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AI-CVD: A New Paradigm for Comprehensive Cardiovascular and Metabolic Disease Risk Assessment Using Multidimensional AI-Enabled CT Biomarkers

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Abbreviations:
AF=atrial fibrillation
AI=artificial intelligence
ASCVD=atherosclerotic cardiovascular disease
AVC=aortic valve calcium
BMD=bone mineral density
CAC=coronary artery calcium
CCTA=coronary computed tomography angiography
CHD=coronary heart disease
CI=confidence interval
CT=computed tomography
COPD=chronic obstructive pulmonary disease
CVD=cardiovascular disease
HF=heart failure
HR=hazard ratio
LAI=liver attenuation index
LV=left ventricular
MESA=Multiethnic Study of Atherosclerosis
MVC=mitral valve calcium
MRI=Magnetic Resonance Imaging
NLST=National Lung Screening Trial
RA=right atrial
RV=right ventricular
SCAPIS - Swedish CArdioPulmonary bioImage Study
T2DM=type 2 diabetes mellitus

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Abstract

Accurate cardiovascular risk assessment is the foundation of preventive cardiology, but conventional risk scores and coronary artery calcium (CAC) scoring provide only a partial view of the biological processes that lead to coronary heart disease, stroke, heart failure, atrial fibrillation, and metabolic diseases. AI-CVD is a multidimensional artificial intelligence framework that expands the value of gated CAC scans and routine non-gated chest CT scans by extracting quantitative imaging biomarkers from the available field of view. The platform includes AI-CAC coronary calcium assessment; cardiac chamber volumetry and ratios; ventricular wall thickness, thoracic aortic calcium; aortic and mitral valve calcification; ascending and descending aortic dimensions; pulmonary artery dimensions; epicardial, visceral, and subcutaneous adiposity; liver attenuation; thoracic skeletal muscle volume and attenuation; lung low- and high-attenuation biomarkers; and thoracic vertebral bone mineral density. These biomarkers show improved prediction or risk stratification for cardiovascular and metabolic diseases beyond risk factors and/or conventional CAC scoring. Persons with borderline or intermediate risk of ASCVD may be suitable for evaluation with AI-CVD with appropriate preventive therapy for those with elevations in these measures. We summarize the technical foundation, evidence base, clinical interpretation, and future research agenda for AI-CVD as a comprehensive CT-based cardiovascular and metabolic phenotyping paradigm.

Keywords: artificial intelligence; coronary artery calcium; metabolic disease; imaging; heart failure; atrial fibrillation; preventive cardiology; opportunistic CT; cardiometabolic risk

Introduction

Cardiovascular disease (CVD) remains the leading cause of morbidity and mortality worldwide despite decades of guideline-directed prevention. Clinical risk assessment begins with population-derived scores, risk-enhancing factors, and selective use of subclinical atherosclerosis testing, particularly coronary artery calcification (CAC), to guide treatment initiation and intensification [1-4]. These tools are essential, but they remain incomplete at the individual level because they are driven largely by risk factors rather than direct measurement of the organs and tissues in which risk is expressed.

The Pooled Cohort Equations and the newer PREVENT CVD risk algorithms estimate future risk using demographic, hemodynamic, lipid, metabolic, and renal measures [3-4]. These models perform well for population-level calibration but can misclassify individuals who already have substantial subclinical disease or who express risk through pathways not fully captured by standard risk factors, such as cardiac chamber remodeling, skeletal muscle dysfunction, liver steatosis, valve calcification, pulmonary vascular remodeling, or bone loss [5-6]. There are other limitations of the PREVENT CVD risk assessment including the need for several laboratory inputs that may not always be available or require multiple visits to a laboratory. This risk algorithm was derived from data in the United States and its performance in international populations is not well-established.

CAC is the most widely validated imaging biomarker for subclinical coronary atherosclerosis and is now central to preventive cardiology decision-making [7-8]. However, conventional CAC reporting is

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still narrow. It primarily reports the Agatston score, which reflects calcified plaque above a fixed density threshold and a minimum area criterion. A CAC scan, however, contains far more information than a single coronary calcium score. The same CT scan also allows for measurement of cardiac chambers, valves, thoracic aorta, pulmonary artery, lungs, liver, vertebral bodies, skeletal muscle, and adipose. While these are routinely captured, they are typically not measured and quantified, allowing for their opportunistic assessment by AI.

AI-CVD was developed to convert this unused CT information into a structured, quantitative, and clinically interpretable comprehensive cardiovascular and metabolic disease phenotypes. The central premise is simple: a single CT scan can serve as a multidimensional risk assessment platform if AI reliably segments relevant anatomy, quantifies disease-linked biomarkers, and integrates them with clinical variables. This review summarizes AI-CVD as a comprehensive paradigm rather than as a collection of isolated algorithms.

What AI-CVD Measures

AI-CVD analyzes gated CAC scans and, where technically appropriate, routine non-gated chest CT scans to produce quantitative biomarkers across cardiovascular, pulmonary, skeletal, hepatic, and body-composition domains. The major components are summarized in **Table 1** and the **Graphical Abstract**. This multi-organ scope is important because cardiovascular events arise from interacting disease pathways: coronary and extra coronary atherosclerosis, myocardial dysfunction and remodeling, atrial cardiomyopathy, valvular calcification, vascular aging, pulmonary vascular disease, systemic inflammation, insulin resistance, sarcopenia/myosteatosis, hepatic steatosis, and skeletal fragility.

Technical Framework

AI-CVD uses deep learning and image-processing pipelines to segment target structures and derive quantitative imaging biomarkers. The platform is designed for two complementary use cases. First, it enhances dedicated CAC scans by extracting more information from the existing non-contrast, low-radiation acquisition. Second, it enables opportunistic risk assessment from routine chest CT scans performed for non-cardiac indications, where CAC and extracardiac findings are often visible but inconsistently quantified [9].

The technical workflow has four general steps: (1) scan ingestion and series selection; (2) automated segmentation of relevant anatomy; (3) biomarker-specific quantification using intensity, geometry, density, volume, and anatomic localization; and (4) risk modeling or structured reporting. For coronary analysis, this includes coronary artery segmentation, noise calibration, density-aware scoring, and spatial filtering. For chamber volumetry, it includes segmentation of the four cardiac chambers and left ventricular (LV) mass from non-contrast CT. For the extracardiac modules, it includes organ- or tissue-specific segmentation of the thoracic aorta, pulmonary artery, lungs, liver, vertebral bodies, skeletal muscle, and adipose compartments.

