DOI: 10.15825/1995-1191-2025-3-97-109

THE POTENTIAL OF MULTISLICE COMPUTED TOMOGRAPHY IN DIAGNOSING CORONARY ARTERY DISEASE IN HEART TRANSPLANT RECIPIENTS: A LITERATURE REVIEW

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Coronary artery disease remains a leading cause of graft failure after heart transplantation (HT). Because the transplanted heart is denervated, graft ischemia is typically asymptomatic, necessitating annual screening to detect cardiac allograft vasculopathy (CAV), monitor established coronary lesions, and evaluate in-stent restenosis. The need for annual invasive coronary angiography, along with its associated risks, including potentially life-threatening complications, underscores the need for safer, yet equally effective, noninvasive diagnostic alternatives for evaluating coronary pathology in heart transplant recipients. Multislice computed tomography coronary angiography (MSCT–CAG) has been successfully employed in the diagnosis of ischemic heart disease (IHD) for many years and is well-established as a noninvasive alternative to conventional coronary angiography. This makes it particularly relevant to investigate its applicability and effectiveness in the post-transplant setting.

Keywords: cardiac allograft vasculopathy, MSCT coronary angiography, heart transplantation.

About 6,000 orthotopic heart transplants (OHTs) are performed worldwide each year. In 2024, a total of 450 OHTs were performed in Russia, 294 of which took place at Shumakov National Medical Research Center of Transplantology and Artificial Organs, Moscow [1]. One of the leading causes of graft loss is cardiac allograft vasculopathy (CAV), a form of coronary artery disease specific to the transplanted heart. According to the International Society for Heart and Lung Transplantation (ISHLT) registry, CAV prevalence steadily increases with time after transplantation, reaching 10% at 1 year, 22% by 4 years, 35% by 7 years, 44% by 10 years, 56% by 15 years, and 59% by 20 years post-transplantation. One in eight transplant recipients develops moderate to severe CAV (grades 2–3) within ten years of surgery (Table 1), and one in four develops it within twenty years [3].

The pathogenesis of CAV is driven by a combination of immune and non-immune factors, resulting in inflammation of the vascular wall followed by proliferative changes, fibrosis, and remodeling of the vessel [4, 5]. The disease process generally evolves in two phases. The initial phase involves endothelial injury, leading to intimal thickening with expansion of the adventitia, while the coronary lumen may initially remain relatively preserved. As the disease progresses, fibroproliferative cellular responses occur, resulting in constrictive remodeling and stenotic narrowing of the coronary artery lumen [6, 7].

Unlike the focal and eccentric atherosclerotic plaques typically seen in ischemic heart disease, CAV is characterized by a diffuse and concentric pattern of involve-

Table 1

Classification system for angiographic signs of heart transplant vasculopathy [2]

Grade	Criteria
ISHLT CAV 0 (minor)	No angiographic lesions
ISHLT CAV 1 (mild)	Left main (LM) stenosis <50%, primary vessel stenosis <70%, branch stenosis <70% (including diffuse lesions); no graft dysfunction
ISHLT CAV 2 (moderate)	LM stenosis >50%, primary vessel stenosis >70%, or stenosis >70% in any second-order branch; no graft dysfunction
ISHLT CAV 3 (severe)	Stenosis of the LM >50%, or stenosis >70% in two or more primary branches or any second-order branch in all three major territories and/or ISHLT CAV1 or CAV2 with graft dysfunction (LVEF <45%, regional wall motion abnormalities, or restrictive diastolic dysfunction)

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ment. Both epicardial and intramural coronary arteries are primarily affected [8, 9].

Risk factors for the development of CAV include: donor age over 45 years, male sex of both donor and recipient, and lipid metabolism disorders in the recipient before and after transplantation (LDL >2.5 mmol/L). Additional risk factors include episodes of acute cellular rejection, presence of donor-specific HLA antibodies, smoking, hypertension, diabetes mellitus, and obesity. Cytomegalovirus (CMV) infection also plays a major role in the pathogenesis of CAV [2, 10, 11].

The main contributing factors leading to CAV are summarized in Table 2.

