMR, Cardiac magnetic resonance; CT, Computed tomography; ECG, Electrocardiographic; ECV, Extracellular volume; iECV, Indexed extracellular volume; EDD, End-diastolic diameter; EF, Ejection fraction; EROA, Effective regurgitant orifice area; CW, Continuous wave; GLS, Global longitudinal strain; LGE, Late gadolinium enhancement; LVOT, Left ventricular outflow ttract; LV, Left ventricle; PISA, Proximal isovelocity surface area; PW, Pulsed wave; PHT, Pressure half time; RF, Regurgitant fraction; RV, Regurgitant volume; SSFP, Steady state free precession; TAVI, Transcatheter aortic valve implantation; TTE, Transthoracic echocardiography; TEE, Transesophageal echocardiography; VC, Vena contracta.

and prominent increase in LV filling pressures. As a consequence, cardiac output and systolic pressure are dramatically reduced.[1]

In patients with chronic AR, volume and pressure overloads lead to progressive dilation and remodeling of the LV. Initially, these mechanisms of adaptation ensure preserved global systolic function. Diastolic pressure and cardiac output remain normal. In the earlier stages of the disease, patients could remain asymptomatic, and the hemodynamics are preserved. However, these compensation mechanisms may become inefficient in the long term, and LV dysfunction can occur with associated clinical symptoms.[2]

AR is an insidious and asymptomatic disease, requiring close and accurate monitoring during long periods to anticipate myocardial damage. If not treated, AR can result in increased LV volume and causes heightened preload and afterload, which may be responsible for LV dilatation and dysfunction. Definitive treatment in case of severe AR is surgical intervention, which prevents heart failure and death and can significantly improve clinical outcome.

Management of patients with severe AR and related indications for surgery is based on clinical symptoms, left ventricular size, and aortic dimensions.[3]

According to the European Society of Cardiology (ESC) guidelines, surgery is recommended in symptomatic patients with severe AR regardless of the LV systolic function and dimensions. In asymptomatic subjects, surgery is indicated in the presence of a LV ejection fraction (EF) ≤ 50% or LV end-systolic diameter > 50 mm.[3] Furthermore left ventricular end-systolic diameter should be indexed to the body surface area (BSA). Indeed, in subjects with low or large BSA who are not overweight, a value >25 mm/m^2 is an indication for surgery. In addition, in selected asymptomatic cases who are at low risk for surgery, surgery may be considered in the presence of a LV end-systolic diameter > 20 mm/m2 or LVEF between 50% and 55%.[3] In individuals with severe AR and an indication for coronary artery bypass grafting or surgery of the ascending aorta or another valve, intervention on the aortic valve is also recommended regardless of clinical symptoms.[3]

Surgery in severe AR is mainly based on valve replacement. However, a repair could be considered in referral centers in selected patients with good expected durability based on the aortic valve morphological characteristics. Transcatheter aortic valve implantation (TAVI) may be evaluated in selected patients with AR and contraindication to valve replacement at experienced centers.[4,5]

Close follow-up is indicated in individuals with severe asymptomatic AR and normal LV systolic function, a relevant increase in LV diameters, or a reduction in LVEF. Intervention may be considered in these patients with a progressive reduction in LVEF or increased ventricular diameter during follow-up and left ventricular end-diastolic diameter (LVEDD) > 65 mm.[3]

It appears clear that correctly grading AR and its hemodynamic repercussion is of crucial importance for patient's management since LV dysfunction caused by severe AR may be irreversible. This emphasizes the role of correct timing for surgical intervention. While echocardiography remains the first line test, other techniques can be used in order to solve specific diagnostic challenges. The role of each imaging modality is explained in the following sections.

2 | TRANSTHORACIC ECHOCARDIOGRAPHY

2.1 | Assessment of the AR mechanism

Transthoracic echocardiography (TTE) (Figure 1A,B,C) is the first-line method for assessing the AR mechanism, severity, secondary degree of LV remodeling, and hemodynamic consequences.

The aortic valve is made up of three semilunar leaflets inserted superiorly to the aortic media and inferiorly to the myocardium of the left ventricular outflow tract (LVOT) and the anterior mitral leaflet.[6] These leaflets are normally symmetrical, with three commissures and free movement in the three aortic sinuses.[1]

For the evaluation of AR, it is important to characterize both the etiology and the mechanism of AR. The etiology of AR is categorized as organic/primary when the regurgitation is mainly the result of structural leaflet abnormalities or as functional/secondary when regurgitation results from aortic root and/or ascending aorta dilatation in the presence of structural normal aortic valve leaflets. For primary AR, the most common causes of primary valve pathology in developed countries are degenerative AR and bicuspid aortic valve disease.[7] Other causes of AR are rheumatic disease, infective endocarditis, traumatic injuries, radiation-induced valve disease, and inflammatory disorders. Functional AR is secondary to aortic dissection, annulo-aortic ectasia, aortic aneurysm, Marfan syndrome, Ehlers–Danlos disease, and aortitis.[7]

