Breakthrough in the prevention of atherosclerosis

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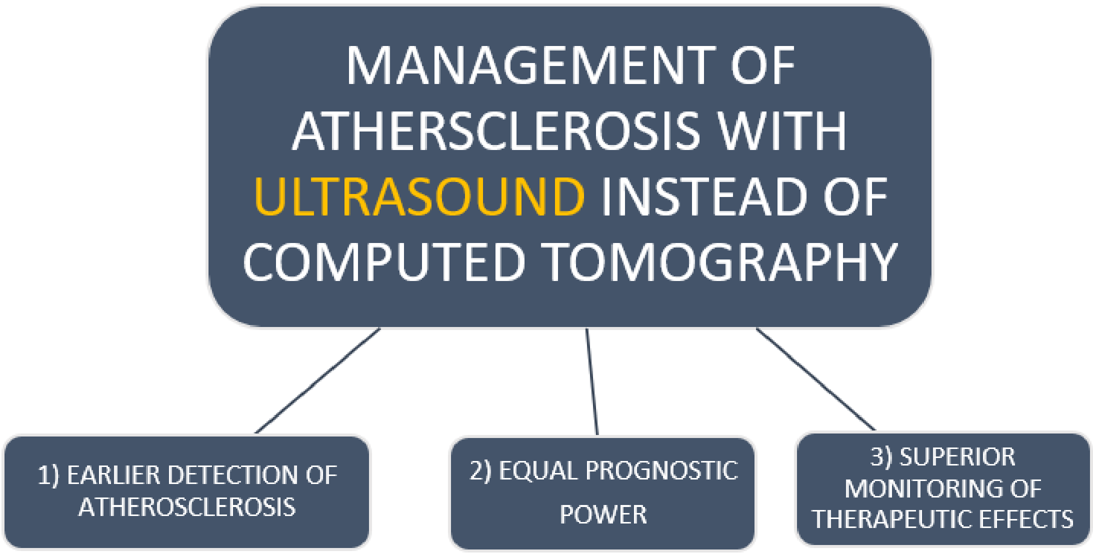
Background

People with atherosclerosis are now identified using imaging techniques and receive recommendations for the secondary prevention of cardiovascular disease. Imaging is therefore of great clinical importance for the risk management of atherosclerosis.

Thanks to Swiss research, the importance of imaging atherosclerosis with a simple ultrasound test in the diagnosis and management of atherosclerosis is being recognized and increasingly put into practice. Compared with the information obtained from computed tomography of the coronary arteries with and without contrast agents, recent research shows that ultrasound of the carotid arteries—with determination of the total plaque burden – 1) detects vulnerable patients earlier, 2) is prognostically equivalent to coronary CT, and 3) is clearly superior to coronary CT in terms of monitoring preventive effects, costs, and the lack of environmental impact and patient radiation exposure.

Current evidence allows us to conclude that in the secondary prevention of atherosclerosis, a paradigm shift away from computed tomography and toward carotid ultrasound is scientifically sound and thus the primary examination module for detecting prognostically relevant atherosclerosis.

Figure 1: Overview of atherosclerosis management



Research results

Part 1

Detection of atherosclerosis using imaging and categorization in primary or secondary prevention

The basic prerequisite for atherosclerosis management is the examination of the arteries using imaging techniques to determine whether secondary prevention is present. The specific procedure for reclassifying a person into secondary prevention is explained in the following example.

Figure 2: Communicating cardiovascular risk in the doctor's office

Source: Image generated by ChatGPT

First, the ASCVD risk, i.e., the risk of fatal or non-fatal heart attack and stroke, is assessed using a risk calculator (e.g., SCORE2/-OP [1]]) based on risk factors. This may be 5% in 10 years, for example (see Figure 2). This risk is considered high for a 50-year-old person, but low-moderate for a 70-year-old person. Since there is a 95% probability that no event will occur in the next 10 years, many people in this situation are understandably unwilling to take medication for the rest of their lives.

Imaging is used as a decision-making aid for or against drug treatment to answer the question of whether atherosclerosis is already visible (=secondary prevention due to diseased arteries) and how high the associated cardiovascular risk is, where lifestyle improvements alone may no longer be sufficient. In order to answer these questions correctly, event data for heart attacks and strokes as well as the associated diagnostic accuracy of the imaging procedures must be known (Table 1).