Due to denervation of the transplanted heart, ischemia often remains clinically silent, and CAV may remain asymptomatic for a prolonged period. When symptoms do appear, they are often nonspecific, such as fatigue, nausea, abdominal discomfort, or heart rhythm disturbances (tachyarrhythmias, bradyarrhythmias, frequent supraventricular or ventricular extrasystoles). By this stage, patients may already exhibit a reduction in left ventricular ejection fraction and symptoms of heart failure, which are associated with a poor prognosis. There-

Table 2

Comparative role of various factors in the pathogenesis of CAV *vs* native atherosclerosis [11]

CAV		Atherosclerosis
	Endothelial cell	
+++	Hyperpermeability	++
+++	Dysfunction	++
	Smooth muscle cell	
++	Proliferation	+++
+++	Apoptosis	++
	Abnormal accumulation and functioning of the extracellular matrix	
+++	Proteoglycan deposition	+
++	Collagen overexpression (fibrosis)	+++
++	Altered myogenic vascular tone (resistance vessels)	+
+++	**Expression of adhesion molecules**	+++
	Inflammatory cells	
+	Platelets	+++
++	Monocytes/macrophages	+++
+++	Lymphocytes	++
	Immune response	
+++	Innate/acquired immunity	++
+++	**Lipid retention**	++
	Risk factors	
++	High blood cholesterol levels	+++
+++	High blood triglyceride levels	+
+	Low blood HDL levels	+++
++	Hyperhomocysteinemia	+
–/+	Infection	–/+
–/+	Age	+++
–/+	Gender	++
–/+	Ethnic predisposition	+
–/+	Previously diagnosed vascular diseases	+++
++	Smoking	+++
++	Diabetes mellitus	+++
++	Arterial hypertension	++
++	Obesity	++
–/+	Physical inactivity	++
++	Special medications	+
–/+	Social class	+
–/+	Psychosocial environment	++
–/+	Type A personalities	–/+
+	Donor-associated diseases	–
–	Family history	++

fore, meticulous monitoring of the cardiac graft for early signs of CAV is necessary.

According to the ISHLT 2023 guidelines, coronary angiography remains the gold standard for diagnosing CAV (Class I, Level of Evidence: C) and is recommended every 1–2 years throughout follow-up. More frequent evaluation may be warranted in patients with previously documented coronary pathology to monitor progression or restenosis after stent implantation [2]. However, invasive coronary angiography (iCAG) carries risks, including life-threatening arrhythmias (bradycardia, tachyarrhythmias, ventricular fibrillation), contrast-induced acute kidney injury, cerebrovascular accidents, coronary artery dissection, and bleeding at the vascular access site. The overall complication rate is approximately 1.8%, with 0.1% mortality [12].

In this regard, the search for non-invasive, reliable diagnostic alternatives for CAV has become increasingly relevant. Earlier non-invasive methods such as dobutamine stress echocardiography and myocardial scintigraphy have demonstrated very low sensitivity (approximately 7%) [13] and are not recommended as screening tools for CAV (Class IIb evidence) [2]. Single-photon emission computed tomography (SPECT) has shown some prognostic utility in diagnosing CAV, with sensitivity up to 84% and specificity 78% for detecting ≥50% stenosis compared to angiography but remains Class IIb evidence [2]. Positron emission tomography (PET) has not gained wide application in routine diagnosis of CAV and falls under Class IIb evidence [2, 14, 15].

Currently, the only non-invasive modality for coronary artery imaging that is widely available in routine clinical practice is multislice computed tomography coronary angiography (MSCT-CAG). This technique is widely used in the diagnosis of IHD, demonstrating a sensitivity ranging from 71% to 100% and a negative predictive value (NPV) of 93–100% when compared with iCAG.

In 2011, Paech et al. analyzed 28 studies involving 3,674 patients, evaluating the performance of 64-slice or higher CT coronary angiography as an alternative to iCAG. The meta-analysis showed a sensitivity of 98.2% and a specificity of 81.6%. The median positive predictive value (PPV), defined as the number of true stenotic segments detected divided by the total number of stenotic segments, was 90.5% (range: 76–100%), while the NPV, defined as the proportion of non-stenosed segments correctly identified, was 99.0% (range: 83–100%).

When evaluating entire coronary vessels, pooled sensitivity was 94.9%, specificity 89.5%, with a median PPV of 75.0% (range: 53–95%) and an NPV of 99.0% (range: 93–100%) [16].

Based on numerous studies [17–28], MSCT-CAG has been incorporated into European guidelines as an alternative to iCAG, with a validated class of evidence [24]. According to data from the CONFIRM registry, introduction of MSCT-CAG has resulted in approximately a 45% reduction in the use of iCAG procedures [20].

Most of the early studies evaluating MSCT-CAG as a non-invasive alternative for the diagnosis of CAV typically included relatively small patient samples (ranging from 10 to 60 individuals) and were performed using 16-slice MSCT scanners, with findings compared directly to iCAG. The results of these studies are presented in Table 3.