Within an explainable AI framework, a key feature of AI-CVD is that its measurements are not treated as opaque or isolated outputs, but as interpretable imaging-derived biomarkers. They can be interpreted individually, as in the case of BMD or aortic valve calcification, or integrated into risk models, as in AI-CVD-HF [10]. This approach is aligned with cardiovascular-kidney-metabolic concepts in which multiple organ systems express shared metabolic, inflammatory, hemodynamic, and atherosclerotic risk pathways [4].

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Advanced Coronary Calcium Assessment: AI-CAC

Conventional Agatston scoring is binary at the voxel level: calcium is counted only when attenuation is at least 130 HU and meets a minimum contiguous area requirement. This design has been clinically successful, but it can miss very small, low-density, or partially calcified plaques, particularly in individuals reported as CAC=0. Agatston-2.0 was developed to address this limitation using an AI-assisted, coronary-anatomy-constrained, continuous quantification framework [11] (**Figure 1**).

In the Agatston-2.0 Part I study, an AI-CAC score >0 was detected in 862 participants, representing 21.7% of individuals classified as CAC=0 by conventional Agatston scoring. Participants with AI-CAC >0 had higher 20-year CHD incidence than those with AI-CAC=0 (7.7% vs. 3.8%, $p<0.0001$), and AI-CAC >0 remained independently associated with incident CHD after adjustment for traditional risk factors (HR 1.71, 95% CI 1.18-2.47). AI-CAC also predicted progression to positive conventional CAC (adjusted HR 1.95, 95% CI 1.70-2.24) [11].

These findings do not diminish the clinical value of CAC=0; rather, they refine it. The power of zero remains a powerful risk concept, but AI-CAC suggests that a subset of conventional CAC=0 individuals have detectable early coronary calcification that carries meaningful long-term risk. This approach may be particularly relevant to opportunistic CAC screening because CARDIA data show that approximately 20% of young adults who were initially free of obesity and traditional cardiovascular risk factors developed detectable CAC over two decades [12]. In broader MESA analyses, AI-derived CAC and chamber metrics significantly improved all-CVD prediction compared with the Agatston score alone, with AUC improvements over 1-, 5-, 10-, and 15-year follow-up [13].

Future coronary development is moving toward more complete non-contrast coronary plaque phenotyping, including density, morphology, location, and potentially calcified and semi-calcified plaque burden. This emerging direction could further bridge the gap between conventional calcium scoring and the plaque-burden concepts used in CCTA while preserving the advantages of non-contrast CT.

Cardiac Chamber Volumetry: Heart Failure, Atrial Fibrillation, and Stroke

AI-CVD cardiac chamber volumetry is one of the clearest examples of why CAC scans should not be treated as coronary-only tests. Left atrial (LA) enlargement, LV enlargement, right ventricular (RV) enlargement, right atrial (RA) enlargement, and increased LV mass represent structural remodeling pathways linked to atrial fibrillation (AF), stroke, heart failure (HF), pulmonary hypertension, and cardiovascular metabolic diseases. AI-CVD allows for the identification of persons with enlarged left atrium (LA) and left ventricle (LV) chambers at increased risk of HF or AF who would not otherwise be identified due to CAC=0 or low 10-year ASCVD risk (**Figure 2**).

In MESA, AI-enabled LA volumetry from CAC scans predicted incident AF as early as 1 year and outperformed the CHARGE-AF risk score at years 1, 2, and 3 and NT-proBNP across years 1-5. AI-LA volume also improved net reclassification when added to CHARGE-AF and NT-proBNP [14]. A related Stroke analysis showed that AI-derived LA volume index and LA/RA and LA/LV volume ratios from CAC scans predicted long-term AF and stroke [15]. In a paired CAC and cardiac MRI analysis, AI-LA volume and human-measured MRI LA volume similarly predicted incident AF and stroke over 15 years, with AUCs for AI-versus MRI of 0.802 vs. 0.798 for AF and 0.762 vs. 0.751 for stroke [16] (**Figure 3**).

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For HF, AI-chamber volumetry outperformed the Agatston score and NT-proBNP and improved risk classification beyond clinical risk factors [17]. In another paired analysis, AI-measured LV volume from CAC scans predicted incident HF comparably to cardiac MRI LV volume measured by human experts [18]. These findings suggest that CAC scans can provide MRI-like structural risk information for selected chamber measurements, at population scale and without the cost and access barriers of MRI.

Left-to-right chamber ratios extend this framework by focusing on the balance between cardiac compartments. In a pooled MESA and Framingham analysis, LV/RV and LA/RA ratios strongly predicted incident HF, with the highest LV/RV percentile showing particularly high cumulative HF incidence [19]. In a separate MESA/Framingham study, individuals without T2DM but with enlarged LA, LV, or LV mass had HF incidence rates comparable to or higher than individuals with T2DM and normal chambers, emphasizing that structural remodeling can identify high-risk individuals who may not meet traditional Stage A HF criteria [20].

Thoracic Aorta, Aortic Dimensions, Pulmonary Artery, and Pulmonary Hypertension Risk

AI-CVD also quantifies extracoronary vascular structures. Thoracic aortic calcium (TAC) reflects systemic vascular aging and extracoronary atherosclerosis. Prior studies have shown that TAC is associated with cardiovascular outcomes and mortality [21-22]. In MESA, TAC was associated with incident CAC and CAC progression, while TAC added prognostic information beyond CAC for CHD mortality in men and was associated with non-coronary cardiovascular mortality in both sexes [23-24]. AI-CVD enables automated TAC quantification at scale [25]. This is clinically important because atherosclerosis is not confined to the coronary arteries; the thoracic aorta can express diffuse vascular injury that may relate to stroke, mortality, and generalized vascular aging.

In addition to calcification, AI-CVD measures aortic dimensions, including the ascending aorta, descending aorta, and related volumetric or diameter features. These measurements support standardized reporting of aortic enlargement and aortic aneurysm risk markers from scans that may already contain the thoracic aorta. Automated measurement may reduce interobserver variability and increase the likelihood that clinically relevant aortic dilatation is consistently identified and flagged for clinician review, including consideration of appropriate follow-up imaging.

The pulmonary artery module measures pulmonary artery dimensions and related vascular geometry. Pulmonary artery enlargement is a recognized imaging marker associated with pulmonary hypertension and cardiopulmonary disease. When combined with chamber data, lung attenuation, and clinical risk factors, pulmonary artery measurements can provide additional context for dyspnea, right-heart strain, HF, emphysema, and pulmonary vascular remodeling [26].

