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Reindolf Anokye
Edith Cowan University

Jack Dalla Via
Edith Cowan University

James Dimmock

Ben Jackson

Carl Schultz

See next page for additional authors

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REVIEW ARTICLE

Impact of Cardiovascular Imaging Results on Medication Use and Adherence: A Systematic Review and Meta-Analysis

Reindolf Anokye, MSc, PhD,[1] Jack Dalla Via, PhD,[2] James Dimmock, PhD,[3] Ben Jackson, PhD,[4,5] Carl Schultz, MD, PhD,[6,7] Mie Schæffer, BSc Med,[8] Joanne M. Dickson, PhD,[2,9,10] Lauren C. Blekkenhorst, PhD,[2,7] Mandy Stanley, PhD,[1] Jonathan M. Hodgson, PhD,[2,7] Joshua R. Lewis, PhD[2,7,11]

Introduction: Cardiovascular imaging results offer valuable information that can guide health decisions, but their impact on medication use and adherence is unclear. This systematic review and meta-analysis aimed to determine the downstream impact of cardiovascular imaging results on medication use and adherence.

Methods: Searches were conducted across databases, including MEDLINE, PsychINFO, EMBASE, and relevant references up to 2024. Data were extracted from studies comparing outcomes for individuals with diseased versus normal arteries and trials comparing outcomes for individuals who were provided imaging results versus those with no access to imaging results and analysed in 2023 and 2024. Pooled odds ratios (ORs) for outcomes were calculated.

Results: The analysis included 29 studies with 24 contributing data points. Initiation (OR:2.77;95% CI:1.82−4.20) and continuation (OR:2.06;95% CI:1.28−3.30) of lipid-lowering medications (LLMs), antihypertensives (OR:2.02;95% CI:1.76−2.33), and antiplatelets (OR:2.47;95% CI:1.68−3.64) were significantly higher in individuals with diseased arteries. The proportion of individuals on LLM increased by 2.7-fold in those with diseased arteries and 1.5-fold in those with normal arteries post-screening. The proportion on LLM increased by 4.2 times in the imaging group and 2.2 times in the "no imaging group" post-screening. There was a significant increase in LLM initiation (OR:2.37;95% CI: 1.17−4.79) in the imaging group, but medication continuation did not significantly differ between the imaging and "no imaging group".

Discussion: Cardiovascular imaging results can prompt initiation of medications, particularly lipid-lowering medications, reflecting a proactive response to identified risk factors. However, evidence regarding medication continuation is mixed, and further research is required.

From the [1]School of Medical and Health Sciences, Edith Cowan University, Perth, Western Australia, Australia; [2]Nutrition & Health Innovation Research Institute, School of Medical and Health Sciences, Edith Cowan University, Royal Perth Hospital Research Foundation, Perth, Western Australia, Australia; [3]Department of Psychology, College of Healthcare Sciences, James Cook University, Townsville, Queensland, Australia; [4]Telethon Kids Institute, Perth, Western Australia, Australia; [5]School of Human Sciences (Exercise and Sport Science), The University of Western Australia, Perth, Western Australia, Australia; [6]Cardiology Department, Royal Perth Hospital, Perth, Western Australia, Australia; [7]Medical School, The University of Western Australia, Perth, Western Australia, Australia; [8]Department of Cardiology, Odense University Hospital, Denmark; [9]School of Arts and Humanities (Psychology Discipline), Edith Cowan University, Joondalup, Western Australia, Australia; [10]Department of Psychological Science, University of Liverpool, UK; and [11]Centre for Kidney Research, Children's Hospital at Westmead, School of Public Health, Sydney Medical School, The University of Sydney, Sydney, New South Wales, Australia

Address reprint requests to: Reindolf Anokye, School of Medical and Health Sciences, Edith Cowan University, 270 Joondalup Drive, Joondalup, WA, 6027, Australia. E-mail: reindolfanokye@yahoo.com.

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INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of premature mortality worldwide.[1] CVD often has a long asymptomatic phase and may not be recognized until it triggers clinical events such as heart attacks or strokes.[2] Asymptomatic adults at increased risk of cardiovascular events are often screened to identify any abnormalities that may trigger such events.[3] Early identification of asymptomatic CVD can inform preventive treatment recommendations.[4]

Traditional CVD risk assessment strategies are widely used to guide preventive interventions.[5] However, due to suboptimal adherence rates in CVD prevention, refinement and personalized approaches to enhance patient outcomes are being widely considered.[6] Non-invasive imaging modalities, such as computed tomography (CT) of the coronary arteries for calculating coronary artery calcification (CAC) and carotid ultrasound (CU) for identifying carotid plaques and assessing intimal medial thickness, are common non-traditional risk assessment approaches used to visualize asymptomatic CVD.[7–9] Several studies have examined the potential impact of non-invasive imaging findings on medication use and adherence, with initiation and continuation of cardiovascular medications being the most commonly studied aspects.[7–10]

Medication initiation is the start of the medication adherence continuum,[11] and an important part of primary CVD prevention as a beginning stage of risk factor control.[12] Continuation of cardiovascular medications can prevent disease progression and reduce the risk of cardiovascular events and complications. [13] Initiation and continuation of cardiovascular medications following imaging is a collaborative decision-making process involving (a) prescribers' tendencies to adjust or initiate treatment based on imaging results, and (b) patients' behaviors or responses, including filling prescriptions and starting and adhering to therapy.[3,14] Patients must be active partners with healthcare professionals in their care. Good communication between patients and healthcare professionals can improve clinical practice and CVD prevention. [15]

Imaging interventions can facilitate informed decision-making and patient engagement through risk communication, and discussions about the potential benefits of treatment or behavior change.[16] Visual images are effective in conveying complex health information.[17] Images can evoke emotional responses,[18] increase message salience and influence individual perceptions and motivation towards adopting recommended behaviors.[16,19] However, the role of cardiovascular imaging in risk assessment for primary prevention, particularly regarding medication use and adherence, is not well established in existing literature and clinical guidelines.[7-9] Previous reviews have focused on particular imaging modalities or have included very few studies.[7,9,10] As such, this systematic review and meta-analysis aimed to determine the downstream impact of cardiovascular imaging results (from CT imaging and carotid ultrasound) on medication use and adherence.