One of the earliest attempts to apply MSCT-CAG in the diagnosis of CAV was conducted by Romeo et al. in 2005, using a 16-slice MSCT scanner. In this study, the authors evaluated 53 patients and analyzed 450 coronary artery segments, based on a 10-segment coronary model. Three patients were excluded due to inability to hold their breath during scanning.

Average time after OHT was 7.6 ± 3.8 years (range: 1–14.5 years), the age range was from 7.6 to 75 years (mean age 48 ± 19 years), there were 40 men and 13 women. Baseline heart rate was 83 ± 13 bpm. Heart rate after 100 mg metoprolol (administered 1 hour before

Table 3

Initial studies on the implementation of MSCT-CAG in heart recipients

Studies evaluating 16-slice MSCT for coronary artery stenosis (*vs* invasive CAG)	G. Romeo et al., 2005
	S. Nunoda et al., 2010
	P. Carrascosa et al., 2009
	E. Usta et al., 2006
	P. Pichler et al., 2008
Studies comparing 16-slice MSCT with intravascular ultrasound (IVUS)	G. Sigurdson et al., 2006
Use of 64-slice MSCT in diagnosing CAV (*vs* invasive CAG)	S. Iyengar et al., 2006
	F. von Ziegler et al., 2009
	C. Kepka et al., 2012
	F. von Ziegler et al., 2012
	T.K. Mittal et al., 2013
Use of 64-slice MSCT in diagnosing CAV (*vs* invasive CAG and IVUS)	T. Schepis et al., 2009
	S.A. Gregory et al., 2006

MSCT-CAG) was 69.5 ± 11 bpm (range: 43–95 bpm). Contrast volume used was 70–90 mL. A complete segmental analysis was achieved in 50 out of 53 patients (88%), with diagnostic image quality in 432 of 450 segments (96%). Coronary calcifications were detected in 15 (30%) of 50 patients. Two cases had severe calcification, significantly limiting analysis; 13 patients had minor calcified plaques. Among 9 coronary stents in 7 patients, only 3 stents were adequately assessed, and 2 cases of restenosis were missed. In 44 (88%) of 50 patients, a complete assessment of the coronary tree was possible. In 22 patients without stenosis confirmed by iCAG, MSCT-CAG correctly showed no stenosis. For detection of significant stenosis (≥50%), MSCT-CAG showed a sensitivity of 83%, specificity of 95%, PPV of 71%, NPV of 95%, and accuracy of 93%. This early 16-slice MSCT study demonstrated good diagnostic accuracy in identifying both significant stenoses and normal coronary arteries, which is effective in screening for CAG [29].

As CT technology advanced, newer studies using higher-slice scanners reported improved sensitivity, specificity, diagnostic accuracy, NPV, PPV, and lower radiation exposure.

The study included 28 male patients (mean age: 53 ± 13 years) who underwent both iCAG and MSCT-CAG within a one-day interval during routine examination. The mean time after OHT was 7.7 ± 4.1 years (range: 4 months to 14 years). One hour before MSCT-CAG, patients received 50–100 mg of metoprolol orally to reduce and stabilize their HR. At the time of scanning, the average HR was 86 ± 13 beats per minute (range: 65–116 bpm). The average contrast volume used was 90 mL.

The coronary artery bed was evaluated according to the 15-segment AHA model. Out of 371 coronary segments analyzed, 302 (81.4%) were of diagnostic quality. Calcified plaques were identified in 6 out of 26 patients (23.1%) but did not affect image interpretation. Segment-level analysis demonstrated a sensitivity of 87.5%, specificity of 97.3%, overall accuracy of 97%, NPV of 99.7%, and PPV of 46.7% for detecting significant stenosis or vessel occlusion.

At the patient level, the sensitivity was 100%, specificity 81%, diagnostic accuracy 84.6%, NPV 100%, and PPV 55.6%. The high NPV at both segment and patient levels suggests that MSCT-CAG is a reliable non-invasive method for ruling out significant coronary stenosis in heart transplant (HT) recipients, comparable to its predictive value in patients with IHD [30].

In 2012, Franz von Ziegler et al. employed advanced dual-source CT scanners to evaluate significant coronary stenosis in HT recipients. The study included 51 patients (43 men; mean age 52.3 ± 13.6 years) who underwent both MSCT-CAG and iCAG within a 1–2 day interval during routine follow-up. The mean time after OHT was 6.9 ± 4.1 years (range: 2 weeks to 15 years). Serum creatinine was monitored 38.1 ± 2.4 hours after MSCT-CAG.