Aortic and Mitral Valve Calcification

AVC and mitral valve or annular calcification (MVC/MAC) are frequently visible on CAC and chest CT scans but are often not quantified [27-30]. MAC has also been associated with incident AF in MESA, supporting its inclusion in broader AI-CVD reporting [31].

The AI-CVD valve modules automate leaflet- annular-region segmentation and calcium quantification. AI-derived aortic valve calcification (AVC) identifies clinical aortic stenosis with an AUC of 0.934 compared with 0.883 for manual expert measurement, correctly identifying 18 of 22 aortic stenosis

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cases compared with 15 of 22 by manual measurement [32]. These data support the potential value of automated valve calcium reporting as part of routine CAC or chest CT interpretation.

Lung Attenuation, Emphysema, and Interstitial Lung Abnormality

The lung is central to cardiopulmonary risk but is usually treated separately from cardiovascular prevention. Current GOLD guidance emphasizes COPD prevention, diagnosis, and management as part of an integrated clinical strategy [33]. AI-CVD lung attenuation analysis quantifies low attenuation, commonly expressed as the percentage of lung voxels below -950 HU, as an emphysema-like biomarker. It can also quantify high attenuation patterns, such as voxels above thresholds used to identify interstitial or fibrosis-like abnormalities, depending on scan protocol and field of view.

The clinical value of lung attenuation goes beyond pulmonary diagnosis. Emphysema, chronic obstructive pulmonary disease (COPD), pulmonary vascular remodeling, and HF often coexist and share risk pathways such as smoking, inflammation, hypoxia, and vascular dysfunction. AI-CVD provides a method for quantifying these lung features in the same report that includes coronary, cardiac, aortic, and metabolic metrics.

In MESA, AI-measured myosteatosis from CAC scans was a strong predictor of future clinically diagnosed COPD and was more strongly associated with COPD than an emphysema-like lung biomarker. Among 5,535 participants, 396 developed COPD over 20 years. Myosteatosis showed an adjusted HR of 2.74 (95% CI 1.81-4.16), compared with 1.50 (95% CI 1.12-2.00) for emphysema-like lung measurements [34]. These data highlight a broader principle: AI-CVD can capture systemic vulnerability, not only organ-specific visible disease.

Liver Attenuation, Adiposity, Myosteatosis, and Cardiometabolic Risk

Cardiovascular and metabolic disease risk assessment are major strengths of the AI-CVD approach. The liver, adipose tissue, and skeletal muscle are visible in CAC and chest CT fields of view and express metabolic dysfunction that may precede overt type 2 diabetes, CVD events, and HF. AI-CVD quantifies liver attenuation, epicardial fat, visceral fat, subcutaneous fat, skeletal muscle volume, and skeletal muscle attenuation.

In MESA, AI-enabled liver steatosis measurement using liver attenuation index (percentage of liver voxels below 40 HU) predicted CVD events and all-cause mortality over 15 years. Participants in the highest liver attenuation index (LAI) quartile had greater 15-year CVD incidence than those in the lowest quartile (19% vs. 12%, $p<0.0001$), and LAI remained associated with CVD (HR 1.43), stroke (HR 1.77), and all-cause mortality (HR 1.36) independent of traditional CVD risk factors and Agatston CAC score [35] (**Figure 3**).

In another MESA analysis of participants without obesity or hyperglycemia at baseline, AI-derived adiposity measures predicted new-onset T2DM over a median 19.7 years. Key predictors included LAI (HR 3.13), subcutaneous fat index (HR 2.85), total visceral fat index (HR 2.49), and epicardial fat index (HR 1.59), comparing high versus low quartiles [36]. This is particularly important because a substantial proportion of diabetes develops in individuals who do not meet traditional obesity or hyperglycemia definitions at baseline.

Myosteatosis represents fat infiltration within skeletal muscle and may reflect systemic inflammation, insulin resistance, mitochondrial dysfunction, frailty, and impaired cardiopulmonary reserve. AI-CVD

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measurement of skeletal muscle attenuation therefore complements adiposity and liver attenuation, providing a more complete phenotype of cardiometabolic health than BMI alone.

Bone Mineral Density and Osteoporosis

Thoracic vertebral bone mineral density (BMD) is another underused feature of CAC and chest CT scans. Osteoporosis and osteopenia are common, underdiagnosed, and clinically consequential, especially because many individuals who qualify for BMD testing do not undergo DEXA. AI-CVD AutoBMD quantifies trabecular vertebral BMD and can report T-scores and Z-scores from CT scans obtained for other reasons [37-38].

In validation work using MESA and Harbor-UCLA data, automated BMD strongly correlated with manual BMD measurements and accurately detected osteoporosis and osteopenia [37]. In paired lung and cardiac CT scans acquired the same day, AutoBMD measurements in lung and cardiac scans were equivalent, with a Pearson correlation coefficient of R2=0.95 and a Bland-Altman mean difference of 1.37 g/cm3 between lung and cardiac scans [38] .

BMD is not a purely skeletal add-on. Low BMD, vascular calcification, inflammation, renal-metabolic disease, frailty, falls, and cardiovascular events often cluster in older adults. Reporting BMD within AI-CVD can therefore improve whole-person preventive care from a scan that was not originally ordered for osteoporosis screening.

Integrated AI-CVD Risk Models

The most comprehensive use of AI-CVD is not simply to report individual measurements but to integrate them into endpoint-specific models. This is especially relevant for HF, where conventional prevention is less mature than ASCVD prevention and where structural imaging markers may identify risk earlier than symptoms or blood biomarkers [39].

AI-CVD-HF combines selected AI-derived features from CAC scans with clinical variables. Selected features included AI-CAC score, LV/RV volume ratio, LA volume, LV mass, visceral fat volume, skeletal muscle mean density, TAC, age, and sex. The 10-year AUC of AI-CVD-HF was 0.83 compared with 0.79 for PREVENT-HF, with improved AUPRC and more favorable calibration [10,40]. In addition, **Figure 5** shows how all the AI-CVD derived features derived from MESA CT scans (highest quartile for each) contribute to total CVD incidence over 18 years [41].