METHODS

This systematic review and meta-analysis was developed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.[20] The study was registered (CRD42022317243) in the International Prospective Register of Systematic Reviews (PROSPERO).

Relevant studies were identified by searching electronic databases, including MEDLINE, Psych INFO, Cumulative Index to Nursing and Allied Health Literature (CINAHL) Plus (via EBSCOhost), and EMBASE, from 1975 to 2022, without language restrictions. MeSH subject headings, including coronary artery calcium score, carotid arteries, carotid atherosclerosis, behaviour, and medication adherence, were combined with free terms (Appendix Table 1). The reference lists of included studies were screened to identify other potential papers of interest. Two investigators (RA and JDV) independently screened all articles for eligibility in three stages: (1) title, (2) abstract, and (3) full-text, extracted data, and assessed the risk of bias. Another investigator (JRL) was consulted when an agreement could not be reached. Authors were contacted (up to three times over 6 weeks if no responses were received) for studies when there was ambiguity around the study eligibility or data to be extracted. If no response was received after the third contact, the studies were excluded, or no data were extracted for the synthesis.

Studies that recruited asymptomatic adult participants (18 years and above) without a known history of coronary heart disease, myocardial infarction, stroke, diagnosed CVD, macrovascular disease, peripheral revascularization, or peripheral artery disease were included. Participants were screened using non-invasive imaging modalities (e.g., carotid ultrasound or computed tomography [CT]), were informed about the imaging results after screening, and medications to prevent cardiovascular complications were prescribed or recommended. Randomized trials, pre-post prospective studies, and retrospective quasi-experimental studies that reported the outcomes of interest were considered in this review.

The outcomes and exposures in the study were categorized into several groups. Participants were divided into imaging and no imaging groups. Those in the imaging group received imaging results regardless of their status (normal or abnormal), while the no imaging group did not have access to imaging results. Also, participants were categorized into disease and no disease groups based on their imaging results. The disease group received feedback indicating the presence of plaque build-up, calcification, increased carotid intima-media thickness (CIMT), or diseased arteries, while the no disease group was informed of normal arteries or received information that they have no arterial disease. Furthermore, participants were stratified into low-, moderate-, and high-risk groups based on their imaging results, with different risk levels determined by coronary artery calcium scores and severity of plaque build-up or calcification. One outcome was initiation, which referred to the commencement of new medications, including when an individual received a prescription, obtained the medication from a pharmacy or took the first dose of a prescribed medication. Other outcomes included the proportion of individuals on medication before and after screening, and medication continuation. Medication continuation referred to the ongoing use of prescribed medications over a specified period following the initial prescription or persistent medication use (See Appendix A for further description). The characteristics of the included studies and data related to exposures and outcomes were extracted (Appendix A). ROBINS-I tool was used for assessing the risk of bias in nonrandomized intervention studies and to grade the quality of evidence.[21] Randomized trials were assessed for risk of bias using Version 2 of the Cochrane risk-of-bias tool for randomized trials (RoB 2).[22] Two authors (RA and JDV) appraised all eligible studies and extracted the data.

An *a priori* list of potential study variables that may affect between-study heterogeneity included study age, sex (proportion of females), imaging (coronary or carotid artery), ethnicity (proportion White), education (proportion college or higher), person who communicated the results (healthcare professionals or study investigators), mode of delivery of result (in-person, written, or telephone), type of results (disease present vs. absent, images only, not specified, scores, both images and scores), follow-up time, and country of study. Meta-regression was performed to evaluate the effect of the above variables on between-study heterogeneity.

Pooled odds ratios (OR) and 95% confidence intervals (CI) were used to measure effect size. For each outcome of interest, the DerSimonian and Laird random-effects model was used to pool the ORs across studies. Heterogeneity was examined using I^2 statistics[23], where $p>0.05$, Q statistics, and I^2 value <50% indicated low statistical heterogeneity, I^2 value 50% to 75% indicated moderate statistical heterogeneity, and I^2 value 75% to 100% indicated high statistical heterogeneity. Statistical significance was considered at $p<0.05$ and the likelihood of publication bias was evaluated by visual inspection of funnel plot and Egger's regression test.[24] Data analysis was conducted in 2023 and 2024.