One hour before scanning, patients received 50–100 mg of oral metoprolol. The average HR prior to beta-blocker administration was 94 ± 14 bpm (range: 63–120 bpm), which decreased to 88 ± 14 bpm (range: 61–116 bpm) following medication. Of the 717 coronary segments analyzed, 714 (99.6%) were of optimal diagnostic quality. Calcified plaques were observed in 11 of 48 patients (22.9%) but did not compromise image interpretation.

On a segmental level, MSCT-CAG demonstrated a sensitivity of 100%, specificity of 98.9%, diagnostic accuracy of 98.9%, PPV of 50%, and NPV of 98.9%. At the patient level, sensitivity was 100%, specificity 86.0%, diagnostic accuracy 93.0%, PPV 33.3%, and NPV 100%. Transplant vasculopathy was diagnosed in 6.5% of recipients. No cases of contrast-induced nephropathy were reported.

The authors concluded that the high NPV (100%), as in the previous study, confirmed the reliability of MSCT-CAG for ruling out significant coronary stenoses in transplant recipients. The reduced rate of segment exclusion (0.4% in 2012 *vs* 18.6% in 2009) highlighted some improvements in imaging quality when using the latest scanners. Consequently, the study suggested that in the absence of significant stenosis on MSCT-CAG, annual CAG may not be necessary [31].

In 2013, Mittal et al. conducted the largest study to date, analyzing 138 HT recipients (2040 coronary segments). The cohort included men aged 22–78 years (53 ± 15 years) and women aged 20–80 years (47 ± 17 years), with a mean post-OHT follow-up of 12 ± 6.2 years (range: 1–25 years). In 109 patients, MSCT-CAG and CAG were performed within 24 hours, while in the remainder, MSCT-CAG was performed within a month. Patients with prior coronary stents and those with GFR <30 ml/min/1.73 m^2 were excluded. Before MSCT-CAG, sublingual nitrates were administered, but beta blockers were not used. mean HT was 82.7 ± 4 bpm. Two patients were excluded due to contrast extravasation.

The contrast volume was 70–90 ml for MSCT-CAG and 40–60 ml for iCAG. Creatinine levels were monitored 2–3 days post-procedure, with no cases of contrast-induced nephropathy reported. Average radiation dose for MSCT-CAG was 17.5 ± 6.9 mSv (range: 10–20 mSv), compared to 5–6 mSv for CAG. Coronary anatomy was assessed using the 15-segment ANA model, with ≥50% stenosis considered as significant.

Calcified plaques were detected in 82 patients; however, only 5 patients (6%) had significant stenosis. Despite relatively high heart rates, diagnostic image quality was obtained in 130 of 136 patients (96%) and in 1900 of 1948 segments (98%), although quality declined in distal segments.

For the detection of stenosis of any degree, MSCT-CAG showed a sensitivity of 98%, specificity of 78%, PPV of 77%, and NPV of 98%. For significant stenoses, sensitivity was 96%, specificity 93%, PPV 72%, and NPV 99%.

The authors confirmed that MSCT-CAG with 64-slice scanners is highly effective for diagnosing CAV, even without reducing HR. The technique demonstrated particularly high reliability in excluding stenosis, with strong concordance between MSCT-CAG and iCAG findings in patients without significant lesions [32]. They emphasized that reliance on the coronary calcium score alone is unreliable in this patient population.

Advances in new-generation CT scanners now enable not only the evaluation of the extent of lesion (severity and length of stenosis) but also characterization of atherosclerotic plaque morphology, including identification of "unstable" lesions, provided that image quality is adequate [33].

Thus, in 2018, Károlyi et al. (Hungary) examined 35 patients, 23 of whom were male (66%), aged 50–61 years (mean age 58). All patients underwent MSCT-CAG (256-slice; slice thickness 0.8 mm; increment 0.4 mm) at 1 and 2 years after OHT. Prior to imaging, they received nitroglycerin and ivabradine (7.5–15 mg) to reduce HR. In addition to standard analysis, quantitative assessment of coronary segments was performed, including lumen volume, total lesion volume, and total lesion burden (calculated as total vessel volume minus lumen volume, divided by total vessel volume). For detected plaques, the following components were evaluated: calcified lesion volume (≥350 HU), non-calcified high-attenuation volume (131–350 HU), non-calcified intermediate-attenuation volume (75–130 HU), and low-attenuation volume (≤75 HU).

CAV progression was defined as the development of any new coronary lesion (≥10% increase in lesion volume) or enlargement of a previously identified lesion. The findings demonstrated that within 2 years after OHT, CAV progression is characterized primarily by the development of non-calcified plaques, while calcified lesions remain unchanged. Moreover, quantitative MSCT-CAG detected a greater proportion of patients with CAV (≥10%) compared to standard qualitative analysis, which can be critical for identifying disease at an early stage. Segment analysis was feasible for vessels ≥2 mm in diameter; although early CAV may involve smaller branches, these are not typically candidates for revascularization.