These findings illustrate why AI-CVD should be framed as a platform rather than a single biomarker. For CHD, AI-CAC and plaque features are central. For AF and stroke, LA volume and atrial remodeling dominate. For HF, LV/RV balance, LA volume, LV mass, adiposity, skeletal muscle density, and vascular calcification all contribute. For incident aortic stenosis prediction, valve calcification and TAC are central. For development of type 2 diabetes, liver attenuation, and upper abdominal visceral adiposity are dominant. For COPD, myosteatosis and lung attenuation are informative. A comprehensive report can therefore be tailored to the endpoint without discarding the broader phenotype.

Clinical Workflow and Reporting

AI-CVD can be implemented in two complementary workflows. In a dedicated CAC scan workflow or even non-gated chest CT scans, the report can include the conventional Agatston score, AI-

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CAC/Agatston-2.0 outputs, chamber volumetry, LV mass, valve and aortic calcification, aorta/PA dimensions, BMD, liver attenuation, adiposity, muscle, and lung attenuation when the field of view and scan quality permit, with the same principles also being applicable opportunistically to clinically acquired chest CT scans, particularly for assessing CAC, BMD, lung attenuation, aorta/PA dimensions, liver attenuation, and body composition.

For clinical adoption, the report should separate (1) validated quantitative measurements, (2) risk-model outputs, and (3) actionable interpretations. For example, a high AI-CAC score may support more intensive ASCVD prevention; enlarged LA may support AF risk evaluation including echocardiography to rule out valvular disease; enlarged LV or high LV/RV ratio may support HF prevention and echocardiography consideration; high AVC may support echocardiographic assessment for aortic stenosis; low BMD may support osteoporosis evaluation; high liver attenuation index or visceral adiposity may support cardiometabolic intervention; and high lung attenuation/low attenuation patterns may support pulmonary evaluation.

Limitations and Research Agenda

Several limitations should be emphasized. First, many AI-CVD results derive from observational cohorts such as MESA and Framingham, which provide strong long-term outcomes but require additional external validation across contemporary scanners, vendors, slice thicknesses, races/ethnicities, and clinical practice settings. Second, not every scan includes the same field of view, so all modules may not be measurable in every patient. Third, thresholds for action require endpoint-specific validation and clinical consensus. Fourth, while AI-CVD improves risk prediction, prospective studies are needed to determine whether AI-CVD-guided interventions improve hard outcomes and reduce downstream costs.

There are also unanswered questions and several considerations for further research include the following: (1) external validation in large paired CAC/CCTA cohorts such as Swedish CArdioPulmonary bioImage Study (SCAPIS); (2) head-to-head comparison of AI-CAC, AI-CVD, and AI-CCTA plaque assessment; (3) prospective implementation studies to test whether AI-CVD reports change physician and patient behavior; (4) endpoint-specific risk models for CHD, HF, AF, stroke, diabetes, COPD, osteoporosis, pulmonary hypertension, and aortic aneurysm; (5) cost-effectiveness studies; and (6) standardized reporting language and thresholds for each module. Further from early detection of lung nodules and other lung biomarkers, AI-CVD will be exploring opportunities for lung cancer risk assessment.

AI-CVD-CXR represents a complementary extension of the same concept to chest radiography, but the current review is focused on CT-based AI-CVD because the attached evidence base and biomarker modules are CT-derived. A future companion article could address AI-CVD-CXR for opportunistic risk prediction from chest X-rays.

Clinical Implications:

For decades, preventive cardiology has relied primarily on a small group of conventional risk factors - cholesterol, blood pressure, diabetes, smoking, obesity, age, and sex - to perform two fundamentally different tasks: determine who is likely to experience a cardiovascular event and decide which biological abnormality should be treated. This approach transformed cardiovascular prevention, but it has an inherent limitation. Risk factors are indirect, upstream exposures; they do not directly measure

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whether disease has developed, which organs are already affected, or through which pathway an individual patient is becoming vulnerable.

AI-CVD imaging biomarkers can be used to determine whom to treat and use conventional risk factors to determine what to treat. This new paradigm does not replace risk factors; it assigns them their proper role. Once AI-CVD identifies a patient with a high-risk phenotype, conventional and emerging biomarkers can guide treatment selection. Elevated LDL or apoB directs lipid-lowering therapy. Hypertension directs antihypertensive treatment. Hyperglycemia, visceral adiposity, or liver steatosis directs cardiometabolic intervention. Smoking and emphysema-like changes demand tobacco cessation and pulmonary management. Left atrial enlargement may justify closer surveillance for atrial fibrillation. Left ventricular remodeling or an abnormal LV/RV relationship may trigger evaluation and prevention of heart failure. Valve calcium may prompt echocardiographic assessment. Thus, imaging identifies the vulnerable patient, while risk factors identify the treatable mechanism.

The proposed clinical framework is therefore straightforward in helping to decide 1) whom to treat: patients with demonstrable subclinical disease, adverse remodeling, or a high-risk AI-CVD phenotype and 2) what to treat: the patient's actionable drivers - atherogenic lipoproteins, blood pressure, glycemia, smoking, obesity, inflammation, physical inactivity, and organ-specific abnormalities. This helps use move beyond asking only "How many risk factors does this patient have?" but instead, more meaningfully ask "What disease is already present, where is it being expressed, and which modifiable pathway should we target?" Prospective implementation and outcomes studies remain necessary, but the conceptual transition is already clear. The future of prevention is not risk-factor abandonment; it is risk-factor precision guided by direct, AI-enabled measurement of disease.

Conclusions

AI-CVD expands cardiovascular prevention from risk-factor estimation and conventional CAC scoring to comprehensive, CT-derived comprehensive cardiovascular and metabolic disease phenotyping. From a single scan, it can quantify coronary calcium and plaque features, cardiac chamber remodeling, valvular and aortic calcification, vascular dimensions, lung and liver abnormalities, adiposity, skeletal muscle quality, and bone mineral density. Available evidence supports improved risk stratification for CHD, total CVD, HF, AF, stroke, diabetes, COPD, osteoporosis, and mortality. For cardiac chamber biomarkers in particular, longitudinal studies support temporality, dose-response, strength of association, consistency, plausibility, and coherence [15-20].

These findings support a new clinical paradigm: use AI-CVD imaging biomarkers to help determine whom to treat and use conventional risk factors and other actionable biomarkers to determine what to treat. AI-CVD does not replace established risk factors; it assigns them a more precise therapeutic role by identifying the patients in whom disease or adverse remodeling is already present. The next step is prospective validation to determine whether AI-CVD-guided prevention improves treatment allocation, clinical outcomes, cost-effectiveness, and preventive care at scale.