The mechanism of AR is categorized according to Carpentier's classification into type I, II, and III.[8] Type I is characterized by normal cusps movement and aortic root dilatation or leaflet perforation. Type II is defined by the presence of leaflet prolapse with excessive movement (IIa), or cusp with free edge fenestration (IIb), and consequent eccentric jet. Type III is characterized by leaflet restrictive motion.[8]

2.2 | Assessment of the severity of AR

The echocardiographic evaluation of the severity of AR is based on a multiparametric assessment which includes qualitative, semi-quantitative, and quantitative parameters[9] (Table 1). The vena contracta and proximal isovelocity surface area (PISA) method are the most recommended parameters. Nonetheless, AR should always be evaluated in the context of other parameters corroborating the AR severity. This approach should be utilized to assess any AR, unless specific signs of severe regurgitation such as flail leaflet or a large coaptation defect are present. Assessment of AR using the extension of regurgitant jets color flow Doppler imaging alone is inadequate and dependent on LV compliance and diastolic pressure gradient.

Color flow Doppler (Figure 1A,E) defines the AR jet in three components: flow convergence, vena contracta, and jet turbulence. The vena contracta (VC) method is based on the estimation of effective regurgitant orifice area (EROA) size. Parasternal long-axis view is indicated for measuring the vena contracta width, immediately below the flow convergence zone.[10] The Nyquist limit for the diameter

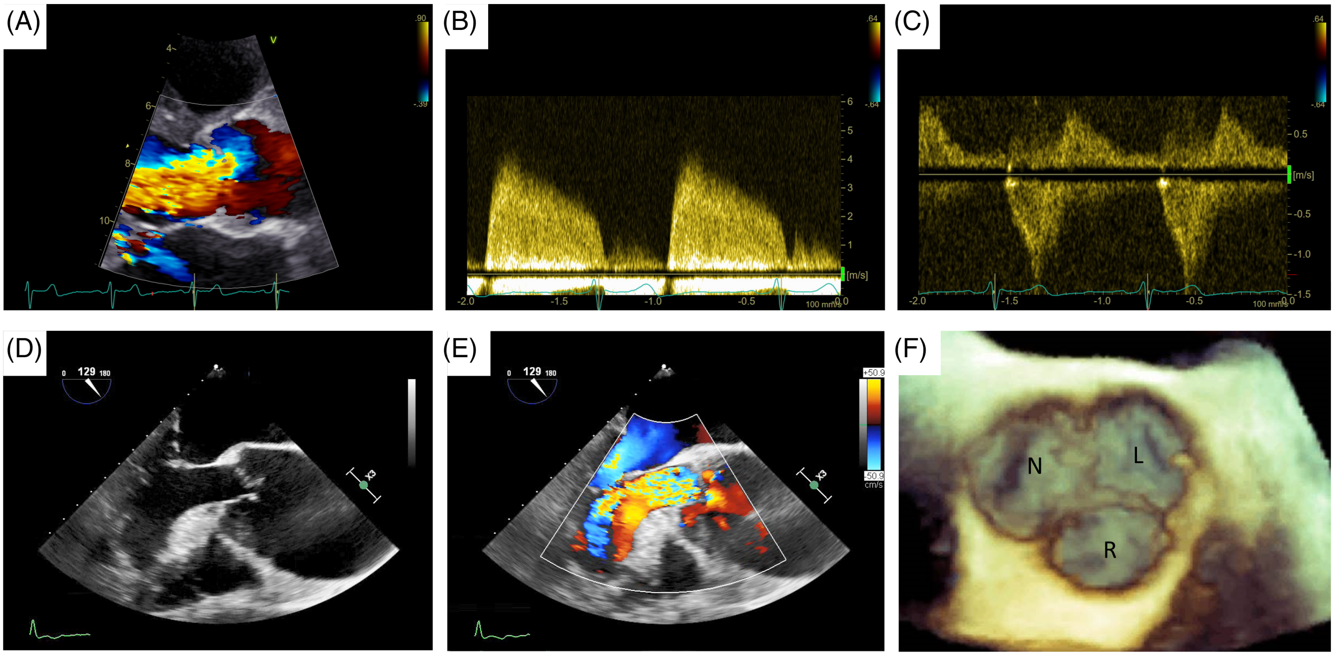

FIGURE 1 Echocardiographic assessment of aortic regurgitation: (A) TTE colorDoppler focused on left ventricle outflow tract showing a large aortic regurgitant jet, (B) TTE CW Doppler of the regurgitant jet, (C) TTE CW Doppler showing holodiastolic flow reversal in the descending aorta. (D,E) TEE assessment of a patient with severe aortic regurgitation due to a large coaptation defect secondary to aortic root dilatation. (F) 3D TEE assessment of the aortic valve 3D: three dimensional. CW, continuous wave; TTE, transthoracic echocardiography; TEE, transoesophageal echocardiography

estimation is set to 50–60 cm/s with the optimization of the ratio between scale and color grade. In addition, a zoomed view of the aortic valve and maximization of frame rate is suggested. This method is independent of hemodynamic factors such as flow rate or diastolic pressure gradient. It is relatively independent of technical errors and can estimate eccentric jets.

