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Prognostic role of asymmetric dimethylarginine following coronary angiography and percutaneous coronary intervention: current evidence, limitations, and future directions

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Abstract

Background. Asymmetric dimethylarginine (ADMA) is an endogenous inhibitor of nitric oxide synthase and a candidate marker of impaired endothelial nitric oxide signaling. Despite procedural advances, coronary artery disease (CAD) remains associated with residual risk after invasive investigation and revascularization. Objective. To critically synthesize evidence linking circulating ADMA to coronary anatomy, plaque characteristics, cardiovascular outcomes, and restenosis in patients undergoing coronary angiography (CAG) or percutaneous coronary intervention (PCI), with special attention to type 2 diabetes mellitus (T2DM). Evidence synthesis. In a meta-analysis of nine prospective CAG/PCI studies (6,374 participants), high versus low ADMA was associated with all-cause mortality (adjusted pooled RR 2.11, 95% CI 1.38–3.21), cardiovascular mortality (2.95, 1.14–7.68), major adverse cardiovascular events (2.10, 1.35–3.27), and restenosis (4.57, 2.52–8.30) [13]. However, other prospective cohorts yielded weaker or null associations for recurrent cardiovascular events [24], and findings vary by population, endpoint, kidney function, and analytical method [14–16,19]. A 2023 optical coherence tomography study associated higher ADMA with plaque vulnerability and subsequent events after PCI [12]. Conclusions. ADMA is a biologically credible research biomarker and frequently correlates with adverse prognosis, but independent association is not synonymous with improved prediction or actionable clinical benefit. Contemporary, assay-standardized, externally validated studies in defined PCI populations, particularly patients with T2DM, are needed.

Keywords

ADMA; asymmetric dimethylarginine; coronary angiography; percutaneous coronary intervention; endothelial dysfunction; cardiovascular prognosis; restenosis; type 2 diabetes mellitus.

1. Introduction. Coronary angiography defines luminal coronary anatomy, while PCI treats flow-limiting lesions; neither procedure directly quantifies systemic vascular biology. Subsequent myocardial infarction, death, and repeat intervention depend on the interaction of residual atherosclerotic burden, thrombosis, lesion characteristics, renal and metabolic comorbidity, and endothelial function. Invasive coronary endothelial testing has demonstrated prognostic relevance, providing a mechanistic reason to investigate circulating surrogates [5]. Yet biomarkers must satisfy a stricter requirement than biological plausibility: they must improve prediction and ideally modify clinical decisions beyond established risk factors [25,26].

ADMA is produced during degradation of methylated proteins and competitively inhibits nitric oxide synthase. Early mechanistic investigations established the regulatory importance of dimethylarginine dimethylaminohydrolase (DDAH), while epidemiological work linked higher ADMA with cardiovascular risk [1–4,15–17]. The question of interest is not whether ADMA has vascular effects, but whether a measured circulating concentration conveys robust, reproducible, clinically useful prognostic information after CAG or PCI. This distinction is particularly relevant because the literature includes overlapping meta-analyses and mixed angiography-only, stable CAD, acute coronary syndrome, and revascularized cohorts [13,14,18].

2. Scope and approach

Evidence was prioritized from indexed primary cohort studies, human mechanistic studies, relevant meta-analyses, methodological investigations, and prognostic-model reporting guidance. Searches and citation checking focused on PubMed-indexed reports about ADMA, coronary angiography, PCI, outcomes, restenosis, endothelial function, diabetes, and assay comparability. Because screening was not conducted under a predefined reproducible protocol, no PRISMA study count, comprehensive search claim, or formal pooled estimate is generated here. Numerical effect sizes are attributed to the original articles or published meta-analyses; they are not recalculated.