Executive Summary

1. **AI-CVD is a comprehensive CT-based cardiovascular and metabolic phenotyping platform**, not simply an improved CAC algorithm. It extracts multiple biomarkers from a single CAC or chest CT scan, including coronary calcium, cardiac remodeling, vascular and valvular calcification, adiposity, liver fat, muscle quality, lung abnormalities, and bone mineral density.

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2. **Conventional risk factors estimate risk but do not directly measure disease.** AI-CVD identifies subclinical disease and organ-level vulnerability that may be missed by cholesterol, blood pressure, glucose, obesity, smoking status, or clinical risk scores alone.
3. **The proposed clinical paradigm is to use AI-CVD imaging biomarkers to determine whom to treat and conventional risk factors to determine what to treat.** Imaging identifies the vulnerable patient; modifiable factors such as LDL, blood pressure, glycemia, smoking, and obesity guide therapy.
4. **Evidence from MESA, Framingham, and related cohorts suggests that AI-CVD can improve risk stratification for CHD, HF, AF, stroke, diabetes, COPD, osteoporosis, and mortality.** Prospective studies are now needed to determine whether AI-CVD-guided prevention improves outcomes and cost-effectiveness.

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Table 1. Core AI-CVD components and their intended clinical interpretation

AI-CVD component	Key AI-derived outputs	Primary risk domains	Clinical interpretation
AI-CAC / Agatston-2.0	Conventional Agatston score; AI-CAC score; calcified and semi-calcified plaque volumes; coronary plaque density, number, location, and vessel distribution; early/low-density calcification signals derived using scan-noise adjustment and individualized HU thresholding	CHD, ASCVD	Refines the "power of zero", improves risk stratification beyond conventional CAC, and may support serial monitoring.
Cardiac chamber volumetry	LA, LV, RA, RV volumes; LV mass; BSA-indexed values	HF, AF, stroke	Detects subclinical myocardial and atrial remodeling that may not be reflected by CAC score or standard risk factors.
Left-to-right chamber ratios	LA/RA, LV/RV, and related ratios	HF, cardiopulmonary imbalance	Captures asymmetric chamber remodeling and may outperform absolute chamber size in selected HF prediction tasks.
Thoracic aortic calcium	Ascending/descending thoracic aortic calcification burden	Systemic atherosclerosis, vascular aging, CVD and stroke risk	Adds extra coronary atherosclerosis information to the CAC report.
Aortic valve calcification	AVC score; leaflet-level calcification patterns	Aortic stenosis, CVD risk	Identifies an often-unreported marker of aortic stenosis and calcific valve disease.
Mitral valve / annular calcification	Mitral valve and mitral annular calcification burden	Valve disease, AF/stroke risk, systemic calcification	Adds information on calcific valve and annular disease that is commonly visible but not routinely quantified.
Aortic dimensions	Ascending aorta, descending aorta, and thoracic aortic volume/diameters	Aortic aneurysm and vascular remodeling	Quantifies thoracic aortic enlargement and supports standardized reporting of aneurysm risk markers.
Pulmonary artery dimensions	Main pulmonary artery diameter/volume and related aorta ratios	Pulmonary hypertension, HF,	Provides a quantitative marker of pulmonary

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		cardiopulmonary disease	vascular remodeling visible on chest CT.
Lung attenuation	Low attenuation index (for emphysema-like burden); high attenuation index (for interstitial/fibrosis-like burden); lobe-level summaries	COPD, emphysema, interstitial lung abnormality, cardiopulmonary risk	Transforms qualitative lung texture into quantitative pulmonary risk biomarkers.
Liver attenuation	Liver attenuation index, including percentage of voxels below 40 HU	Fatty liver disease, insulin resistance, CVD, stroke, mortality, diabetes	Identifies hepatic steatosis as a systemic cardiometabolic risk marker without additional scanning.
Adiposity	Epicardial fat, upper thoracic visceral fat, subcutaneous fat volumes or indices	Diabetes, CHD, HF, cardiometabolic risk	Quantifies regional fat depots with differential metabolic and cardiovascular implications.
Skeletal muscle and BMD	Thoracic skeletal muscle volume and attenuation; vertebral trabecular BMD with T-score/Z-score	Myosteatosis, sarcopenia, frailty, diabetes, COPD, osteoporosis	Adds body-composition and skeletal health information to metabolic risk assessment.

The table groups related outputs; a given report may include additional quantitative sub features depending on scan type, field of view, reconstruction, and regulatory/clinical configuration.
Abbreviations: AI-CAC: artificial intelligence coronary artery calcium; CAC: coronary artery calcium; CAD: coronary artery disease; CHD: coronary heart disease; ASCVD: atherosclerotic cardiovascular disease; LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle; BSA: body surface area; HF: heart failure; AF: atrial fibrillation; AVC: aortic valve calcification; CVD: cardiovascular disease; CT: computed tomography; HU: Hounsfield unit; COPD: chronic obstructive pulmonary disease; BMD: bone mineral density.

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Table 2. Evidence Map Across AI-CVD Components

Domain	Study/source	Cohort	Key result	Implication
AI-CAC / Agatston-2.0	Agatston-2.0 Part I [11]	MESA + FHS; conventional CAC=0	AI-CAC >0 in 21.7%; 20-year CHD 7.7% vs. 3.8% in those with AI-CAC=0; adjusted HR 1.71 for CHD.	Shows how AI-CAC can identify those with earlier or borderline CAC who are at higher CHD risk that the traditional Agatston score may score 0.
All CVD prediction	NPJ Digital Medicine AI-CAC [13]	MESA; 5,830 participants; 15 years	AI-CAC outperformed Agatston for CVD prediction across 1, 5, 10, and 15 years.	Shows CAC scan contains actionable risk information beyond Agatston score.
AF and stroke	JACC Advances LA volumetry [16]; JCCT LA AF [14]; Stroke LA/ratio metrics [15]	MESA with paired CAC/MRI; 15 years	AI-LA volume predicted AF and stroke comparably to MRI LA volume; AI-derived LA volume index and LA/RA and LA/LV ratios predicted long-term AF and stroke; AI-LA improved CHARGE-AF/NT-proBNP reclassification.	Supports atrial remodeling assessment from CAC scans.
Heart failure	JCCT LV/HF [17-18]; chamber/diabetes analysis [20]; AI-CVD-HF [10]	MESA and FHS analyses	AI chamber volumes predicted HF comparably to MRI, enlarged chambers identified HF risk comparable to diabetes in selected groups, and AI-CVD-HF outperformed PREVENT-HF.	Supports endpoint-specific HF prevention model from CT biomarkers.
Valve disease	FHS AVC abstract [32]	FHS Offspring; 742 participants	AI-CVD AVC AUC 0.934 for aortic stenosis vs. 0.883 for manual measurement.	Supports automated opportunistic valve calcium reporting.