RESULTS

The search identified 3,989 articles. Of these, 29 studies (9 randomized and 20 non-randomized studies) met the eligibility criteria (Figure 1). The details of all included studies are described in Appendix Table 2. After full-text screening, studies reporting outcomes related to medication initiation, the proportion of individuals on cardiovascular medications pre-post screening, and medication continuation were included. A total of 24 studies reporting at least one outcome of interest were included in the meta-analysis. Some authors presented study results in formats from which extracted data could not be obtained.[14,25–29] The corresponding authors were contacted to obtain additional unpublished data. Two authors [26,28] provided data related to the proportion of individuals on antiplatelets, LLMs, antihypertensives, and initiation of cardiovascular medication. For the remaining studies, the authors contacted did not respond or could not provide original data (owing to the length of time since the papers were published or because of the aims of the study); therefore, they were excluded from the analysis. [14,25,27,29]

The 29 included studies were published between 1996 and 2023 (Appendix Table 2). The randomized trials included in the meta-analysis (n=9) collectively enrolled 11,169 participants. Several dimensions of medication adherence, including medication initiation (n=17), continuation (n=6), and discontinuation (n=2) were reported. However, meta-analysis was only possible for outcomes related to medication initiation, the proportion of individuals on medication before and after screening, and medication continuation. The mean age of the participants across studies ranged from 43 to 70 years, and most studies were conducted in the United States of America (USA) (n=21). The screening results were communicated by research investigators (n=12), healthcare providers (n=6), in person (n=10), via letter/mail (n=4), or telephone (n=1). Outcomes related to initiation, continuation, and proportion of individuals on medication (pre-post screening) were measured using different instruments and approaches, including self-report questionnaire designed for the study, review of patient medical records, review of clinic containers for

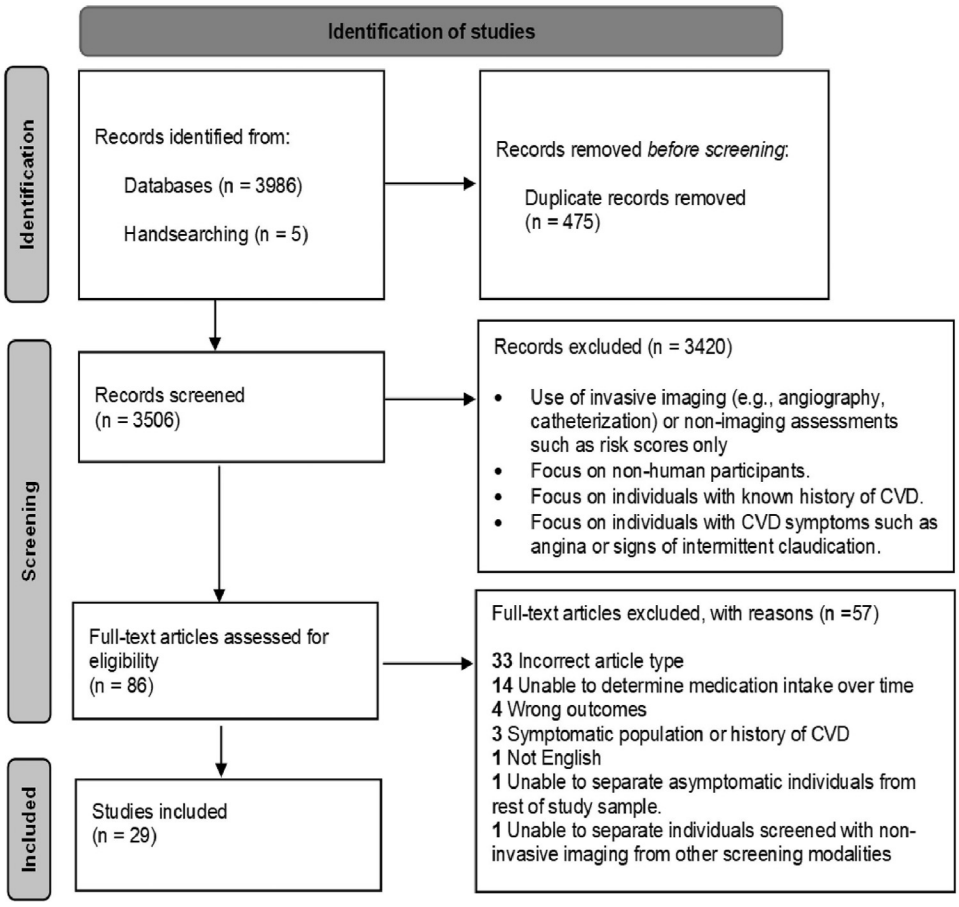

Figure 1. PRISMA flowchart.

all medications used, pill count, and Morisky medication adherence questionnaire (Appendix Table 2).

Twelve studies (n=13,051 participants) reported outcomes on initiation of LLM, seven studies (n=7,571 participants) reported outcomes on initiation of antihypertensives, and eight studies (n=8,910 participants) reported outcomes on initiation of antiplatelets in individuals with diseased *vs.* those with normal arteries (Table 1). The pooled estimates of the odds of LLM, antihypertensives, and antiplatelet medication initiation were significantly higher in individuals with diseased arteries (Figure 2).

Five studies (n=9,913 participants) reported outcomes on the initiation of LLM, three studies (n= 8,055 participants) reported outcomes on the initiation of antihypertensives, and four studies (n= 9,019 participants) reported outcomes on the initiation of antiplatelets in participants within the low-, moderate-, or high-risk categories. Compared with the low-risk groups, medication initiation rates were significantly higher in individuals classified as moderate-risk and high-risk. LLM initiation was 3.01 times higher in the moderate-risk group and 6.80 times higher in the high-risk group. The odds of antihypertensive initiation were 2.66 times higher in the high-risk group. Antiplatelet medication initiation was 2.34 times higher in the moderate-risk group and 3.59 times higher in the high-risk group (Table 1).