Thus, quantitative analysis of MSCT images identifies more patients with progressive vasculopathy than qualitative assessment. Coronary wall thickening during the first 2 years after OHT is predominantly related to non-calcified plaque components and may represent early manifestation of CAV [34].

In 2020, Foldyna et al. conducted another study using quantitative analysis of coronary segments with a second-generation 128-slice MSCT scanner. A total of 50 patients (84% male; mean age 53.6 ± 11.9 years) were included, with and without previously verified vasculopathy, and a mean follow-up of 6.7 ± 4.7 years after OHT. The interval between CAG and MSCT-CAG was one day. The study focused on quantifying lumen volume, wall volume, and segment length. The following indices were calculated: volume-length ratio VLR (ratio of lumen volume to segment length; mm^3/mm), wall burden WB (wall volume ÷ (wall volume + lumen volume); %), and plaque composition (proportions of calcified, fibrous, fibrous-fatty, and soft plaques). Results showed that WB, VLR, and the proportion of fibrous tissue are reliable markers of vasculopathy and may assist in diagnosing CAV at an early stage, when lumen size is still preserved, which is an advantage over CAG.

An 18-segment coronary model (Society of Cardiovascular Computed Tomography, SCCT) was used for analysis, evaluating a total of 632 coronary segments. Image quality was scored according to SCCT recommendations (1 = excellent, 2 = good, 3 = fair, 4 = poor), and segments rated as poor were excluded. Coronary lesions were classified visually by degree and type of stenosis, following the guidelines of the American College of Cardiology/American Heart Association (ACC/AHA) Task Force.

By degree of stenosis:
- 0 degree: 0–24%
- 1 degree: 25–49%
- 2 degree: 50–74%
- 3 degree: 75–90% and above
- 4 degree: 100%.

By type of lesion:
- Type A: stenosis <10 mm in length; concentric lesion
- Type B: stenosis 10–20 mm in length; eccentric lesion
- Type C: stenosis >20 mm in length.

By CAV classification:
- No CAV: no stenosis or stenosis 1–24%
- Mild CAV: stenosis 25–49%, type A or B
- Moderate CAV: Stenosis ≥50%, type A–C.

Average radiation dose was 5.8 mSv. The study cohort consisted of 42 men (84%) and 8 women (16%), with a CAV prevalence of 38% (19 out of 50). Mean heart rate was 74.1 ± 8.5 beats per minute. MSCT-CAG provided diagnostic-quality images for 692 coronary segments, of which 632 (91.4%) were suitable for comparison with CAG data; 56 segments were excluded due to poor image quality. Among the 632 evaluable segments, 190 (30.1%) were proximal and 442 (69.9%) were distal.

Coronary wall analysis revealed that fibrous tissue accounted for 44.7%, fibro-fatty tissue for 18.6%, soft plaques for 8.5%, and calcified plaques for 1.0%. Distal segments were more frequently affected than proximal segments. The volume indexed by segment length (VLR)

was significantly higher in segments with CAV than in those without. Similarly, wall burden (WB; lumen/(wall + lumen) volume) was greater in segments with CAV compared to unaffected segments. The vascular wall in CAV was predominantly composed of fibrous and calcified tissue, whereas the proportions of fibro-fatty and soft plaques did not differ between CAV and non-CAV segments.

This study demonstrated that MSCT-CAG is highly effective in detecting severe stenoses ≥50%, with results correlating well with iCAG in HT recipients (NPV 98–100%, sensitivity 78%, specificity 75%). Moreover, MSCT-CAG can detect CAV at early stages through quantitative assessment of coronary wall plaque volume and composition, providing opportunities for timely adjustment of drug therapy [35].

In 2022, Ojha et al. assessed the diagnostic accuracy of dual-source MSCT (192-detector, 384-slice) for detecting CAV in comparison with iCAG. Thirty-eight patients (27 men) were included in the study, with a mean age of 33.66 ± 11.45 years and a mean post-OHT interval ranging from 10 to 226 months (median 23.5 months). One to two hours before MSCT-CAG, patients received 25–50 mg of oral metoprolol, followed by nitroglycerin immediately before scanning. The mean HR during imaging was 91 ± 13.86 beats per minute (range 74–146). Calcium score was measured at baseline.