Table 1: Atherosclerosis quantification with coronary artery calcification (CAC) and carotid atherosclerosis (cTPA), associated diagnostic accuracy (AUC), test sensitivity and specificity, and associated risk in 5 years. Literature: ARCO [2]**and MESA study** [3]**.**

DIAGNOSTIC ACCURACY OF IMAGING (MESA CAC, ARCO cTPA)								
MESA Coronary Heart Disease								
	CHD	No CHD	ALL	Sens	Spec	AUC1	STANDARD ERROR	5 YEAR RISK
CAC 0	31	2330	2361	0.8072	0.5733	0.738	0.0189	0.7
CAC 1-99	44	1049	1093	0.8072	0.5733			2.1
CAC 100+	90	685	775	0.5422	0.8314			5.8
ALL	165	4064	4229					
MESA MACE						AUC2	STANDARD ERROR	5 YEAR RISK
CAC 0	54	2307	2361	0.7787	0.5788	0.715	0.0161	1.2
CAC 1-99	73	1020	1093	0.7787	0.5788			3.3
CAC 100+	116	659	775	0.54	0.86			7.5
ALL	243	3986	4229					
ARCO MACE						AUC3	STANDARD ERROR	5 YEAR RISK
TPA<22	3	1413	1416	0.9474	0.5074	0.798	0.0214	0.3
TPA 22-61	12	707	719	0.9474	0.5074			1.2
TPA 62+	42	665	707	0.7368	0.7612			6.4
ALL	57	2785	2842					

CHD: coronary heart disease = risk of heart attack
MACE: major adverse cardiac event = risk of heart attack or stroke

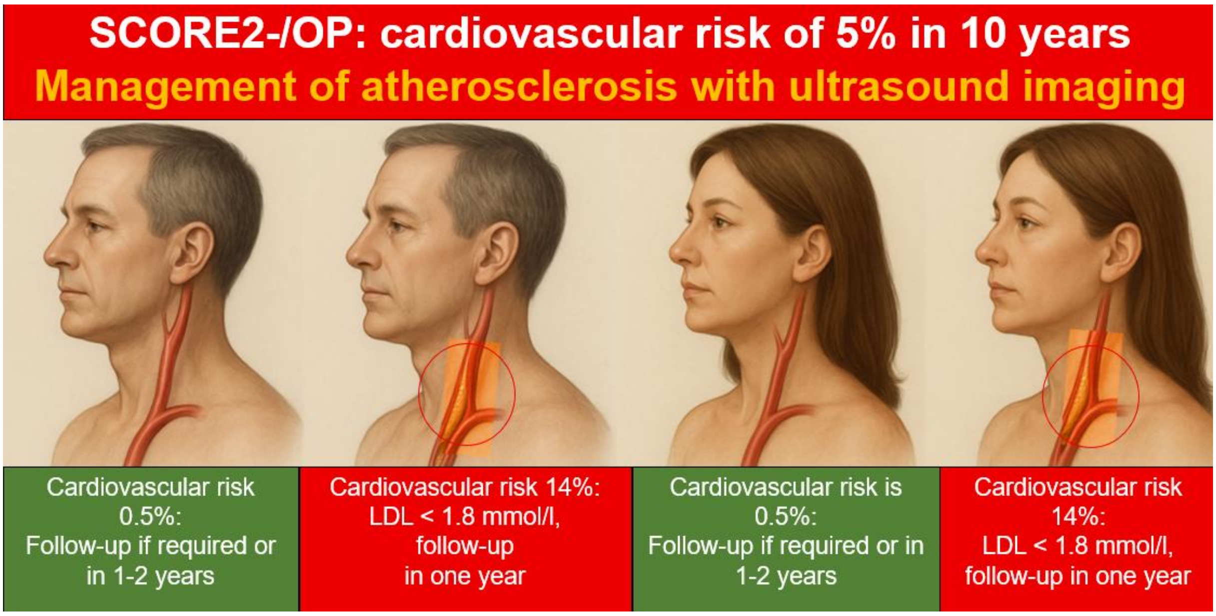
In a second step (Table 2), it is necessary to modify the cardiovascular risk according to SCORE2/-OP of 5% based on imaging, which is calculated using Bayes' theorem [4]. Following our example, in the absence of atherosclerosis in the imaging, the cardiovascular risk decreases from 5% to 2% with coronary CT and from 5% to 0.5% with no or only minimal carotid atherosclerosis of 0-21 mm^2. If, on the other hand, the test shows mild atherosclerosis, the risk increases from 5% to 9% with coronary CT and from 5% to 9% with carotid atherosclerosis of 22-61 mm^2. If, on the other hand, there is already at least moderate atherosclerosis, the cardiovascular risk increases from 5% to at least 17% with coronary CT and from 5% to at least 14% with carotid atherosclerosis of 62 mm^2 or more. The risk is therefore tripled, which naturally has implications for preventive medication.

Figure 3 explains the effect of imaging on preventive drug therapy based on LDL cholesterol. In individuals with no or minimal atherosclerosis, the SCORE2/OP risk determined on the basis of cardiovascular risk factors decreases by a factor of approximately ten from 5%, while the risk increases approximately threefold with significant evidence of atherosclerosis. If significant atherosclerosis is detected, patients are placed on secondary prevention, where a target range for LDL < 1.8 mmol/l is aimed for.