VC width <3 mm is associated with mild AR, intermediate values of 3–6 mm with moderate AR, and values >6 mm indicate severe AR.

However, in case of AR with multiple jets, the value of VC is limited, and it is not advisable to add single widths. Another limitation of this method concerns the assumption of VC. Indeed, VC measurement is based on the circular shape of EROA, however, the orifice can be irregular (or triangular) and therefore change in diameter in different views.

PISA method allows the direct estimation of EROA and regurgitant volume. This approach is based on the study of flow convergence generated around the regurgitant orifice and the hemispheric concentric velocity shells related to the flow area.[11] Specifically, the parasternal long-axis view is preferred for eccentric jets, while the apical five or three-chamber view in the case of central jets.[12] A zoom view of AR color flow imaging is suggested, and the Nyquist limit is shifted in the direction of the jet to acquire a hemispheric proximal flow convergence area. Measurement of PISA radius is recommended using the first aliasing during diastole, removing the color Doppler to correctly visualize the regurgitating orifice. Consequently, the calculation of EROA and regurgitant volume is performed by the measurement of the peak velocity and velocity-time integral (VTI) from the AR continuous-wave Doppler signal.

An EROA <10 mm^2 or regurgitant volume < 30 mL is classified as mild AR. An EROA of 10–19 mm^2 or regurgitant volume of 30–44 mL is subclassified in mild-to-moderate AR, while in moderate-to-severe AR an EROA of 20–29 mm^2 or regurgitant volume of 45–59 mL is present. Finally, an EROA ≥30 mm^2 or regurgitant volume ≥ 60 ml indicates severe AR.

However, this method is affected by several limitations.[13,14] The presence of valve calcifications reduces the feasibility. It is not also reliable in the presence of multiple jets, and alteration of the PISA assumption determines a severity under-or overestimation (e.g., noncircular orifice, adjacent contrasting flows, or nonplanar flow convergence).

Several other parameters should be evaluated to confirm the AR severity. The ratio between jet width and LVOT diameter indicates a severe AR when >65%.[15] The pressure half time (PHT) from the continuous wave (CW) regurgitant Doppler trace depends from the LV and aortic compliance, and it is associated with severe AR if <200 ms. The regurgitant fraction is calculated from the regurgitation volume divided by the LVOT stroke volume and indicates severe AR when ≥50%.

The strongest additional method for assessing AR is diastolic flow reversal in aorta (Figure 1C).[16] Specifically, holodiastolic flow reversal in the descending aorta, with end-diastolic velocity ≥ 20 cm/s at peak R wave, is associated with severe AR. Pulsed wave Doppler sample is

TABLE 1 AR severity subclasses according to the European Association of Cardiovascular Imaging[9]

AR severity classes	Mild	Moderate		Severe
AR severity subclasses	Mild	Mild-to-moderate	Moderate-to-severe	Severe
Qualitative parameters				
Aortic valve morphology	Normal/abnormal	Normal/abnormal	Abnormal/prolapse/ moderate coaptation defect	Abnormal/**flail/large coaptation defect**
Color flow AR jet width	Small in central jets	Intermediate	**Large in central jet**, variable in eccentric jets	**Large in central jet**, variable in eccentric jets
Color flow convergence	None or very small	Intermediate	Intermediate	Large
CW signal of AR jet	Incomplete/faint	Dense	Dense	Dense
Diastolic flow reversal in descending aorta	Brief, proto-diastolic flow reversal	Intermediate	Holodiastolic flow reversal (end-diastolic velocity 10 to <20 cm/s)	**Holodiastolic flow reversal** (end-diastolic velocity ≥ 20 cm/s)
Diastolic flow reversal in abdominal aorta	Absent	Absent	Present	Present
Semi-quantitative parameters				
VC width (mm)	<3	3–6	3–6	**>6**
Jet width/LVOT diameter (%)	<25	25–45	46–64	**≥65**
Jet CSA/LVOT CSA (%)	<5	5–20	21–59	**≥60**
Pressure half-time (ms)	>500	Intermediate, 500 to 200	Intermediate, 500 to 200	**<200**
Quantitative parameters				
EROA (mm^2)	<10	10–19	20–29	≥30
R vol (mL)	<30	30–44	45–59	≥60
RF (%)	<30	30–39	40–49	≥50
CMR parameters				
RF (%)	<30	30–39	40–49	≥50
Structural parameters				
LV size	Usually normal	Normal or dilatated	Usually dilatated	Usually dilatated

Note: In bold: specific signs for severe AR.