The prevalence of CAV (grades 1–5 stenosis) was 44.7% (n = 17) according to MSCT-CAG and 39.5% (n = 15) by iCAG. Significant CAV lesions (grades 3–5) were detected in 21.1% (n = 8) by MSCT-CAG and in 15.8% (n = 6) by iCAG. Image quality was considered satisfactory in 557 out of 576 segments (96.7%). The mean radiation dose was 4.24 ± 2.15 mSv for MSCT-CAG and 4.8 ± 1.8 mSv for CAG, with an average contrast volume of 42 ml.

At the patient level, the detection of signs of vasculopathy of any degree had a sensitivity of 100%, specificity of 91.3%, PPV of 88.2%, NPV of 100%, and overall accuracy of 94.7%. For significant stenoses, sensitivity was 100%, specificity 94%, PPV 75%, NPV 100%, and accuracy 95%. Comparable results were obtained in segmental analysis (sensitivity 96%, specificity 97.6%, PPV 80%, NPV 99.6%).

This study demonstrated that dual-source MSCT, even at a relatively low radiation dose (4.24 ± 2.16 mSv), provides high diagnostic accuracy with excellent sensitivity, specificity, and NPV for detecting both early CAV and significant coronary stenoses when compared with iCAG [36].

In 2021, Nous et al. reported their experience implementing MSCT-CAG as a screening tool for CAV at the University Medical Center Rotterdam (the Netherlands). Between February 2018 and May 2019, 129 patients aged 43–64 years (mean 55), 8–17 years post-OHT (mean 11 years), were included. Men accounted for 65% of the cohort, and 13% had a history of percutaneous coronary intervention (PCI). At this center, elective CAG was routinely performed in the first and fourth years after OHT or when ischemia was suspected, while annual cardiac MRI and SPECT were used in the remaining years.

Before MSCT-CAG, all patients received nitroglycerin; in cases with an HR ≥70 bpm, intravenous metoprolol (5.0–7.5 mg) was administered. On average, HR decreased by 15% to 75 ± 11 bpm, with no conduction disturbances or hypotension observed. MSCT scanners with dual sources of the 2nd generation (29%, n = 37) and 3rd generation (71%, n = 92) were used, applying a prospective ECG-triggered mode. Images were reconstructed with a slice thickness of 0.6 mm and an increment of 0.3 mm. Calcium score was obtained prior to contrast injection.

In most coronary segments with significant CAV, non-calcified plaques predominated (64%). Diagnostic image quality was achieved in 118 of 129 patients (92%), with a mean radiation dose of 2.1 mSv (range 1.6–2.8). Significant stenoses were identified in 19 patients (15%), of whom 15 were newly diagnosed and 4 had been previously recognized. Three patients were not referred for PCI due to chronic total occlusions or stenotic lesions in small-caliber branches (<2 mm).

At 90-day follow-up, 9 of 19 patients with significant stenoses (47%) underwent further evaluation with SPECT, MRI, and CAG. Additional investigations were not performed in 10 patients (53%) because lesions had already been deemed unpromising on prior iCAG. In 8 of 9 patients (89%), hemodynamically significant stenoses were confirmed: 4 underwent stenting, 2 underwent stenting combined with modification of drug therapy, and 1 received drug therapy adjustment only (e.g., statin dose increase or switch from mycophenolate mofetil to mTOR inhibitors).

Four patients with hemodynamically insignificant stenoses on MSCT-CAG underwent iCAG, after which two underwent PCI and seven required therapy adjustments. Within 90 days to 1 year, one patient with ventricular tachycardia underwent CAG and PCI despite no significant stenosis being detected by MSCT-CAG. Beyond 1 year, three patients developed major adverse cardiovascular events (MACE). These findings demonstrate that MSCT can be effectively integrated into clinical practice, providing high-quality imaging at a low radiation dose, reducing the need for invasive procedures, and enabling early detection of vasculopathy to guide timely therapy adjustments [37].

In a study by Szymon Pawlak et al., it was acknowledged that 64-slice MSCT may underestimate the progression of CAV or previously stented segments. Therefore, iCAG was performed in all 209 patients with known vasculopathy. For MSCT-CAG evaluation, 107 patients without graft dysfunction and without hemodynamically significant stenoses on CAG performed 2 years earlier

were selected (26 women, mean age 50 ± 17 years, mean time after OHT 7 years, range 4–11.5 years). All patients received sublingual nitrates, and those with an HR >90 bpm prior to MSCT-CAG additionally received 5 mg ivabradine. Control CAG was performed only when MSCT-CAG suggested significant stenosis. As a result, CAG was not performed in 98 patients without evidence of stenotic lesions. In 8 of 9 patients, stenoses detected by MSCT-CAG were confirmed by CAG, and PCI was performed in 6 of them. As in previous studies, calcium index values were not informative for diagnosing CAV. No cases of contrast-induced nephropathy were observed [38].