3. Biological rationale for an association with cardiovascular outcomes

3.1. Formation, clearance, and the DDAH–NO axis. ADMA arises after proteolysis of proteins containing methylated arginine residues. DDAH-dependent hydrolysis is an important metabolic pathway, and experimental DDAH manipulation alters NO production [1,2]. Increased ADMA may competitively inhibit endothelial nitric oxide synthase and reduce NO-dependent vasodilatation; NO also regulates platelet-endothelial interactions, leukocyte adhesion, and vascular smooth-muscle responses [3,4]. These mechanisms explain why ADMA might track diffuse vascular pathology, but circulating ADMA is not an interchangeable measurement of endothelial NO production or coronary microvascular function [3–5]. A key interpretative limitation is that ADMA is influenced by several biological compartments. Renal impairment, inflammatory and metabolic conditions, and altered protein turnover may affect concentrations, sometimes independently of the vascular lesion responsible for the index angiogram [4,7,10,22]. Moreover, association with the biological pathway does not prove that lowering ADMA itself improves outcomes; mechanistic experiments and observational prediction studies answer different questions [1,3,4].

3.2. Atherosclerotic burden and endothelial phenotype

An early coronary study reported an association between ADMA and the presence and severity of angiographic CAD [6], while a meta-analysis of 16 case–control studies (2,939 cases and 1,774 controls) found higher ADMA in CAD than in controls (weighted mean difference 0.248, 95% CI 0.156–0.340) [17]. These observations support a relationship with disease burden, but case–control contrasts cannot establish future risk or discriminate whether elevated ADMA is a cause, consequence, or correlated marker of atherosclerosis. Indeed, plaque morphology, luminal stenosis, microvascular function and circulating biochemical activity are not equivalent constructs [5,6,12].

4. Prospective prognostic evidence after coronary angiography

4.1. Large angiographic cohorts

In the Ludwigshafen Risk and Cardiovascular Health (LURIC) study, Meinitzer and colleagues measured ADMA in 3,238 participants referred for coronary angiography, of whom 2,543 had angiographic CAD and 695 did not. During a median

5.45-year follow-up, increasing ADMA was associated with total and cardiovascular mortality among participants with CAD [7]. The design supports an association in an angiographically characterized high-risk population, but its findings should not be described as a PCI-specific result: many patients underwent diagnostic evaluation rather than a uniform stenting procedure [7]. The AtheroGene cohort assessed baseline ADMA in 1,874 patients with CAD. During a mean 2.6-year observation period, the highest ADMA third had an HR of 2.48 (95% CI 1.52–4.06) for cardiovascular death or nonfatal myocardial infarction versus the lowest third; a multibiomarker model also retained ADMA as a risk marker [8]. This is supportive evidence for prospective association, although improved clinical decisions from biomarker-guided care were not directly tested [8,25].

4.2. Context from broader perspective evidence

Willeit and colleagues combined 22 prospective studies with 19,842 participants: comparing the highest with the lowest third of ADMA yielded an RR of 1.42 (95% CI 1.29–1.56) for cardiovascular disease [15]. Schlesinger and colleagues identified a similar but more modest pooled association for incident CVD (RR 1.33, 95% CI 1.22–1.45), alongside an association with all-cause mortality (RR 1.52, 1.37–1.68) [16]. These estimates support the general relevance of ADMA, yet they cannot be transferred without qualification to contemporary PCI patients because populations and outcomes were broader [15,16].

5. Evidence after PCI

5.1. Preprocedural ADMA and adverse events

In 153 patients with stable angina undergoing PCI, Lu and colleagues measured ADMA before intervention. Across increasing ADMA tertiles, adverse event counts over a median 16 months were 6, 17, and 28; compared with the lowest tertile, adjusted relative risks were 3.0 (95% CI 1.2–7.7) and 5.3 (2.2–12.9) in the middle and highest tertiles, respectively [9]. This was an influential direct PCI study, but the small cohort and era of intervention require cautious extrapolation to current drug-eluting stents, antithrombotic regimens, and lipid-lowering therapies [9].