Extractable data were available for trials that reported outcomes on LLM initiation (6 studies, n=5,215 participants), antihypertensive initiation (5 studies, n=4,490 participants), and antiplatelet medication initiation (4 studies, n=2,705 participants) in the imaging vs. no imaging groups (Table 1). Overall, the pooled estimates of the odds of LLM initiation were significantly higher in the imaging group than in the no-imaging group. However, no significant difference was observed for antihypertensive and antiplatelet medication initiation (Figure 3).

Extractable data were available for four studies that reported outcomes related to medication continuation in individuals with no disease vs. disease. Reported outcomes included continuation of LLM (n= 2,150 participants) and antiplatelet therapy (n=1,802 participants) after imaging. The pooled estimates of the odds of LLM

Table 1. Pooled Estimates for Odds of medication initiation, Proportion on Medication (Prepost), and Continuation (Unadjusted)

Exposures/Outcomes	No. of studies (No. of events/group total)	Odds Ratio (95% CI)	I^2
Medication Initiation			
No disease vs. disease			
Lipid-lowering medication			
No disease (Absence)	12 (666 / 6,979)	1 (Ref.)	
Any disease (Presence)	12 (1,379 / 6,072)	2.77 (1.82–4.20)	89%
Antihypertensive medication			
No disease (Absence)	7 (382 / 4,121)	1 (Ref.)	
Any disease (Presence)	7 (601 / 3,450)	2.02 (1.76–2.33)	0%
Antiplatelet medication			
No disease (Absence)	8 (538 / 4,348)	1 (Ref.)	
Any disease (Presence)	8 (1,309 / 4,562)	2.47 (1.68–3.64)	79%
Low risk vs. moderate risk, low risk vs. high risk			
Lipid-lowering medication			
Low	5 (365 / 4,604)	1 (Ref.)	
Moderate	5 (456 / 2,702)	3.01 (1.66–5.45)	89%
High	5 (473 / 2,607)	6.80 (2.67–17.30)	94%
Antihypertensives			
Low	3 (400 / 3,992)	1 (Ref.)	
Moderate	3 (354 / 2,285)	1.62 (1.39–1.89)	0%
High	3 (365 / 1,778)	2.66 (2.27–3.12)	0%
Antiplatelets			
Low	4 (576 / 4,327)	1 (Ref.)	
Moderate	4 (715 / 2,692)	2.34 (1.36–4.04)	88%
High	4 (629 / 2,000)	3.59 (2.35–5.49)	70%
No imaging vs imaging			
Lipid-lowering medication			
No imaging	6 (239 / 2,365)	1 (Ref.)	
Imaging	6 (644 / 2,850)	2.37 (1.17–4.79)	90%
Antihypertensives			
No imaging	5 (211 / 2,036)	1 (Ref.)	
Imaging	5 (344 / 2,454)	1.10 (0.75–1.63)	51%
Antiplatelets			
No imaging	4 (54 / 1,044)	1 (Ref.)	
Imaging	4 (108 / 1,661)	1.09 (0.77–1.53)	0%
Medication continuation			
No disease vs disease			
Lipid-lowering medication			
No disease (Absence)	4 (375 / 544)	1 (Ref.)	
Any disease (Presence)	4 (1206 / 1,606)	2.06 (1.28–3.30)	86%
Antiplatelets			
No disease (Absence)	3 (411 / 632)	1 (Ref.)	
Any disease (Presence)	3 (768 / 1,170)	1.28 (0.75–2.18)	75%
No imaging vs imaging			
Lipid-lowering medication			
No imaging	3 (423 / 475)	1 (Ref.)	
Imaging	3 (607 / 678)	1.14 (0.75–1.74)	9%

(*continued on next page*)

Table 1. Pooled Estimates for Odds of medication initiation, Proportion on Medication (Prepost), and Continuation (Unadjusted) (*continued*)

Exposures/Outcomes	No. of studies (No. of events/group total)	Odds Ratio (95% CI)	I^2
Proportion on medication (pre-post)			
No disease / Disease (pre-post imaging)			
Lipid-lowering medication			
No disease (Absence)	6 (Pre - 334 / 3,059)	1 (Ref.)	
	6 (Post - 554 / 3,059)	1.58 (0.98–2.56)	88%
Any disease (Presence)	6 (Pre - 552 / 2,154)	1 (Ref.)	
	6 (Post - 1071 / 2,154)	2.71 (1.60–4.61)	92%
Antihypertensives			
No disease (Absence)	5 (Pre - 412 / 1,787)	1 (Ref.)	
	5 (Post - 514 / 1,787)	1.26 (0.97–1.65)	59%
Any disease (Presence)	5 (Pre - 603 / 1,789)	1 (Ref.)	
	5 (Post - 745 / 1,789)	1.32 (1.01–1.74)	68%
Antiplatelets			
No disease (Absence)	6 (Pre - 332 / 3,059)	1 (Ref.)	
	6 (Post - 506 / 3,059)	1.16 (0.60–2.24)	93%
Any disease (Presence)	6 (Pre - 394 / 2,154)	1 (Ref.)	
	6 (Post - 588 / 2,154)	1.67 (1.02–2.74)	89%
Low-risk, moderate-risk, high-risk (pre-post imaging)			
Lipid-lowering medication			
Low	3 (Pre - 169 / 1,280)	1 (Ref.)	
	3 (Post - 251 / 1,280)	1.64 (1.29–2.08)	7%
Moderate	3 (Pre - 282 / 1,073)	1 (Ref.)	
	3 (Post - 436 / 1,073)	1.80 (1.05–3.10)	84%
High	3 (Pre - 67 /177)	1 (Ref.)	
	3 (Post - 122 / 177)	4.13 (2.49–6.84)	6%
Antihypertensives			
Low	3 (Pre - 285 / 1,281)	1 (Ref.)	
	3 (Post - 382 / 1,281)	1.40 (0.98–2.01)	64%
Moderate	3 (Pre - 333 / 1,075)	1 (Ref.)	
	3 (Post - 434 / 1,075)	1.43 (1.04–1.97)	59%
High	3 (Pre - 76 / 177)	1 (Ref.)	
	3 (Post - 101 / 177)	1.62 (0.78–3.34)	56%
Antiplatelets			
Low	3 (Pre - 99 / 1,280)	1 (Ref.)	
	3 (Post - 76 / 1,280)	0.73 (0.53–1.01)	0%
Moderate	3 (Pre - 121 / 1,073)	1 (Ref.)	
	3 (Post - 132 / 1,073)	1.12 (0.84–1.49)	0%
High	3 (Pre - 48 / 177)	1 (Ref.)	
	3 (Post - 55 / 177)	1.26 (0.75–2.11)	0%
No imaging / Imaging (pre-post screening)			
Lipid-lowering medication			
No imaging	4 (Pre - 371 / 2,666)	1 (Ref.)	
	4 (Post - 476 / 2,333)	2.23 (1.12–4.45)	90%
Imaging	4 (Pre - 553 / 3,309)	1 (Ref.)	
	4 (Post - 896 / 3,106)	4.20 (1.68–10.51)	97%
Antihypertensives			
No imaging	3 (Pre - 746 / 2,541)	1 (Ref.)	
	3 (Post - 744 / 2,267)	1.35 (0.94–1.94)	81%
Imaging	3 (Pre - 990 / 3,208)	1 (Ref.)	
	3 (Post - 1173 / 3,030)	1.99 (1.05–3.76)	96%