Abbreviations: AR, aortic regurgitation; CSA, cross-sectional area; CW, continuous wave; EROA, effective regurgitant orifice area; LA, left atrium; LV, left ventricle; RF, regurgitant fraction; R Vol, regurgitant volume; VC, vena contracta.

placed in the upper descending aorta, just below the left subclavian artery, with a Doppler filter <10 cm/s.

The presence of diastolic reversal flow in the abdominal aorta is a parameter with a high specificity for severe AR.

Three-dimensional (3D) echocardiography provides incremental information about aortic valve anatomy and AR hemodynamic consequences on cardiac chambers geometry and function. Indeed, this method allows a better evaluation of volumes and LVEF.[16] In addition, 3D color flow Doppler enables direct planimetry assessment of the VC area, revealing a circular or more elliptical shape of the jet, by using multiplanar reconstructions parallel to the axis of the AR flow.[17]

Transesophageal echocardiography (TEE) (Figure 1D,E) is recommended in case of indeterminate AR severity due to an inconclusive TTE or the need for further diagnostic definition in case of discrepancy between AR severity and clinical status.[9] In addition, TEE improves the accuracy of TTE in specific situations such as endocarditis, and isolated aortic root dilation.[18–20] Furthermore, 3D TEE (Figure 1F) is useful for evaluating the aortic valve morphology and the AR mechanism (in particular valuable in selecting patients for aortic valve repair), while the multiplanar color Doppler allows to calculate the 3D vena contracta.[16]

Exercise echocardiography helps assess symptomatic status in patients with severe and asymptomatic AR. Furthermore, this method can provide additional parameters such as LV contractile reserve[21] Exercise-induced change in LVEF is determined by several components such as systolic contractile function, degree of volume overload variation in preload, and resistance during effort.[9]

2.3 | Left ventricle remodeling assessment

Hemodynamic changes on the LV alter its dimensions and systolic function. Generally, LV dilatation is present in chronic AR of moderate-to-severe grade. Instead, LV size is preserved in acute severe or chronic mild AR. LV is identified as dilated in presence of end-diastolic diameter > 56 mm, end-diastolic volume > 82 ml/m2,