The most directly relevant published synthesis is the 2022 meta-analysis by Wang and colleagues, based on nine prospective studies and 6,374 patients undergoing CAG/PCI. Its pooled adjusted high-versus-low ADMA risk ratios were 2.11 for all-cause death, 2.95 for cardiovascular death, 2.10 for major adverse cardiovascular events (MACE), and 4.57 for restenosis [13]. These results are sizable, but they combine distinct procedures and populations; a pooled association is not evidence that all four outcomes are predicted with similar precision in an individual PCI subgroup. Importantly, the mortality and MACE estimates arise from largely overlapping literature rather than independent replications across separate meta-analyses [13,14].

5.2. Acute infarction and primary PCI

Patients treated for acute myocardial infarction differ fundamentally from elective stable-CAD PCI patients: acute inflammation, myocardial necrosis, reperfusion injury, and renal hemodynamic changes may modify both circulating biomarkers and outcome risk. In a prospective cohort of 249 acute-MI patients, Zeller and colleagues reported an adjusted mortality HR of 4.83 (95% CI 1.59–14.71) for the highest ADMA tertile over one year, although the estimate was imprecise [10]. In a separate primary-PCI STEMI investigation, elevated admission ADMA was associated with impaired myocardial reperfusion and greater one-year mortality [11]. These studies support hypothesis generation, but their acute-phase sampling and short-term endpoints should not be conflated with long-term ADMA assessment months or years after PCI [10,11].

Importantly, the 2021 CAD meta-analysis by Ye and colleagues reported a non-significant association with all-cause mortality in its acute-coronary-syndrome subgroup (RR 2.11, 95% CI 0.93–4.78), and in studies with follow-up of one year or less (RR 2.15, 0.56–8.25) [14]. A wide confidence interval crossing unity is uncertainty, not proof of absence of association; it nevertheless weakens claims that ADMA consistently predicts early events in every acute population [14].

5.3. Plaque vulnerability evaluated by intravascular imaging

In 245 stable-CAD patients undergoing OCT-guided PCI, Huang and colleagues reported that higher ADMA was associated with intraplaque lipid and calcium, macrophage and microvessel features, and a higher OCT-derived vulnerability score.

Higher ADMA was also associated with subsequent adverse cardiovascular outcomes [12]. This study provides a plausible bridge between a circulating biochemical signal and structural plaque properties. However, single-center observational imaging studies face residual confounding, selection bias (patients eligible for OCT-guided PCI), and limited events for multivariable prediction. Whether ADMA adds information beyond validated imaging and clinical models remains unresolved [12,25].

5.4. Restenosis is not identical to MACE

Neointimal proliferation and vascular healing offer a mechanistic link between impaired NO signaling and restenosis; nevertheless, stent-era factors such as device platform, lesion complexity, diabetes, and follow-up imaging are critical. Wang and colleagues reported a pooled ADMA–restenosis RR of 4.57 (95% CI 2.52–8.30), but this should not be interpreted as a calibrated restenosis probability for modern drug-eluting stents [13]. Investigators should distinguish angiographic in-stent restenosis, clinically driven target-lesion revascularization, target-vessel revascularization, and hard ischemic events; the components may be causally and prognostically different [9,13,18].

6. Contradictory findings and sources of heterogeneity

A critical reading must include negative results. In the prospective KAROLA study (1,148 patients with stable coronary heart disease; median follow-up 8.1 years), adjusted ADMA was not associated with recurrent cardiovascular events (HR per standard deviation 1.02, 95% CI 0.86–1.21); the association with all-cause death was also not statistically clear (HR 1.15, 0.95–1.37) [24]. In patients with T2DM and microalbuminuria, Zobel and colleagues found ADMA associated with all-cause mortality but not their cardiovascular composite after adjustment [22]. Both results constrain any unconditional statement that elevated ADMA is universally an independent cardiovascular prognostic marker [22,24].