Table 2: Calculation of cardiovascular retest risk based on the results of cohort studies depending on the amount of plaque in CT (coronary calcification) and carotid atherosclerosis (cTPA, total plaque area in mm^2) using Bayes' theorem [4].

POST TEST RESULTS FOR POSITIVE AND NEGATIVE TESTS (BAYES THEOREM)					
IMAGING TEST NEGATIVE					
	Risk	Sens	Spec	Bayes neg	Risk change
MESA CHD	0.05	0.807	0.5733	0.017	0.033
MESA MACE	0.05	0.779	0.5788	0.020	0.030
ARCO MACE	0.05	0.9474	0.5074	0.005	0.045
IMAGING TEST POSITIVE LOW RISK					
	Risk	Sens	Spec	Bayes pos	
MESA CHD	0.05	0.807	0.5733	0.091	0.041
MESA MACE	0.05	0.779	0.579	0.089	0.039
ARCO MACE	0.05	0.947	0.507	0.092	0.042
IMAGING TEST POSITIVE RELEVANT RISK					
	Risk	Sens	Spec	Bayes pos	
MESA CHD	0.05	0.542	0.8314	0.145	0.095
MESA MACE	0.05	0.540	0.86	0.169	0.119
ARCO MACE	0.05	0.737	0.761	0.140	0.090

Figure 3: Ultrasound of the carotid artery detects healthy and diseased arteries, i.e., the situation of primary (left) and secondary prevention (right).



Source: Image generated by ChatGPT

Summary: Imaging allows categorization into primary and secondary prevention, so it is not a purely risk-based assessment, but rather identifies individuals with arterial disease. The result is easier to communicate and motivates affected individuals to adhere to the target values for effective prevention.

Research results

Part 2

Earlier detection of atherosclerosis with ultrasound compared to coronary calcification: Varifo study and comparative studies

Varifo study:
Diagnostic and therapeutic differences in cardiovascular risk assessment using SCORE2/-OP, carotid plaques, and coronary calcifications [5]

In this Varifo study, which was presented at the Cardiology Congress in Zurich in June 2025 (rapid fire communication), the age- and sex-specific prevalence of total carotid plaque area (cTPA) and coronary calcium Agatston score (CAC) was compared in 907 patients (age 59.9 ± 9.4, 34% female). This is therefore probably the largest direct comparison study in Switzerland.
The prevalence of atherosclerosis (cTPA>21mm^2; CAC>10) increased with age. Between the ages of 30 and 55, the prevalence of carotid plaque was 18%–23% higher than the prevalence of coronary calcification (Figure 4).

Figure 4: Direct comparison of the prevalence of carotid plaques (cTPA method) and coronary calcifications (computed tomography) by age group.

Prognostic comparison of cTPA with coronary calcium for cardiovascular risk stratification

varifo
vascular risk foundation

Results (1b): Prevalence by age classes at baseline

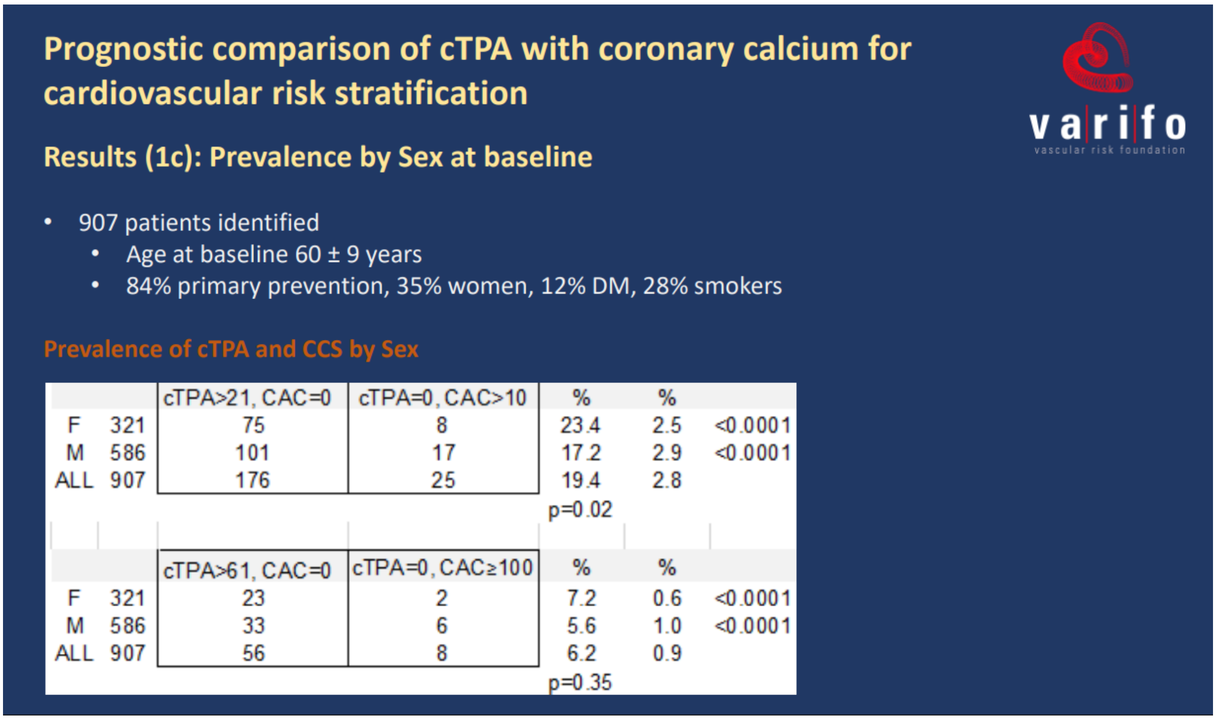
- 907 patients identified
  - Age at baseline 60 ± 9 years
  - 84% primary prevention, 35% women, 12% DM, 28% smokers