A recent meta-analysis of 13 prospective studies on MSCT-CAG after OHT provided robust evidence supporting its implementation in cardiac transplant recipients. The analysis demonstrated a weighted mean sensitivity of 94%, specificity of 92%, negative predictive value of 99%, and positive predictive value of 67% for detecting stenoses >50% compared with invasive angiography. The incorporation of quantitative plaque analysis was shown to further enhance sensitivity for detecting cardiac transplant vasculopathy. In total, CT angiogram data from 615 patients were prospectively evaluated [39]. In most studies, the coronary tree was segmented according to the 16-segment American Heart Association classification (see Figure).

A total of 9,481 coronary segments were analyzed across the studies, with the time after OHT ranging from 3 to 8 years. Average patient age ranged from 40 to 58 years, and the study populations were predominantly male (75–100%). Most investigations employed first-generation MSCT scanners with single- or dual-source technology (16- or 64-slice).

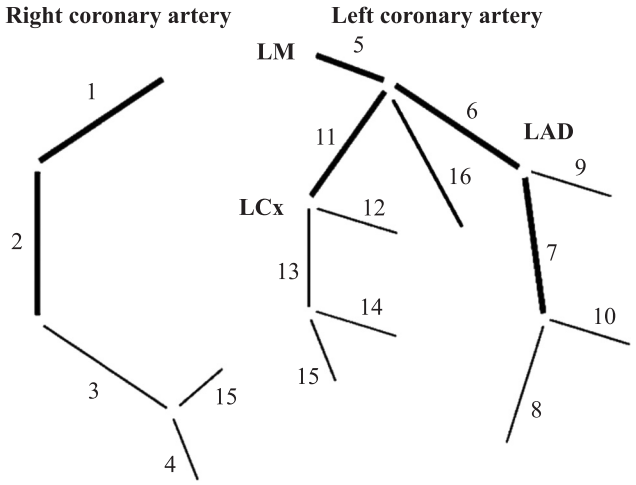

Fig. Schematic representation of the 16-segment coronary artery classification based on the American Heart Association (AHA) guidelines. Right coronary artery (RCA): segment 1 – proximal RCA; 2 – mid RCA; 3 – distal RCA; 4 – posterior descending branch. Left coronary artery (LCA): 5 – left main (LM); 6 – proximal left anterior descending (LAD); 7 – mid LAD; 8 – distal LAD; 9 – first diagonal; 10 – second diagonal; 11 – proximal left circumflex (LCx); 12 – first obtuse marginal; 13 – distal LCx; 14 – posterolateral artery; 15 – posterior descending artery; 16 – ramus intermedius [35]

Contraindications for MSCT after OHT were:
- allergy to iodine [49];
- significant decrease in glomerular filtration rate (GFR <30 ml/min/1.73 m^2 or serum creatinine >1.4 mg/dl) [49];
- pregnancy;
- claustrophobia or inability to hold your breath.

Limitations of MSCT after OHT were:
- severe general condition of the patient;
- high body mass index;
- arrhythmias or persistent tachycardia;
- pronounced coronary artery calcification or presence of stents in the coronary arteries [39, 49].

In several studies, authors reported a decline in coronary artery image quality due to the high HR characteristic of the denervated heart, which led to exclusion of a substantial number of coronary segments from analysis – particularly when older-generation MSCT scanners were used. To address this issue, beta-blockers were administered in some studies [29, 30, 31, 40, 41, 42], either as metoprolol 50–100 mg orally or 10–12 mg intravenously. Although the target HR was not always achieved, a reduction of 10–15 bpm was obtained, with mean pre-scan HR ranging from 69 to 90 bpm (average 84 bpm). An HR >85 bpm was associated with a significant decrease in image quality.

Sigurdsson and Schepis did not use β-blockers; nevertheless, despite high HR, they reported good to excellent image quality [42, 43].

Data from Nous F.M. et al. showed that beta-blockers reduce HR by an average of 15% [37]. Studies have also evaluated ivabradine, which has shown a safe and effective reduction in HR in OHT recipients with sinus rhythm [34, 45]. An equally important consideration in this patient population is monitoring renal function, as the risk of contrast-induced nephropathy (CIN) is increased due to pre-existing renal impairment, most often related to long-term use of calcineurin inhibitors. For this reason, many studies excluded patients with serum creatinine levels above 1.4 mg/dl. CIN was defined as a ≥25% increase in creatinine or an absolute rise of 44 μmol/L. Post-contrast creatinine testing was generally performed one day after MSCT. Although the mean contrast volume used in MSCT (60–115 ml) was slightly higher than that in CAG, none of the studies reported cases of CIN. However, this observation period may have been insufficient, since serum creatinine typically peaks 72 hours after contrast administration.