Variation may arise from different CAD stages, biomarker distribution, baseline kidney function, assay methods, the timing of blood collection relative to ischemia and PCI, treatment patterns, definitions of clinical endpoints, and statistical adjustment. Studies also report effects across tertiles, quartiles, or continuous scales. Such effect

sizes cannot be directly compared without considering exposure contrast and adjustment sets [7–16,19,22,24]. In meta-analysis, aggregation of observational cohorts does not remove confounding or rescue incompatible endpoint definitions [13–16].

7. The importance of type 2 diabetes mellitus

7.1. Diabetes as a biologically relevant but heterogeneous subgroup

T2DM combines hyperglycemia, insulin resistance, dyslipidemia, inflammation, and often impaired renal function, all potentially linked to vascular NO signaling. In the LURIC cohort, diabetes was associated with ADMA, but many other factors were correlated as well [7]. Dedicated T2DM evidence is therefore essential rather than relying solely on pooled analyses where diabetes is a covariate [7,18,20–23].

Krzyzanowska and colleagues prospectively followed 125 patients with T2DM: the highest baseline ADMA tertile was associated with increased risk of their composite cardiovascular endpoint (adjusted HR 2.37, 95% CI 1.05–5.35), while the joint consideration of ADMA and C-reactive protein was associated with a stronger contrast [20]. In a separate 270-patient T2DM cohort followed for a median 5.7 years, ADMA predicted MACE (highest versus lowest tertile HR 2.31, 95% CI 1.10–4.81) despite no significant baseline correlation between ADMA and HbA1c ($r = -0.09$, $P = 0.13$) [21]. The latter suggests that ADMA need not behave as a simple measure of chronic glycemia, although reported subgroup interaction with glycemic control requires replication [21]. Counterbalancing these studies, Zobel and colleagues found that among 200 patients with T2DM and microalbuminuria ADMA related to mortality but not the cardiovascular composite after multivariable adjustment, while symmetric dimethylarginine demonstrated a different pattern [22]. An Edinburgh T2DM cohort investigated genetic/environmental determinants of dimethylarginines and cardiovascular associations, reinforcing the relevance of biology and confounder structure rather than just a single fixed ADMA threshold [23]. Thus, even within T2DM, ADMA prognostic properties may vary by renal status, background vascular disease, endpoint, and treatment [20–23].

7.2. Research implications for post-PCI T2DM cohorts

A well-defined cohort specifically enrolling T2DM patients after PCI could address a genuine evidence gap: whether ADMA is associated with later adverse events after accounting for age, kidney function, HbA1c, lipid measures, left ventricular ejection fraction, baseline coronary complexity, and medications. Baseline should be explicitly defined as either before PCI, shortly after PCI, or at a later stable visit; the meaning of ADMA at each time point differs. When participants have undergone PCI months to years before recruitment, changes measured over the next six months reflect longitudinal follow-up, not the immediate biological response to stent placement [9–14,20–24].

If clinical event counts are limited, investigators should avoid claiming definitive MACE prediction. More feasible prespecified outcomes may include associations of ADMA with clinical/metabolic phenotypes and within-person changes, clearly separated from exploratory clinical outcomes. Three-group comparisons (T2DM with PCI, T2DM without PCI, PCI without T2DM) can test differences in circulating ADMA but do not, by themselves, measure prognosis unless prospective events are collected [20–24,25].

8. Analytical measurement and clinical interpretability

The numerical interpretation of ADMA depends on assay technology, calibration, specimen preparation, and preanalytical conditions. In a method-comparison study, Sudová and colleagues reported that immunoassay-derived ADMA concentrations were almost 30% higher than values obtained with a chromatographic mass-spectrometry method in their sample; agreement was not interchangeable despite correlation [19]. This has immediate implications for using cutoff values transported across laboratories. A threshold reported in one cohort and assay cannot be presented as a universally validated clinical boundary [19].

Clinical studies should report assay manufacturer/platform, units (typically μmol/L), sample matrix, collection timing, storage conditions, quality-control performance, intra-/inter-assay variation if available, missing-data handling, and blinding of laboratory personnel to outcomes. Analytical reliability is a prerequisite for external prognostic replication. A correlation with clinical outcomes does not establish

that serial ADMA monitoring is useful or that therapeutic changes driven by its result improve patient outcomes [18,19,25,26].