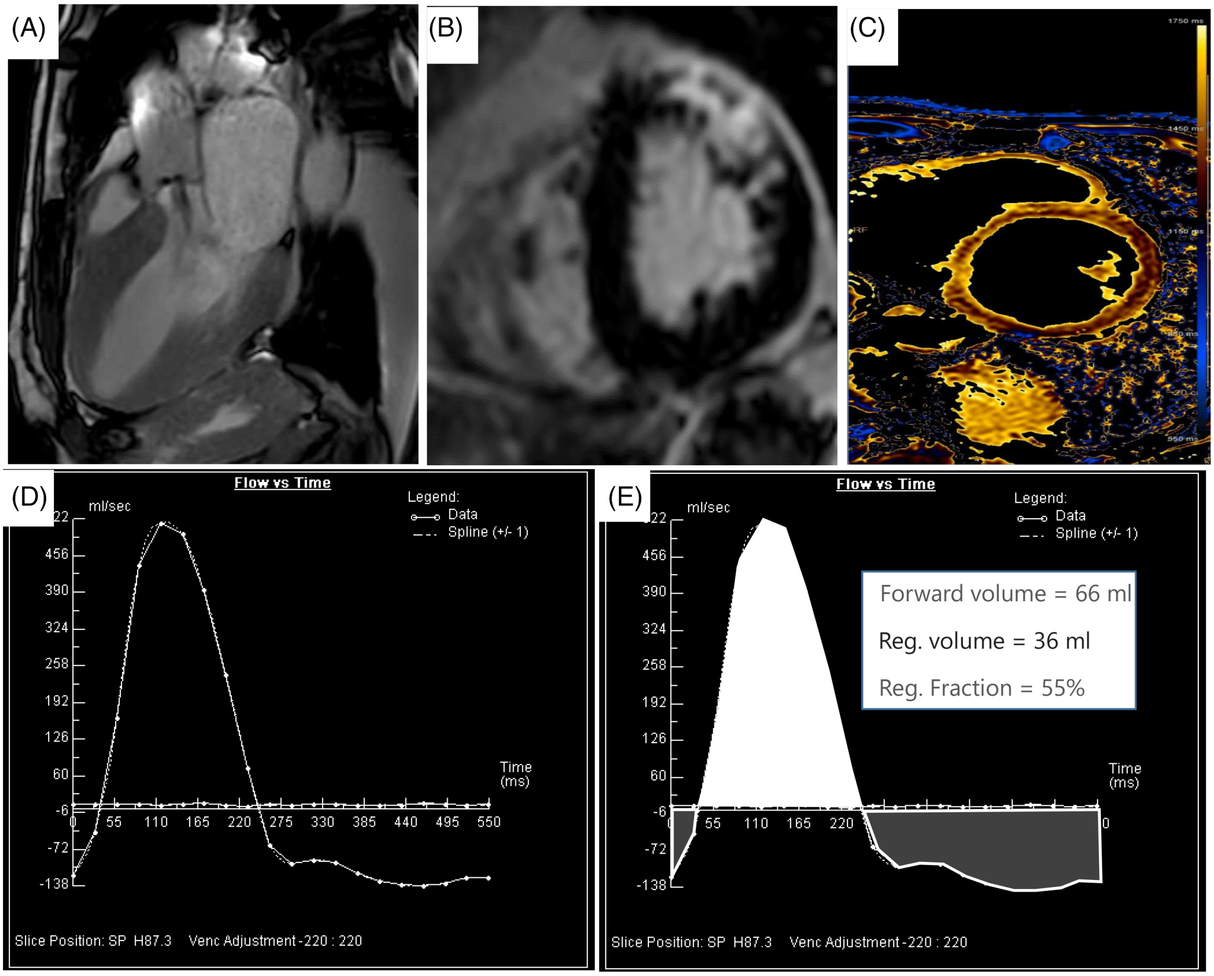

FIGURE 2 Cardiac magnetic resonance assessment of aortic regurgitation: (A) Visualization of a "jet" (hypointense signal due to phase loss of proton spin due to turbulent flow) of aortic insufficiency with a cine-SSFP sequence; (B) late gadolinium enhancement sequence showing an intramyocardial enhancement of the anterior and antero-lateral wall of the left ventricle. (C) Native T1 mapping to assess the presence of interstitial fibrosis, (D) flow/time curve phase contrast derived: positive values refer to forward flow while negative values indicate backward (regurgitant) flow, (E) example of measurement of regurgitant volume (Reg. volume) and regurgitant fraction (Reg. fraction) using flow/time curve phase contrast derived. SSFP, steady state free precession

end-systolic diameter > 40 mm, or end-systolic volume > 30 ml/m2.[9] In addition, chronic AR generates a progressive, irreversible damage to the LV with a reduction in EF. This reduction in LVEF to a value <50% might represent a late stage of myocardial overload and irreversible damage could be prevented by better cut-off values. Indeed, some reports have already suggested that the cut-off level of LVEF should probably be raised to 55%.[22]

Perhaps a better method to quantify myocardial function is by means of myocardial deformation imaging. Global longitudinal strain (GLS) is a simple and very robust parameter derived from bidimensional (2D) echocardiographic images. Several studies have shown that in asymptomatic patients with more than moderate chronic AR and preserved LVEF, worsening LV-GLS was associated with longer term mortality, providing incremental prognostic value and improved reclassification.[23] In addition, in patients with a preserved LVEF but with a reduced GLS, long term outcome is worse than those with a preserved GLS after AVR, indicating that this early systolic dysfunction is already associated with adverse remodeling impacting outcome.[24]

3 | ROLE OF CARDIAC MAGNETIC RESONANCE IN AORTIC REGURGITATION

Cardiac magnetic resonance (CMR) (Figure 2) has an emerging role in the assessment of the patient with AR for several reasons. First, it represents the current reference standard for evaluating cardiac volumes, mass, and systolic function.[25] Second, it characterizes the myocardial tissue, providing additional prognostic information.[26] Third, CMR allows anatomical and functional assessment of the aortic valve and the entire thoracic aorta. Finally, CMR provides accurate quantification of AR regurgitant volume and fraction. CMR is recommended by both ESC and American Heart Association/ American College of Cardiology (AHA/ACC) as a valuable complementary modality to quantify AR where echocardiography results are inconclusive.[27]

3.1 | Assessment of aortic valve

Morphology and function of the aortic valve is assessed with steady-state free precession (SSFP) pulse sequences (Figure 2A).

A stack of cines SSFP covering the aortic valve is used to assess the planimetry of the aortic valve which provides the size of the aortic valve area.[28]

However, the spatial resolution of CMR is often not enough to visualize small vegetations in infective endocarditis and small valvular masses especially if high mobile.[28]

Moreover, cine SSFP imaging is affected by arrhythmias.