Age Class	30-45	46-50	51-55	56-60	61-65	66-70	75-99
Number of patients per age group	39	90	124	186	181	130	157
Prevalence cTPA ≥ 21 mm^2 (%)	41	66	60	60	76	81	89
Prevalence CAC ≥ 10 (%)	18	40	43	47	64	69	79
Prevalence difference (%)	23	26	18	13	13	12	10

The question of how often prognostically relevant atherosclerosis is missed is particularly important: the situation of cTPA>21 mm^2/ CAC=0 versus cTPA=0 mm^2 /

CAC>10 was 23.4% versus 2.5% in women ($p<0.0001$) and 17.2% versus 2.9% in men ($p<0.0001$), and the difference in prevalence was statistically higher in women ($p=0.02$). When comparing plaques with higher risk (TPA>62 mm^2 + CAC=0 or CAC100 + TPA=0 mm^2 showed similar distributions: 7.2% versus 0.6% in women and 5.6% versus 1.0% in men (both $p<0.0001$), and the difference between the sexes was $p=0.35$ (see **Figure 5).**

Figure 5: Direct comparison of the prevalence of carotid plaques (cTPA method) and coronary calcifications (computed tomography) by gender and severity of atherosclerosis.



		cTPA>21, CAC=0	cTPA=0, CAC>10	%	%	
F	321	75	8	23.4	2.5	<0.0001
M	586	101	17	17.2	2.9	<0.0001
ALL	907	176	25	19.4	2.8	
				p=0.02		

		cTPA>61, CAC=0	cTPA=0, CAC≥100	%	%	
F	321	23	2	7.2	0.6	<0.0001
M	586	33	6	5.6	1.0	<0.0001
ALL	907	56	8	6.2	0.9	
				p=0.35		

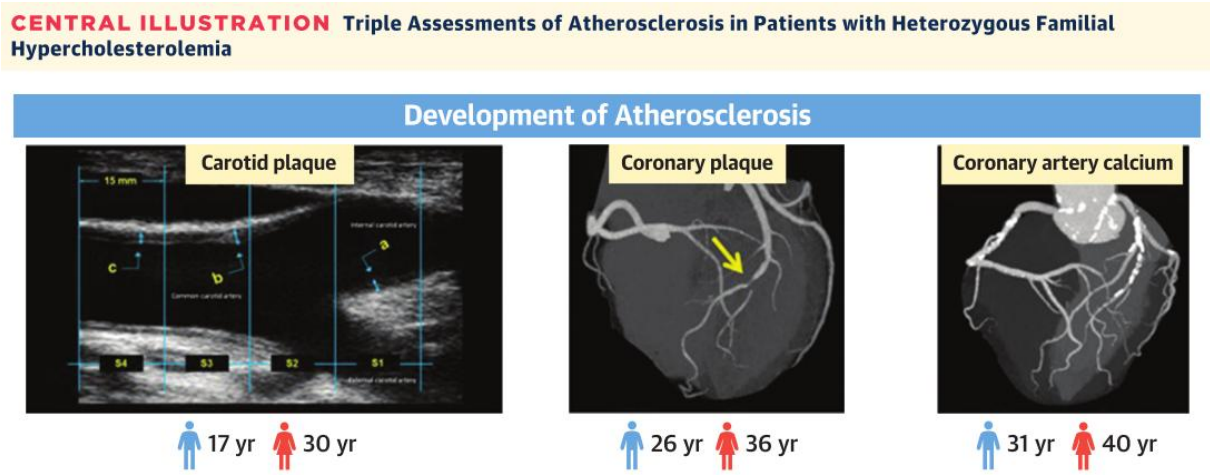
Comparative studies

Similar to our study, Gudmundsson stratified female and male subjects according to CAC and cTPA values in the **REFINE-Reykjavik study**. In the absence of CAC, the presence of any carotid plaque was found in 36% of women and 19% of men (Chi^2 = 5.01, p= 0.024 for the sex difference), while the absence of carotid plaque was found in 5.5% of women and 4.6% of men with CAC>0 (p NS for the sex difference). A significant proportion of CAC (e.g., CAC ≥100) without carotid plaque was present in only 1.4% of women and only 1.6% of men [[6].