9. From independent association to incremental prediction

The expression "independent predictor" is often used when a biomarker retains a nonzero coefficient in an adjusted model. Such evidence is weaker than demonstrating clinical usefulness. The latter requires a prespecified comparison between a well-calibrated clinical model and the same model plus ADMA, using discrimination (e.g., C-statistic), calibration, net benefit across relevant risk thresholds, and external validation. Small P values or large risk ratios do not automatically imply better patient stratification [25,26].

Published CAG/PCI meta-analyses quantify associations, not the effect of ADMA-guided testing on treatment selection or patient outcomes [13,14]. Likewise, some reported single-study cutoffs may be optimistic if selected in the same sample used to assess prognosis. With rare outcomes, overfitted multivariable models and data-driven threshold searches may be misleading; validation cohorts and transparent model reporting are essential [25]. Clinically meaningful incremental prediction remains the central unanswered question.

10. Evidence summary

Evidence source	**Population/design**	**Principal finding**	**Key caveat**
Lu et al., 2003 [9]	153 stable-angina PCI patients; prospective	Increasing baseline ADMA tertiles tracked post-PCI events	Small, early intervention-era study
Meinitzer et al., 2007 [7]	3,238 angiography patients; LURIC	ADMA associated with mortality in angiographic CAD	Not a uniform PCI cohort
AtheroGene [8]	1,874 CAD patients; prospective	Top-third ADMA associated with CV death/nonfatal MI	Association not intervention benefit
Wang et al., 2022 [13]	9 CAG/PCI cohorts; n=6,374	Pooled RR for MACE 2.10 (1.35–3.27)	Mixed procedures, endpoint definitions

Ye et al., 2021 [14]	11 CAD cohorts; n=9,496	Pooled RR for MACE 1.71 (1.27–2.32)	ACS/short follow-up subgroups uncertain
Huang et al., 2023 [12]	245 OCT-guided PCI patients	ADMA linked to vulnerable plaque and events	Selection and residual confounding
KAROLA [24]	1,148 stable CHD patients; prospective	Adjusted recurrent-CVD HR 1.02 (0.86–1.21)	Important null finding
Zobel et al., 2017 [22]	200 T2DM + microalbuminuria	ADMA related to mortality, not adjusted CVD composite	Diabetes subgroup heterogeneity

11. Practical priorities for future studies

First, restrict the target population and time origin: diagnostic CAG without PCI, elective PCI, primary PCI for STEMI, and late post-PCI follow-up are analytically distinct clinical questions [7,9–14]. Second, prespecify the endpoint and its components; death, infarction, stent restenosis, and repeat revascularization should not be treated as interchangeable [9,13]. Third, standardize assay and adjustment for kidney function, diabetes severity, statin therapy, and coronary complexity [7,19–24]. Fourth, evaluate repeated ADMA measurement only if temporal stability and within-patient variation are characterized [19]. Finally, report both baseline-model performance and the additional benefit, if any, of adding ADMA, ideally with external validation and decision-analytic assessment [25,26].

12. Conclusions

Circulating ADMA is mechanistically relevant to endothelial NO signaling and has repeatedly been associated with mortality and cardiovascular outcomes in angiographic CAD and post-PCI cohorts. Published meta-analyses report elevated pooled risks, while intravascular imaging studies extend the argument to plaque vulnerability [12–16]. However, null prospective findings, renal and metabolic confounding, mixed populations, changes in PCI technology, and assay non-equivalence prevent treating ADMA as an established standalone prognostic test [19,22,24]. Particularly in T2DM, carefully designed prospective post-PCI studies should determine whether ADMA meaningfully improves standardized clinical risk models. For now, ADMA is a

promising research biomarker rather than a routinely validated decision-making tool [18,25,26].

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