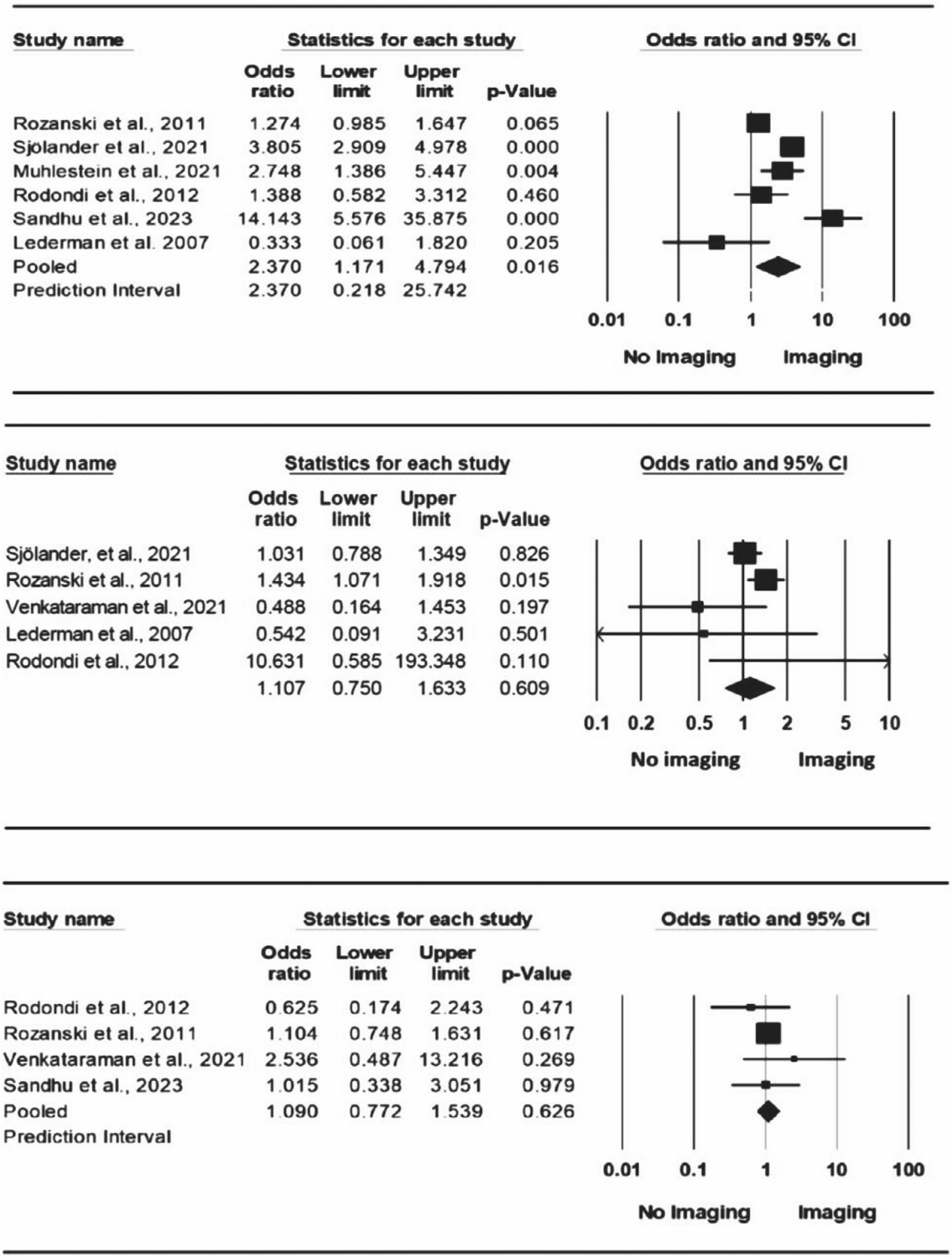

Figure 2. Forest plots and pooled odds ratios for initiation of LLM (top), Antiplatelet (middle), and Antihypertensives (bottom) in disease and no-disease groups.

continuation were significantly higher in individuals with diseased arteries; however, the odds of antiplatelet continuation were not significantly higher in this group (Table 1).