In the **PESA study**, in which 4,066 patients were examined for the presence of carotid plaques and coronary calcification, evaluated by gender and with an average age of 45.8±4.3 years, the presence of carotid plaques was found in 36% of men (24% of women), but CAC was found in only 25% of men and only 5% of women [7].

In individuals with **familial hypercholesterolemia,** the authors calculated the average age for the onset of carotid plaques, coronary plaques, and coronary calcification, respectively, and found an age of 17, 26, and 31 years in male patients and 30, 36, and 40 years in female patients [8]. In familial hypercholesterolemia, coronary calcification occurred 14 years later in men and 10 years later in women than carotid plaques. Carotid plaques are also associated with a higher incidence of developing coronary calcification in the future [[9]

Figure 6: Direct comparison of the age of men and women with familial hypercholesterolemia at which carotid plaques, coronary plaques (non-calcified), and coronary calcifications first become visible



We and others have thus consistently shown that coronary calcification is less common or becomes visible later than carotid atherosclerosis. Our study provides further information on sex and age, with the difference in prevalence of underdiagnosed significant carotid atherosclerosis being higher in those under 55 years of age, especially in women, when compared to coronary calcification.

Conclusion: atherosclerosis is primarily visible in the carotid arteries and can therefore be treated at an early stage. The absence of coronary calcification, especially in people under the age of 55, is no guarantee of the absence of significant carotid atherosclerosis.

Prognostic significance of carotid atherosclerosis equivalent to coronary calcification

Varifo study:
Prognostic comparison of cTPA with coronary calcium for cardiovascular risk stratification

In this Varifo study, which was presented at the Cardiologists' Congress in Zurich in June 2025 (rapid fire communication) [[5]we used standard imaging techniques for the total area of carotid plaques (cTPA) and the coronary calcium Agatston score (CAC), which were measured between 2003 and 2024, conducted a follow-up examination of 472 cardiology patients (69% male, age 60 ± 9 years, 12% in secondary prevention) over 10 ± 7 years and observed 63 events (15 PTCA, 4 CABG, 11 AMI, 5 STROKE/TIA, 28 deaths). Using multivariate regression analysis with 7 clinical and 3 laboratory variables, the hazard ratio for cTPA>62 mm^2 was 1.42 (95% CI 1.03–1.95, p=0.03) and for CAC>100 was 1.74 (95% CI 1.39–2.19, p<0.0001). When using 3 categories of risk markers (low-medium, high, very high) in the Cox regression, the hazard ratio for the post-test risk for cTPA was 1.56 (95% CI 1.14–2.13, p=0.0053) and a hazard ratio of 2.26 (95% CI 1.61–3.16, p<0.0001) for CAC, while SCORE2/-OP was excluded from the model. The improvement in reclassification was not statistically significant for cTPA (NRI 3% ± 12%, p=0.76), but was significant for CAC (NRI 29% ± 11%, p=0.01). The area under the curve was statistically comparable for SCORE2/-OP, SCOTPA, and SCOCAC (0.64, 0.63, and 0.69, respectively, p=NS for the difference, see Figures 7, 8, and Table 3).

Figure 7: Varifo study on the prognostic significance of carotid atherosclerosis and coronary calcification in a long-term comparison of Kardiolab cardiology practices 2002-2024: Study basis