3.2 | Assessment of thoracic aorta

The assessment of the aortic root is crucial for the correct identification of the AR etiology (e.g., hypertension, aortic dissection, and Marfan syndrome) as well as for the surgical planning of aortic root repair/replacement.[29] Furthermore, the CMR is accurate for evaluating the thoracic aorta.[29,30] The use of SSFP sequences has become prevalent because they broadly provide high contrast between liquid compartments and surrounding tissue, making it suitable for vessel imaging.[31] Moreover, the 3-dimensional contrast-enhancement magnetic resonance angiography (the so called “3D Whole Heart”) is helpful to visualize and assess the thoracic aorta and for the surgical plan and follow-up after the intervention.[32] Eventually, four dimensional (4D) flow MRI is emerging as a tool that can be used to study both thoracic aorta anatomy and aortic flow dynamics. This new sequences allow quantification of blood flow volume and velocity and study of velocity-encoded 3D path lines.[33,34]

3.3 | Left ventricular remodeling

SSFP pulse sequences are validated for evaluating LV volume, mass, and function.[35] Ventricular volumes are calculated from a short-axis stack of 6–8 mm thick slices with an interslice gap of 4 mm. CMR can assess regional fibrosis using late gadolinium enhancement (LGE) imaging (Figure 2B), which represents the noninvasive reference standard to quantify myocardial fibrosis.[28] A recent study found that in a third of patients with AR was present LGE and that it was independently associated with a 2.5-fold increase in mortality in patients with moderate or severe AR. Moreover, in patients with LGE, aortic valve surgery was associated with a better prognosis as compared to medical therapy.[36] In addition to LGE assessment, T1 mapping techniques are useful in quantifying the extracellular matrix expansion, which can be inferred to represent diffuse interstitial fibrosis in several cardiac disorders, including those caused by chronic hemodynamic (volume and/or pressure) overload in valvular heart disease (Figure 2C).[37] In the most recent literature, two surrogate CMR biomarkers for diffuse interstitial fibrosis have emerged: global extracellular volume fraction (ECV), which measures the proportion of LV myocardium that is extracellular matrix; and indexed ECV (iECV),the amount of extracellular matrix correlated to LV mass.[38] Senapati et al. demonstrated that iECV is more strongly associated with AR severity and adverse clinical outcomes than ECV or replacement fibrosis. Because iECV represents the total LV fibrosis burden, it better characterizes the remodeling changes occurring in progressive AR that lead to cellular and extracellular expansion. Future multicenter randomized studies are needed to validate using iECV as a CMR prognostic marker for clinical outcomes.

3.4 | Aortic regurgitation quantification

Phase-contrast sequences (Figure 2D,E) are used to assess the AR severity. Specifically, phase contrast velocity mapping planned at the sino-tubular junction level can quantify regurgitant volumes (RV) and fractions (RF).[25] RV and RF are prognostic independent predictors in patients with AR. Indeed, A RF of >33% and a RV > 42 ml predict the likelihood of intervention.[28] In another study, all patients with RF <26% had no need for surgery.[39] In Table 1 are reported the cut off of RF to establish AR severity in CMR according to the recent recommendation of the European Association of Cardiovascular Imaging.[9]

However, the RV and RF measurements are reproducible and reliable only when the flow is laminar or noncomplex. When there are pathologies that determinate complex flow, such as a bicuspid valve or ascending aortic dilatation, the measurements may be biased and no longer reliable, as has been shown by Frida Truedsson et al.[40]

Finally, 4D flow CMR has been studied as an alternative method for flow measurement. According to the literature, values obtained by 4D flow CMR are more accurate than the 2D Phase Contrast in the presence of turbulent flows.[41,42] However, 4D flow is still not commonly used in clinical routines to quantify AR.

4 | ROLE OF CT IN AORTIC REGURGITATION

Computed tomography (CT) is frequently used preoperatively in patients with AR as it gives accurate information about the aorta size and morphology of the valve. It can also rule out the presence of associated coronary artery disease. The scan protocol includes the use of iodinated contrast agents and electrocardiographic (ECG) gating, with retrospective reconstruction or prospective ECG triggering. Retrospective ECG gating offers a bigger acquisition of data by scanning through multiple cardiac cycles. This allows the imager to postprocess different cardiac phases but gives the patient a higher amount of radiation. Prospective ECG gating allows the acquirement of images once triggered by the R peak on the ECG tracing leading to a lower exposition for the patient.[43,44]