Extractable data were available for three trials (n=1,153 participants) that reported outcomes on LLM continuation in two groups (no imaging vs. imaging) (Table 1). Overall, the pooled estimates of the odds of

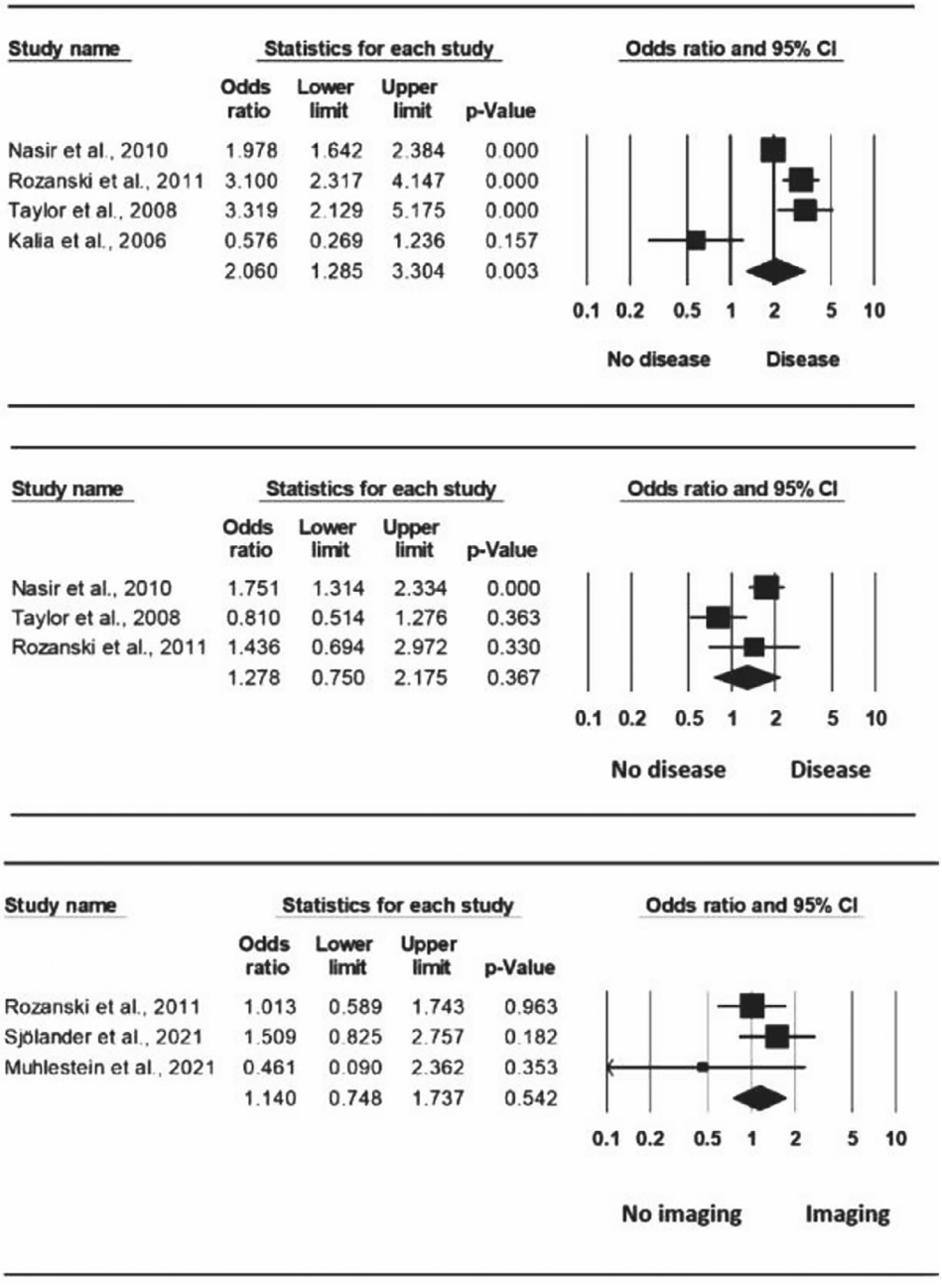

Figure 3. Forest plots and pool odds ratios for initiation of LLM (top), initiation of Antihypertensives (middle), and initiation of Antiplatelet (bottom) in the imaging vs. no imaging groups.

LLM continuation were not significantly higher in the imaging group (Appendix Figure 1).

Extractable data were available for studies that reported outcomes for proportion of individuals on LLM (6 studies, *n*=5,213 participants), antihypertensive medication (5 studies, *n*=3,576 participants), and antiplatelets (6 studies, *n*=5,213 participants) before and after imaging. These findings indicate that the number or proportion of individuals on LLM increased following cardiovascular imaging, with higher odds observed among those with diseased arteries. The odds increased by 1.58 times in the no-disease group and by 2.71 times in the disease group post-imaging compared to pre-imaging (Table 1).

Extractable data were available for studies that reported outcomes for proportion of individuals on

LLM (3 studies, *n*= 2,530 participants), antihypertensives (three studies, *n*=2,533 participants), and antiplatelets (3 studies, *n*=2,530 participants) before and after imaging in the low-, moderate-, and high-risk groups. The proportion of individuals on LLM before and after imaging varied across different risk levels. The odds increased by 1.64 times post-screening in the low-risk group, 1.80 times in the moderate-risk group and 4.13 times in the high-risk group, respectively. There was no variation in the odds of individuals taking antihypertensives and antiplatelets across different risk levels pre-post imaging (Table 1).