Several studies have focused on radiation exposure during MSCT-CAG and the development of protocols to minimize it. Reported radiation doses ranged from 3 to 18 mSv, approximately twice the average dose of iCAG [29, 40, 43]. With 16-slice MSCT, the mean radiation

dose was 14.7 ± 2.2 mSv, whereas the introduction of dual-source MSCT reduced the dose to 4.5 ± 1.2 mSv [31], which is comparable to iCAG (5.6 ± 3.6 mSv). In most studies, retrospective ECG-gated image reconstruction was used due to higher heart rates, but this method resulted in higher doses (10.2–17.5 mSv).

Given the serious concerns about cumulative radiation exposure, significant efforts have been directed toward dose reduction. Heart transplant recipients are exposed to approximately 3.5 times more radiation than the general population, with an average cumulative dose of 84 mSv over a 10-year follow-up period [46, 47]. This substantially increases cancer risk, particularly in women and younger patients. Bastarrika G. et al. later achieved a reduction in dose to 4.5 mSv while preserving diagnostic image quality by using prospective ECG-triggered MSCT with systolic phase acquisition [48].

Protocols for radiation dose reduction and optimized image reconstruction have also been developed. The use of ECG-controlled tube current modulation – where full tube current is delivered between 30% and 80% of the cardiac cycle – can lower the effective dose by approximately 40% and reduce the lifetime risk of cancer compared with standard retrospective scanning, particularly in women and younger patients, while preserving diagnostic image quality.

In 2014, Beitzke D. et al. demonstrated that with 128-row dual-source MSCT, radiation dose can be significantly reduced through the use of prospective scanning combined with automatic tube voltage selection, without compromising diagnostic quality (slice thickness 0.6 mm). Even in patients with elevated heart rates, this approach achieved a dose reduction of up to 50% [46].

Three scanning protocols were compared:

1) Retrospective scanning mode – tube voltage set at 120 kVp on both tubes, tube current at 320 mA. Adaptive tube current modulation was applied, with the scanner selecting the optimal ripple window depending on HR. This approach was associated with the highest radiation doses.
2) Prospective sequential scanning with ECG triggering – tube voltage set at 120 kVp on both tubes, tube current at 320 mA. The main acquisition window was set at 30–70% of the RR interval. This technique showed no significant advantage over retrospective scanning.
3) Prospective sequential scanning with a narrow systolic window – main acquisition window set at 35–45% of the RR interval, with automatic tube voltage adjustment enabled.

In 2017, Bartykowszki A. et al., using a 256-slice MSCT (tube voltage 100–129 kV, tube current 100 mA, gantry rotation time 270 ms) with prospective ECG triggering, achieved an average effective radiation dose as low as 3.7 mSv [48].

The latest third-generation 384-slice scanners with dual sources and 192 detectors, when combined with advanced radiation reduction and image reconstruction protocols, provide faster scanning and broader coverage at lower radiation doses, while offering improved spatial and temporal resolution that more closely aligns with CAG. Moreover, despite relatively high heart rates, the proportion of uninterpretable segments remained low (3.3%), in contrast to earlier studies [35, 41, 43].

Thus, adoption of these advanced radiation reduction and image reconstruction protocols can reduce radiation exposure while maintaining diagnostic image quality in heart transplant recipients.

CONCLUSION

Although iCAG remains the gold standard for diagnosing CAV, it requires hospitalization, carries procedural risks, and may cause patient discomfort. MSCT-CAG offers a non-invasive, safer alternative with high sensitivity (86–89%) and specificity (89–99%) for evaluating the coronary arteries after heart transplantation. With the growing number of transplant recipients, annual non-invasive outpatient screening using MSCT-CAG could facilitate early detection and monitoring of CAV progression, help stratify patients for hospitalization, and reduce both healthcare costs and hospital burden.

Heart recipients with previously verified stenoses or coronary stents are not suitable for 64-slice MSCT, and when MSCT-CAG data are inconclusive, iCAG is still recommended. Conversely, if MSCT shows no evidence of stenosis, iCAG may not be required. Special attention should be given to minimizing contrast-induced nephropathy in patients after OHT and applying optimized radiation reduction protocols in combination with a reduction in the frequency of these procedures.

While MSCT-CAG heart recipients shows high sensitivity, its lower specificity means that CAV may occasionally be underestimated. Therefore, further research is needed to refine its diagnostic accuracy.

The authors declare no conflict of interest.

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The article was submitted to the journal on 17.03.2025