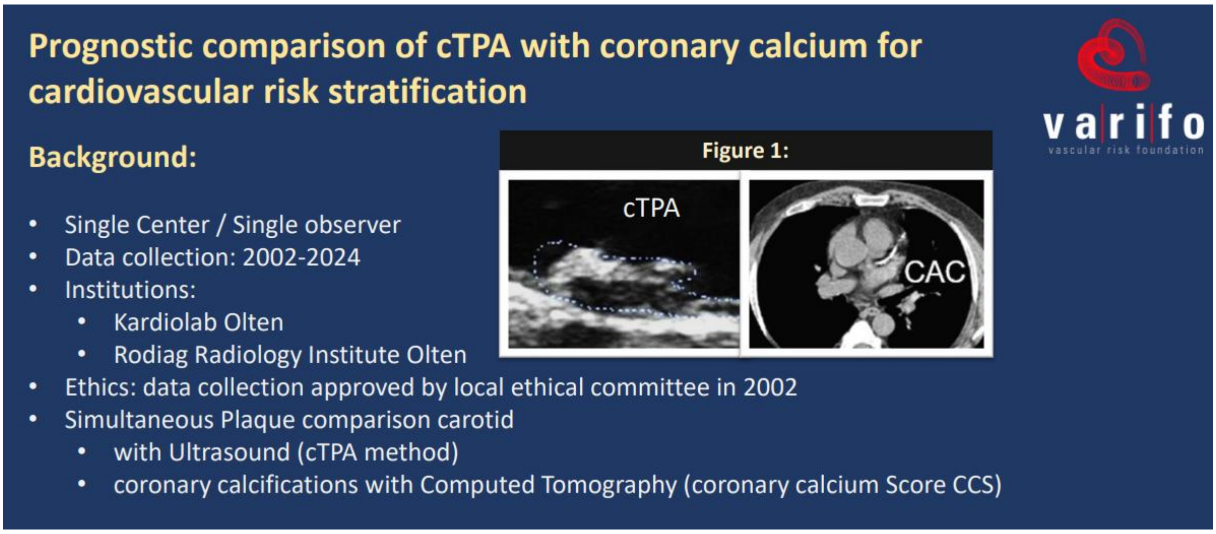


Figure 8: Varifo study on the prognostic significance of carotid atherosclerosis and coronary calcification in a long-term comparison of the Kardiolab cardiology practice 2002-2024: Results for AUC, Kaplan Meier survival curves

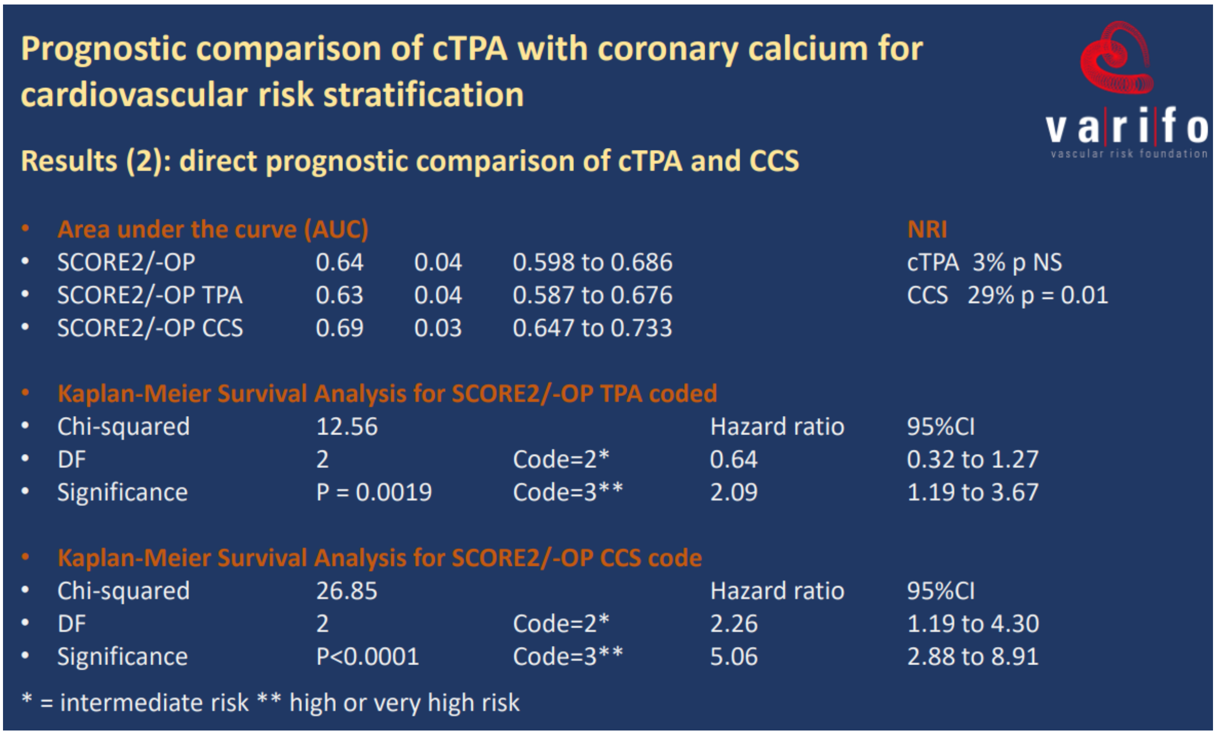


Table 3: Multivariate hazard ratios, univariate C-statistics, and net reclassification improvement (NRI) of SCORE2/-OP, SCORE2/-OP plus CAC code, and SCORE2/-OP plus cTPA code

N=473	HR (95% CI)	P value	C-statistic	P value	NRI ± SE
CVD or DEATH (N=63)					
SCORE2/-OP alone	---	---	0.64	0.0003	---
+ CACCode	2.26 (1.61-3.16)	<0.0001	0.66	<0.0001	29% ± 11%
+ cTPACode	1.38 (1.01-1.89)	0.0427	0.60	0.0082	3% ± 11%

Comparative studies

Coronary calcification has been extensively studied for its prognostic value in relation to serious adverse cardiac events [10], [11], but direct comparisons with the prognosis of myocardial infarction, stroke, or both have rarely been performed to date.