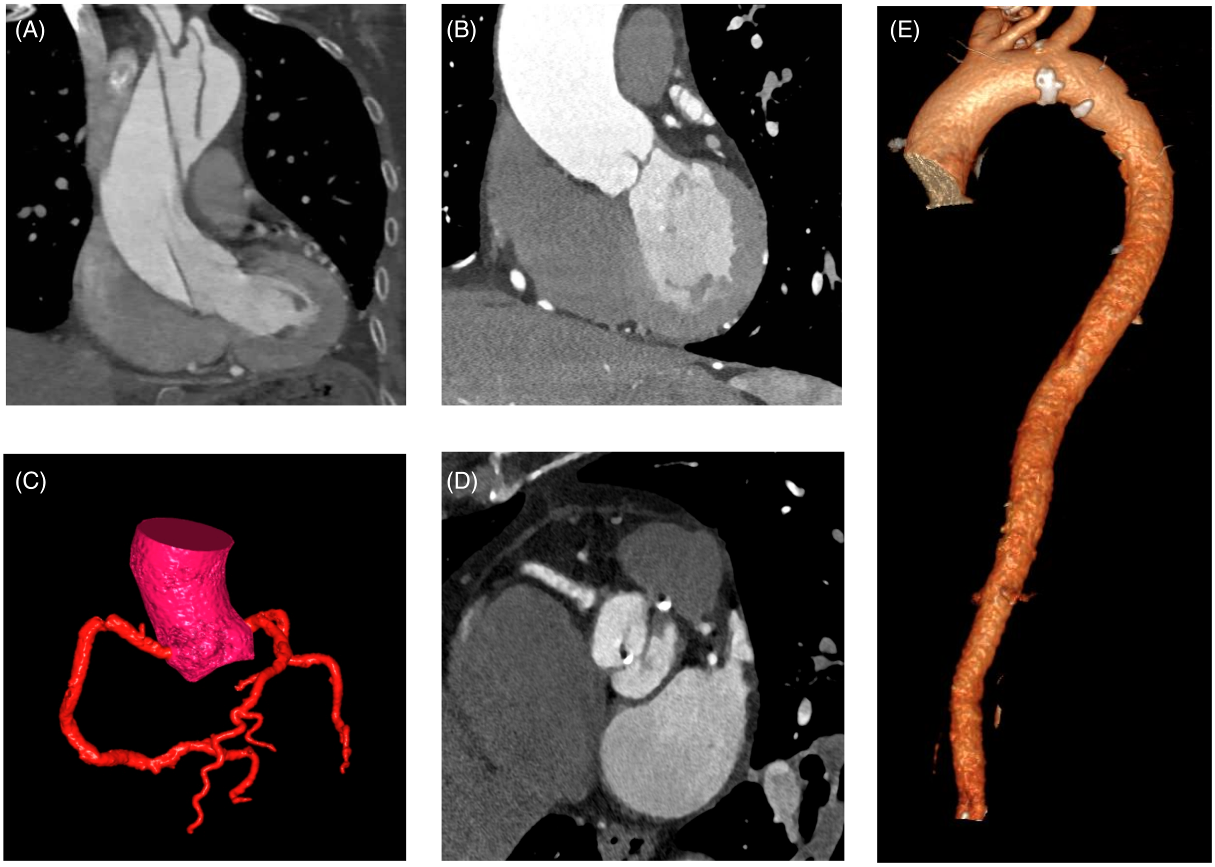

FIGURE 3 Computed tomography assessment of aortic regurgitation. (A) type A aortic dissection in a patient with AR, (B) aorta ascendens dilatation in a patient with moderate AR (C), volume rendering of the coronary arteries, (D) Bicuspid aortic valve with degeneration of the valve leaflets, (E) volume rendering of the thoracoabdominal aorta. AR, aortic RegurgitationFlow

4.1 | Assessment of the aorta

Imaging and measuring the aortic size is fundamental for stratification and surgical planning of patients with AR. CT can provide a detailed anatomic assessment of the thoracic aorta. Maximum diameters are measured specifically at four levels: annulus, sinus of Valsalva, sinotubular junction, and tubular ascending aorta (Figure 3B,C).[3] One of the major causes of acute AR is aortic dissection (Figure 3A). Contrast-enhanced, ECG-gated CT angiography, thanks to its wide availability, short scan time, a sensibility of 100%, and a specificity of 98% is commonly used to rule out aortic dissections.[45,46] Furthermore, CT leads to an optimal visualization of the aortic root and the valvular plan (Figure 3D).

Frazao et al. compared the agreement of TTE, CT, and CMR in sizing the aortic root. While CT and CMR did not show significant differences, TTE longitudinal measurement was significantly smaller than the measurements from CCT and CMR. Moreover, since the aortic root is not symmetric (especially in bicuspid aortic valve), a one-plane echocardiography measurement can lead to an underestimation of the real size of the aortic valve.[47–49] Various methods have been proposed to assess the aortic root in CT. The latest evidence suggests calculating diameters using the inner-inner edge technique by taking sinus-to-sinus diameters rather than sinus-to-commissure as the sinus-to-commissure method can underestimate the real enlargement.[3,47,49,50]