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Liver and diabetes	BMJ Open DRC liver [35]; Diabetology & Metabolic Syndrome T2DM [36]	MESA; 15-23 years	High LAI associated with CVD, stroke, mortality, and incident T2DM; adiposity metrics also predicted T2DM.	Adds metabolic risk assessment beyond BMI, glucose, and CAC.
Muscle/lung and COPD	RSNA myosteatosis/COPD [34]	MESA; 5,535 participants; 20 years	Myosteatosis predicted COPD independently and more strongly than emphysema-like lung attenuation.	Adds systemic vulnerability and body-composition risk phenotyping.
Bone health	JACR AutoBMD [37]; EJRO AutoBMD [38]	MESA and paired cardiac/lung CT cohorts	AutoBMD correlated with manual BMD and upper thoracic vs. full thoracic AutoBMD were equivalent.	Enables osteoporosis/osteopenia assessment without additional radiation.

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Figure Legends

Graphical Abstract. AI-CVD comprehensive cardiovascular and metabolic risk assessment. The platform extracts multiple disease-relevant imaging biomarkers from a single CAC or chest CT scan in illustrating who to treat, including those coronary artery disease, heart failure risk, atrial fibrillation risk, stroke risk, aortic and mitral valve calcification, fatty liver disease, osteoporosis, myosteatosis, emphysema , pulmonary hypertension risk, and aortic aneurysm risk, and what to treat being the risk factors we focus on, including dyslipidemia, hypertension, hyperglycemia, and other factors.

Figure 1. Schematic overview of the AI-assisted coronary artery calcium (Agatston-2.0) quantification framework. Step 1: Automated deep learning–based segmentation of the coronary artery tree, including the left main, left anterior descending, left circumflex, and right coronary arteries. Step 2: AI-assisted intensity calibration and noise normalization using physical phantom measurements when available, or patient-derived reference tissues when phantoms are absent. Step 3: Voxel-wise relative density weighting using a continuous probabilistic transformation to enhance sensitivity to low-burden and small calcifications, particularly in thin-slice CT acquisitions. Spatial near-neighbor filtering is applied to suppress noise and aggregate biologically coherent calcification, with the final AI-CAC score computed as the sum of all weighted voxels within the coronary artery mask. Right panel: Comparison of conventional Agatston-1.0 and Agatston-2.0 approaches. Traditional Agatston-1.0 relies on thick-slice CT (~3.0 mm), which may miss small or low-density calcifications due to partial volume effects. In contrast, Agatston-2.0 leverages thin-slice imaging (~0.6 mm) and probabilistic voxel-wise weighting to improve detection of small and early calcifications, thereby enhancing sensitivity to subclinical coronary artery disease. Cumulative incidence of coronary heart disease stratified by AI-CAC status among participants with CAC= 0 at baseline. Kaplan–Meier curves show the cumulative incidence of coronary heart disease (CHD) during follow-up in (A) the Multi-Ethnic Study of Atherosclerosis (MESA), (B) the Framingham Heart Study (FHS; Generations 2 and 3), and (C) the pooled cohort. Analyses were restricted to participants with CAC = 0 at baseline and stratified by AI-CAC status (AI-CAC score= 0 vs AI-CAC score > 0). The dashed blue line represents all participants with CAC = 0, the green line represents participants with AI-CAC score = 0, and the red line represents participants with AI-CAC score > 0. Numbers of participants in each group are shown in the legends. P values were calculated using the log-rank test. Adapted from Naghavi et al.[11]

Figure 2. Case examples of patients with CAC=0 or low calculated ASCVD risk who have enlarged cardiac chambers by AI-CVD.

Figure 3. Time-Dependent ROC AUC for 15-Year Atrial Fibrillation and Stroke Prediction (A) AI-CAC and CMRI LA demonstrated nonsignificant difference for prediction of 15-year AF. AI-CAC LA showed significantly greater discrimination for 15-year AF than Agatston CAC score and NT-proBNP. (B) AI-CAC and CMRI volumetry demonstrated nonsignificant difference for prediction of 15-year stroke. AI-CAC volumetry showed significantly greater discrimination for 15-year stroke than Agatston CAC score and NT-proBNP. Adapted from Naghavi et al.[14,17]

Figure 4. Cumulative Incidence of CVD Events over 15 Years Stratified by Quartiles of Liver Attenuation Index. Adapted from Naghavi et al.[35]

Figure 5. Cumulative Incidence of CVD Events by Top Quartile of AI-CVD Components. Adapted from Naghavi et al.[41]

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Disclosures: Drs. Wong, Mechanic, Agatston, and Kloner are advisors to Heart Lung AI. Mr. Atlas, Mr. Zhang, and Drs. Mirjalili, Zarei, Masrouri, and Naghavi are employees of Heart Lung AI.