In the **MESA cohort** [12], CAC was compared with carotid plaque (in the carotid bulb and internal carotid artery) and carotid intima-media thickness (cIMT) in 6,779 patients with an average follow-up period of 9.5 years. Using Cox proportional hazards regression, the hazard ratio for CAC was 3.12 (95% CI: 2.44-3.99) and for carotid plaque 1.61 (95% CI: 1.17-2.21), while the results for cIMT were not statistically significant (1.20 [95% CI: 0.94-1.52]). Unfortunately, plaque within the common carotid artery was not examined, which reduces the prognostic significance of carotid plaque in this study.

In the **BIOIMAGE cohort** [13], CAC was compared with global carotid plaque burden using total carotid plaque volume (cTPV), a measure of plaque burden highly correlated with cTPA, in 5808 patients with a follow-up period of 2.7 years [[14]Using Cox proportional hazards regression, the hazard ratio for CAC was 2.87 (95% CI: 1.73-4.74) and for cTPA 2.97 (95% CI: 1.92-4.60).

In the REFINE cohort [6], CAC was compared with cTPA in 948 patients (509 women) with a mean follow-up of 6.5 years. Both cTPA and CAC provided similar prognostic information using different pretest models: cTPA AUC was 0.77 (95% CI: 0.71–0.83, $p=0.019$) and CAC AUC was 0.80 (95% CI: 0.74–0.86, $p<0.0001$). Our study expands the knowledge about the prognostic ability of cTPA and CAC over the long term.

Conclusion: Using the total burden of carotid plaques (Varifo-Kardiolab cohort, Bioimage cohort, REFINE cohort), the prognostic information provided by carotid atherosclerosis is equivalent to that provided by coronary calcifications.

Monitoring of therapeutic effects on the development of carotid atherosclerosis over time and correlation with cardiovascular events

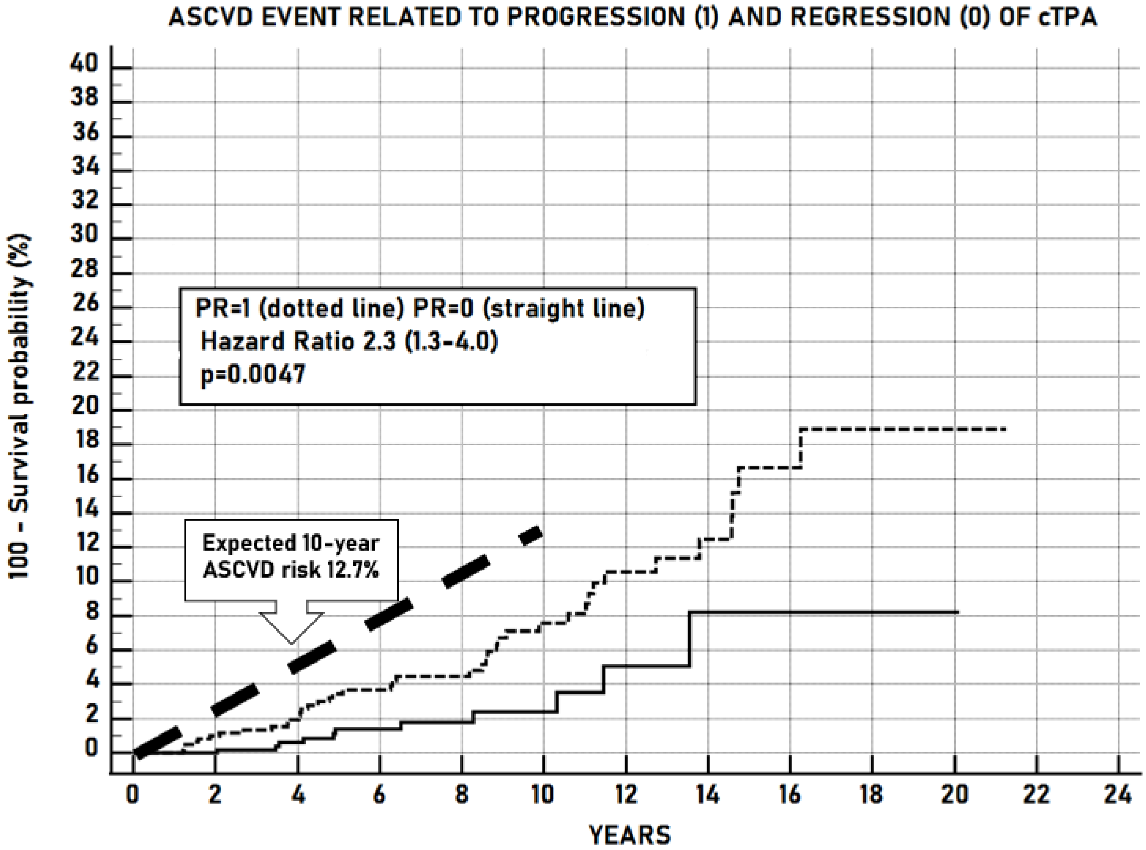
Varifo study:
Carotid plaque progression and regression: an observational long-term study on atherosclerosis management