Extractable data were available for studies that reported outcomes for individuals on LLM (4 studies, *n*=5,975 participants) and antihypertensives (three studies, *n*=5,749 participants) prepost screening in the imaging and no imaging groups. The proportion of individuals on LLM increased after screening in both groups but was higher in the imaging group. Individuals on LLM increased by 4.2 times in the imaging group and 2.2 times in those with no access to imaging results after screening. Individuals on antihypertensives increased by 1.99 times in the imaging group and 1.35 times in the no-imaging group post-screening (Table 1).

Common sources of bias in the randomized studies included the selection of reported results and the randomization process. Overall, 11% of randomized trials were rated as low or high risk of bias, and 78 % of studies were rated as having some concerns (Appendix Figure 2). Common sources of bias in the non-randomized studies included bias in selecting study participants, bias in the measurement of outcomes, bias due to confounding, and bias due to missing data (Appendix Figure 3). For the reported outcomes, low between-study heterogeneity ($I^2 \leq 50\%$) was observed in 10 (29%) cases, moderate heterogeneity (I^2 =50%–75%) was observed in 8 (24%) cases, and high heterogeneity (I^2 = 75%–100%) was observed in 16 (47%) cases (Table 1). Potential drivers of heterogeneity identified included differences in follow-up duration, age, sex (male or female), race, type of screening results provided, and participants' educational level.

Sensitivity analysis was performed by creating pooled estimates of the effect after excluding each study individually for each outcome. Minimal changes in the direction of the results (the proportion of individuals on LLM in the group with no disease and the odds of continuation of antiplatelet therapy in the group informed about the presence of coronary or carotid artery disease significantly increased when a study was removed) were observed, supporting the robustness of the pooled results. The likelihood of publication bias was evaluated by visual inspection of the funnel plots and Egger's regression test.[24] There was no evidence of publication bias in any of the reported outcomes (Appendix Figure 4).

The meta-regression showed significant effects of age, sex, race, and follow-up duration on the proportion of individuals on medications before and after screening. Age, sex, race, and follow-up duration were significantly associated with the proportion of individuals on LLM, with approximately 63% of the variation attributed to differences in follow-up duration (Appendix Figure 5). A significant positive relationship was observed between follow-up duration and the proportion of individuals on LLM post-imaging (β = 0.0146, p=0.002), whereas a significant negative relationship existed between age and proportion on LLM (β = -0.0600, p = 0.006) (see Appendix A). Regarding the proportion of individuals on antiplatelets after screening, age and sex were found to be significantly associated, potentially explaining 80% and 99% of the variation, respectively (see Appendix Figure 6). A significant negative relationship was observed between age (β= -0.0547, p=0.003), being female (β=-0.0241, p=0.000), and the proportion of individuals on antiplatelet medications (see Appendix A). The type of results provided and follow-up duration were significantly associated with the proportion of individuals on antihypertensives, explaining the variations observed across the studies (see Appendix Figure 7). Notably, a significant positive relationship was found between receiving results in the form of scores and images of arterial disease and being on antihypertensive medication (β=0.5705, p=0.012). A significant positive relationship between follow-up duration and the proportion of individuals on antihypertensive medication was also observed (β=0.0117, p=0.000) (see Appendix A). Meta-regression also showed significant effects of participants' level of education, type of results provided and method of delivery on initiation of LLM. Approximately 92% of the variation in LLM initiation can be attributed to differences in educational level (see Appendix Figure 8). Notably, a significant positive relationship (β=1.5018, p=0.013) was found between the provision of results in the form of scores and images and LLM initiation (see Appendix A). The method of delivery also had a significant effect on LLM initiation, contributing to 51% of the variation in LLM initiation. (see Appendix Figure 8). Appendix Figure 9 provides a visual summary of the effect estimates (Odds Ratios) from individual studies and their confidence intervals.

DISCUSSION

In this systematic review and meta-analysis, outcomes related to medication use and adherence such as initiation, continuation, and the proportion of individuals on

cardiovascular medications before and after provision of imaging results were reported. The findings showed that medication initiation and continuation after screening were largely influenced by abnormal imaging findings. Medication initiation rates and the proportion of individuals on medications were also higher in participants classified as moderate-risk and high-risk after imaging. Significantly higher rates of LLM initiation in the imaging group were observed. However, no significant difference in medication continuation rates was observed between the imaging and no-imaging groups.

Findings related to increased rates of LLM initiation and continuation due to the identification of diseased arteries are corroborated by a previous study.[8] Gupta et al.[8] showed that identification of calcified coronary artery plaque led to significantly higher odds of LLM initiation and continuation. Individuals informed about abnormalities in the coronary or carotid arteries may perceive their risk of experiencing cardiovascular events to be higher and may act to avoid danger.[16] Imaging findings can also influence physicians' decisions regarding aggressive risk factor therapy, which can potentially influence the rates of medication initiation and continuation following imaging.