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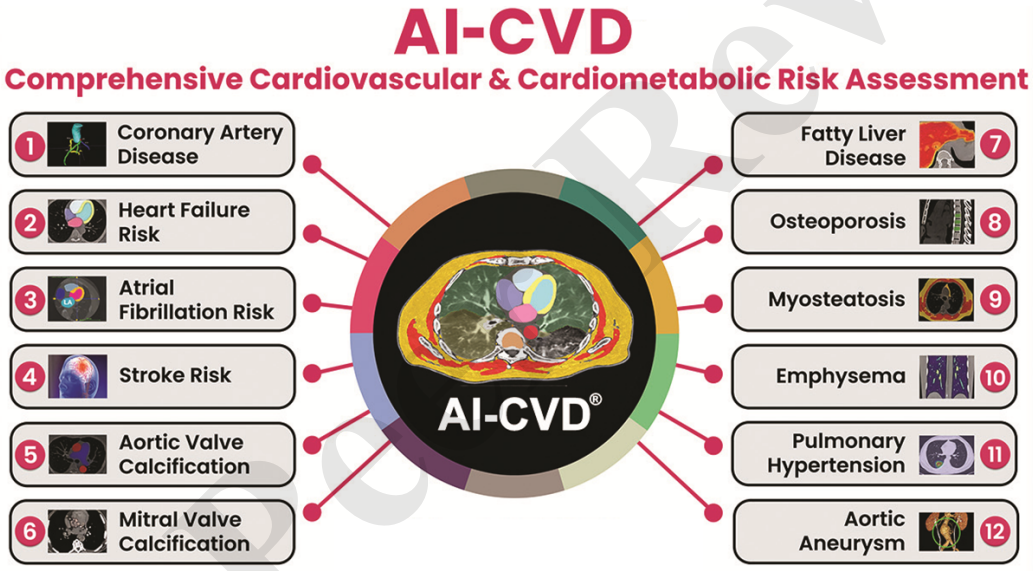
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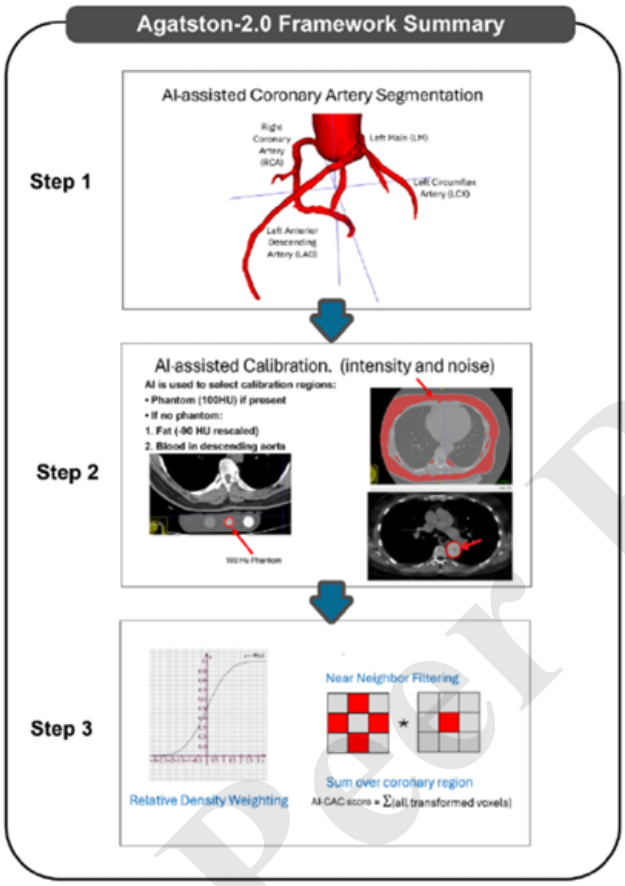
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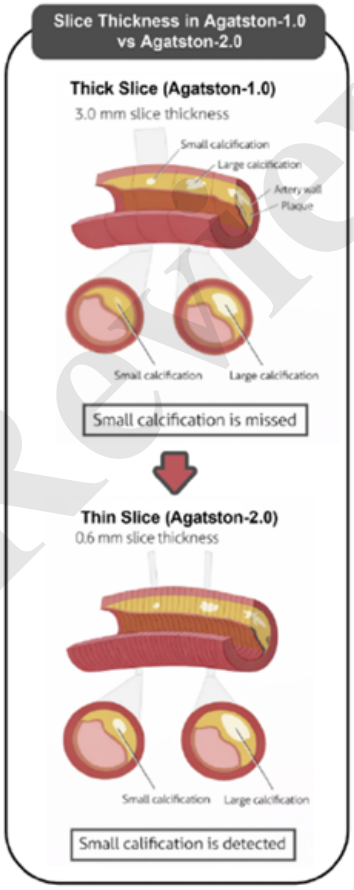
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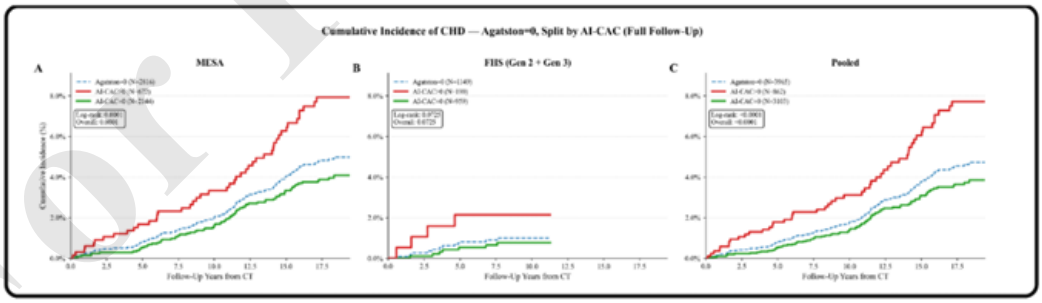
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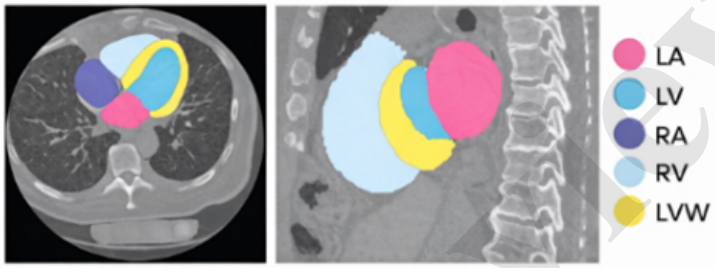
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Case Example 1

Female
Age: 57
CAC Score: 0
10-Year ASCVD Risk: 1.4%
(Low Risk)

LA Volume (cc): 84.6
LV Volume (cc): 121.7
CTR: 0.5

This case developed HF and Afib.

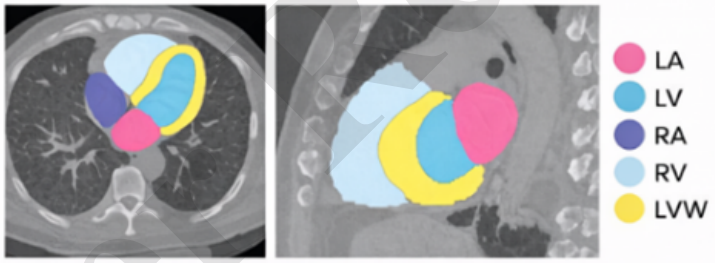


Case Example 2

Male
Age: 55
CAC Score: 0
10-Year ASCVD Risk: 4.8%
(Low Risk)

LA Volume (cc): 82.2
LV Volume (cc): 132.5
CTR. 0.48

This case developed HF.