CT can be useful to rule out coronary artery disease (CAD) in patients who must undergo an intervention (Figure 3E).[51] 2021 ESC/European Association Cardiovascular Thoracic Surgery (EACTS) guidelines for the management of valvular heart disease propose the use of CT as an alternative to coronary angiography to rule out CAD in patients with low atherosclerosis risk seeing the high negative predictive value of the CT.[3]

4.2 | Aortic valve anatomy and regurgitation

CT can help to evaluate aortic valve morphology and pathology (e.g., calcifications, prolapse, infective process, rheumatic diseases). Koo et al. demonstrated that the functional classification of AR is useful for TAVI planning and that CT can show cusp abnormalities that can lead to tailored intervention.[52] Moreover, a precise understanding of the aortic root and valve via CT is helpful in predicting surgical reparability and can spare the patient unnecessary aortic root replacement.[52]

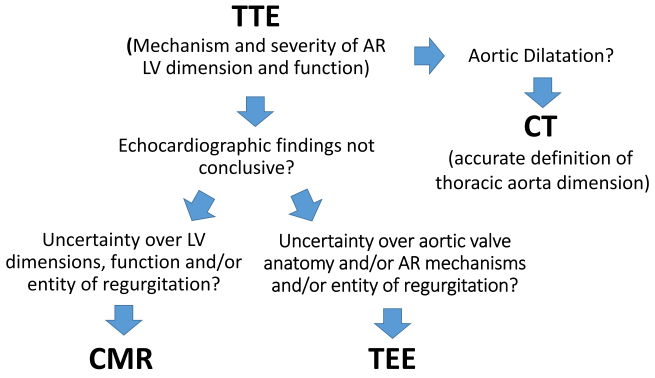

FIGURE 4 How to choose the right imaging modality in patients with aortic regurgitation. CMR, cardiac magnetic resonance; CT, computed tomography; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography

Endocarditis is one of the major causes of AR. In the last few years, the importance of a deep understanding of the pathological findings before the surgery is increased, and so has the importance of CT.[53,54] Retrospective electrocardiographic (ECG) gated CT is the best modality to visualize and localize abscess and calcification.[53] Latest literature remarks on the superiority of TEE for small vegetations but underlines the better comprehension of the CT for the periannular complications, the presence of intracoronary embolism, and prosthetic valve infection.[53,55,56]

The importance of CT is also emerging for assessing valve functionality and complications. Several studies has been showing how CT can be helpful in evaluating aortic prosthetic valve dysfunctions by assessing opening and closing angles with a strong correlation with the fluoroscopy results. Moreover, CT is useful for highlighting the presence of a fibrous pannus or thrombi.[57–59]

4.3 | LV sizing and myocardial fibrosis

CMR remains the gold standard for LV volume, function and mass measurement. Nevertheless, in patients with contraindication to CMR, ECG-gated CT offers a useful tool to define LV size and systolic function.[60] Moreover, Klein et al. found a correlation between LV mass/BSA measurement, sizing the LV with CT and adverse cardiac major events.[61]

As iodinated-contrast agents and gadolinium-based materials have similar pharmacokinetics, cardiac CT can be used as an alternative to CMR in assessing focal myocardial fibrosis.[62]

Moreover, extracellular volume can be calculated with cardiac CT in order to assess diffuse fibrosis.[63] However, no studies have explored so far the role of CT tissue characterization in patients with AR.

5 | CONCLUSIONS

Multimodality imaging provides great complementary data that can be tailored to the single individual and to the specific clinical case (Figure 4).

Echocardiography remains the first technique to be used in patients with AR. The assessment starts with TTE in order to precise the mechanism and severity of regurgitation. Then, TTE defines the hemodynamic impact of valvulopathy. In particular, LV remodeling is assessed by measuring volumes and myocardial function. The latter, classically identified with LVEF can be more precisely measured using deformation parameters, in particular GLS.

In presence of an inadequate acoustic window and when the parameters used to grade the regurgitation are discordant, TEE and CMR can be valuable additional techniques in patients with AR. In particular, CMR not only represents the gold standard for the assessment of volumes and EF but is used to accurately determine the regurgitant volume and fraction of AR providing in doubtful cases diriment information for surgical indication. Furthermore, CMR tissue characterization with LGE and T1 mapping can be used to better stratify patient's prognosis.

Finally, in patients candidate to surgery, CT provides accurate measurement of thoracic aorta and can rule out coronary artery disease. Moreover, CT can be particularly useful to study patients with aortic prosthesis dysfunction and to identify periannular complication in patients with endocarditis of the aortic valve.

The study of flow patterns with 4D-flow CMR or with particle image velocimetry or vector flow mapping in echocardiography are still in the realm of research. However, we expect that in the future they will provide other precious piece of information to further improve AR patient's care.

CONFLICT OF INTEREST

The authors declare no relationship with industry and financial associations within the past two years that pose a conflict of interest in the submitted article.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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