The significant increase in LLM initiation observed in the imaging group compared to the no-imaging group suggests that both physicians and patients may be more inclined to start treatment following cardiovascular imaging. Imaging results provide tangible evidence of arterial condition and potential cardiovascular risks, motivating individuals to act. Decisions regarding initiation of cardiovascular medications in the no-imaging groups were influenced by findings from traditional or non-imaging CVD risk assessments. Therefore, it appears that adding cardiovascular imaging to traditional risk assessment strategies can enhance clinical decision-making and preventive behaviors. The findings align with current guidelines by the European Society of Cardiology (ESC) and the American Heart Association/American College of Cardiology (AHA/ACC) advocating for the integration of cardiovascular imaging with traditional risk factor assessment for the primary prevention of CVD.[30-32]

Meta-regression revealed some factors potentially influencing the findings. There was a negative relationship between age and log RR of the proportion of individuals on LLM and antiplatelet therapy, suggesting that an increase in age tends to decrease the likelihood of LLM and antiplatelet intake. As follow-up duration increased, the proportion of individuals on LLM and antihypertensive medication also increased, implying a lasting impact of cardiovascular imaging on LLM and antihypertensive medication intake. Being female was associated with a lower likelihood of being on antiplatelets. Others have reported that several factors may be responsible for adherence to prescribed medications, including costs of drugs, wait times, patient-clinician communication or relationship, age, gender, and educational status.[33] Considering these factors in the context of medication adherence for CVD prevention is crucial as certain individuals may require additional support to effectively adhere to recommendations.

In several instances, no significant increase in the pooled OR for the proportion of individuals on antiplatelet medications was observed. This may be attributed to the uncertainty in defining a population that would benefit from antiplatelet medication use for the primary prevention of CVD.[34] The US Preventive Services Task Force advises against low-dose aspirin use in persons 60 years of age or older for the primary prevention of stroke or heart attack.[35] Approximately half of the studies in this meta-analysis recruited participants with an average age of 60 years. This can influence physicians' decisions regarding treatment and antiplatelet medication.

Moreover, it's noteworthy that most of the studies included in the meta-analysis were conducted or published before 2023, and there have been changes in practice over this period. Considering the continuous advancements in imaging technologies and revisions to clinical guidelines for cardiovascular imaging, any recommendations or treatment decisions based on the findings from this review should be made in the context of current guidelines by organizations such as the ESC, AHA/ACC, and the World Health Organization.

Limitations

This review has various strengths, overcoming several limitations of previous reviews. These include examination of medication initiation, continuation, and proportion of individuals on medications (before and after screening). Specific CVD risk thresholds were also considered in the analysis. Data from randomized trials and non-randomized studies were considered. In other reviews that examined outcomes related to medication intake after vascular screening, the focus was often limited to imaging assessments of the coronary or carotid arteries. In this review, data were extracted from studies using imaging assessments of both the coronary and carotid arteries. By including studies using these two cardiovascular screening approaches, the review provides a more comprehensive assessment of medication use and adherence in the context of cardiovascular imaging. This study's sensitivity analysis also revealed the robustness of the pooled results.

This review had some limitations. Reported use of varied ways of communicating findings to participants, relying on different professionals and mediums of results communication and lifestyle counselling, and lack of outcome data in the studies included in this review limit the ability to make firm conclusions about these findings. Some studies did not report how participants were supported in adhering to recommendations. Standardization of risk communication strategies and support will enable comparisons between studies, strengthen conclusions, and reduce research waste. The inclusion of surveys as a follow-up measure in several of the included studies raises the possibility of reporting bias and risk of recall bias. The use of self-reported and standalone measures of medication adherence in several studies can potentially impact the validity and reliability of the findings. Hence, there is a need for standardization of outcome measures and rigorous methods to assess medication use and adherence. The limited availability of data on some domains of medication adherence and outcomes reported in studies included in this review also represents a limitation. Some domains of medication adherence, including implementation of the dosage regimen and discontinuation, could not be determined due to lack of data. Due to the limited number of studies reporting outcomes on the continuation of antihypertensives, there was insufficient data available for a meta-analysis of outcomes related to the continuation of antihypertensives and overall heterogeneity was high for several outcomes assessed. Despite the limitations discussed, this study is a useful addition to understanding the role of cardiovascular imaging in cardiovascular risk modification strategies.

CONCLUSIONS

Findings suggest that imaging results can influence prescribers' tendencies to adjust or initiate treatment, and patients' behaviors or responses, including filling prescriptions and taking the first dose of a prescribed medication. Higher rates of medication initiation, particularly lipid-lowering medication, were observed when prescribers and patients had access to imaging results. As such, integrating imaging into current cardiovascular risk assessment approaches can potentially influence cardiovascular risk management. However, evidence regarding medication continuation is mixed, suggesting the need for further research on more effective risk communication and modification strategies in cardiovascular disease prevention.

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CREDIT AUTHOR STATEMENT

Reindolf Anokye: Conceptualization, Methodology, Formal analysis, Validation, Writing — original draft, Writing — review & editing, Visualization, Investigation, Data curation, Project administration. Jack Dalla Via: Conceptualization, Methodology, Formal analysis, Validation, Writing — review & editing, Investigation. James Dimmock: Methodology, Writing — review & editing. Ben Jackson: Methodology, Writing — review & editing. Carl Schultz: Methodology, Writing — review & editing. Mie Schæffer: Writing — review & editing. Joanne M. Dickson: Methodology, Writing — review & editing. Lauren C. Blekkenhorst: Methodology, Writing — review & editing, Supervision. Mandy Stanley: Methodology, Writing — review & editing, Supervision. Jonathan M. Hodgson: Methodology, Writing — review & editing, Supervision. Joshua R. Lewis: Conceptualization, Methodology, Formal analysis, Validation, Writing — review & editing, Supervision.

SUPPLEMENTAL MATERIAL

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