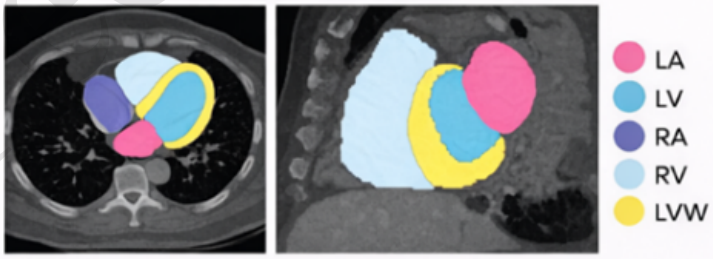


Case Example 3

Female
Age: 60
CAC Score: 0
10-Year ASCVD Risk: 4.1%
(Low Risk)

LA Volume (cc): 76.2
LV Volume (cc): 117.1
CTR. 0.49

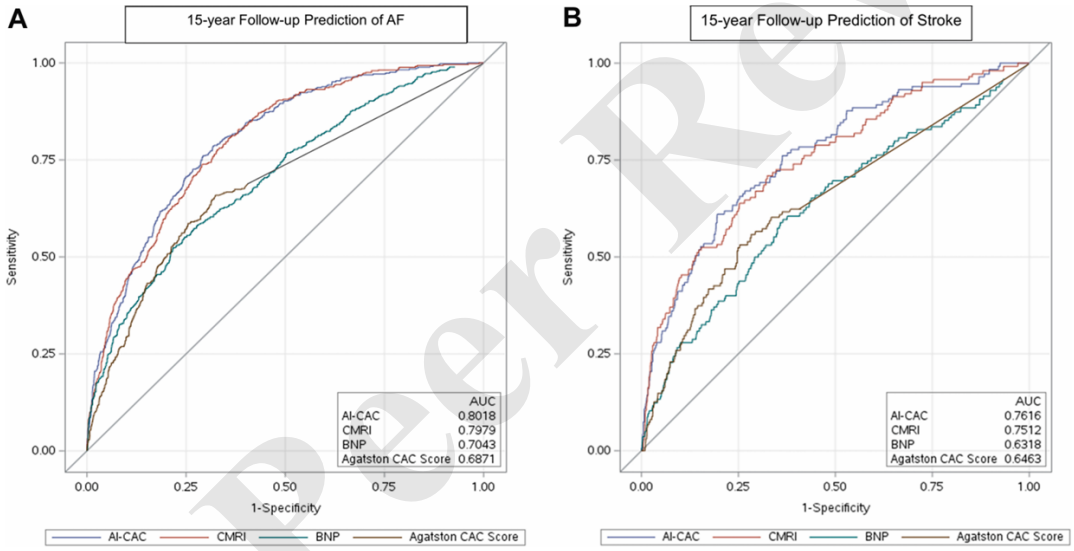
This case developed Afib and stroke.



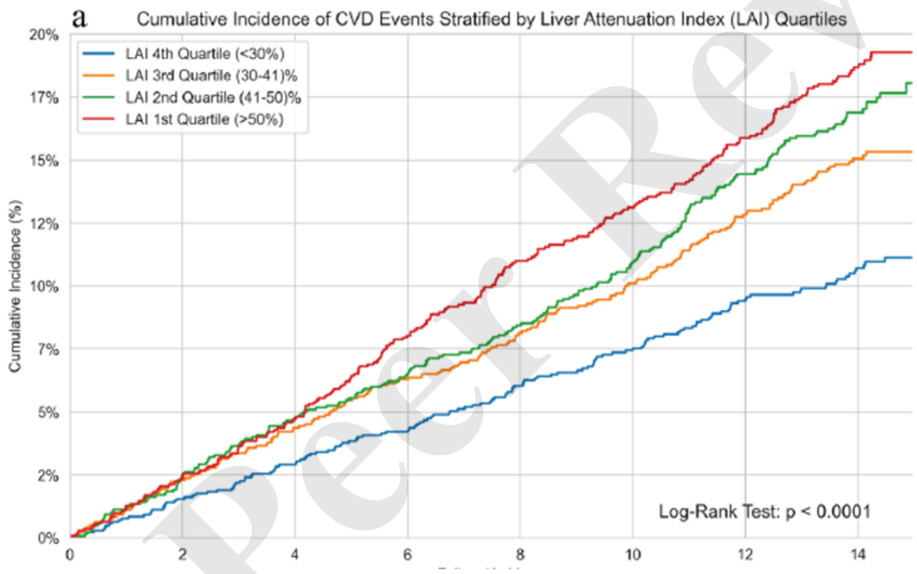
Abbreviations & Definitions

ASCVD	Atherosclerotic cardiovascular disease	LV	Left Ventricle
CAC	Coronary Artery Calcium	LV	Right Ventricular Wall
CTR	Cardiothoracic Ratio	RA	Right Atrium
LA	Left Atrium	RV	Right Ventricle

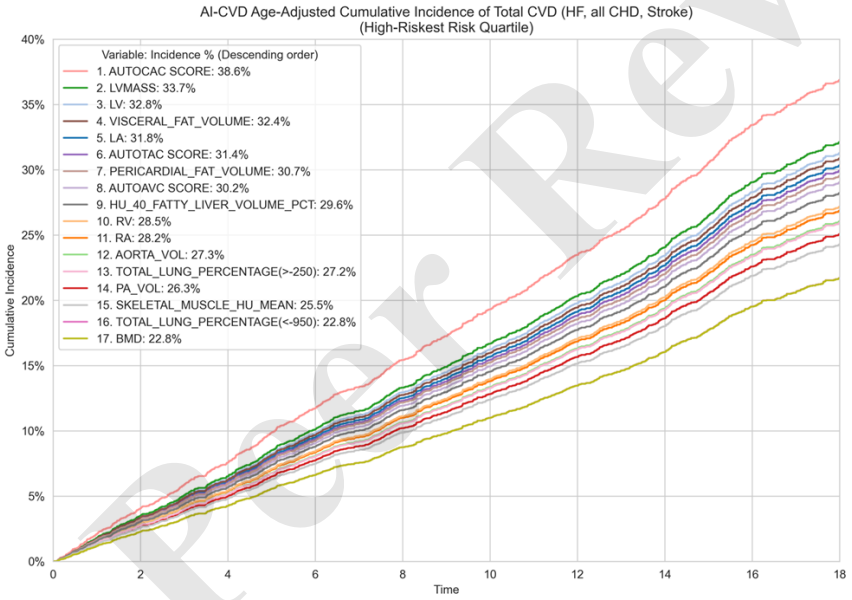
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