Varifo study [[15]presented at the Cardiology Congress in Lausanne in June 2024 (rapid fire communication): monitoring plaque burden over time in an outpatient cardiology facility with regular visits can help identify patients who need more intensive preventive care. A population of 1,224 patients (average age 57 years, 38% women) underwent repeated vascular ultrasound examinations to quantify plaque burden in the carotid artery (cTPA) every 1.3 years on average, with a median cardiovascular follow-up of 6.5 years. Patients who did not achieve treatment goals or whose plaque burden increased were advised to intensify preventive treatment. Expected cardiovascular events were calculated based on a previous, untreated cohort [[2]and compared with the observed events. Hazard ratios were calculated for plaque progression and regression. We found the following results: during the follow-up period, the following 52 cardiovascular events were recorded in the progression and regression groups: heart attack 7 versus 1, stroke/TIA 7 versus 4, aortocoronary bypass surgery 7 versus 1, coronary stenting 14 versus 4, death from any cause 1 versus 0, coronary heart disease or peripheral arterial disease 5 versus 1, and total events 41 versus 11, or 6.7% versus 1.8% ($p<0.0001$). The mean SCORE2/-OP risk was lower in the regressors ($p=0.0009$) and the hazard ratio of patients with plaque progression versus plaque regression was 3.54 (95% CI: 1.80-7.00, $p=0.0003$). In the multiple regression analysis, the most recently measured SCORE2/-OP risk was significantly associated with the cTPA difference between patients with and without plaque progression (F-ratio 25.69, $p<0.0001$).

Monitoring carotid plaque over time is an additional tool for observing the effects of preventive therapy in modern cardiology. It appears to lead to a regression of carotid atherosclerosis and a clinically relevant reduction in ASCVD event rates by around 50% compared to patients with plaque progression. The absence of fatal events in our study suggests a clinical effect of preventive medical therapy on overall mortality. In patients with cTPA levels that increase over time, more intensive preventive medical evaluation and intervention may be warranted, involving enhanced preventive medical measures or diagnostic tests (e.g., Lp(a), homocysteine, and renal function).

In our opinion, serial carotid ultrasound is currently the most reliable test for monitoring atherosclerosis over time.

Figure 9: Kaplan-Meier survival curves for cTPA progressors (dotted line) and regressors (straight line) in 1,224 patients. The estimated 10-year ASCVD risk, calculated from SCORE2/-OP and the post-test risk of an untreated cohort [[2]is also shown



Comparative studies

As early as 2002, **Spence** et al. demonstrated that the cTPA method (Figure 10) was a tool for monitoring the success of preventive vascular therapy in patients at his clinic for atherosclerosis prevention in Ontario, Canada [16].

Figure 10: Original work by Spence [16]**showing a plaque in the common carotid artery (total area of this relatively large plaque 55 mm²).**

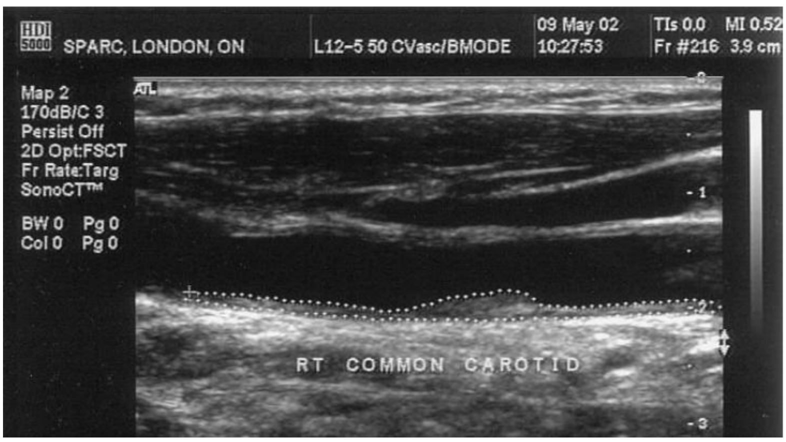
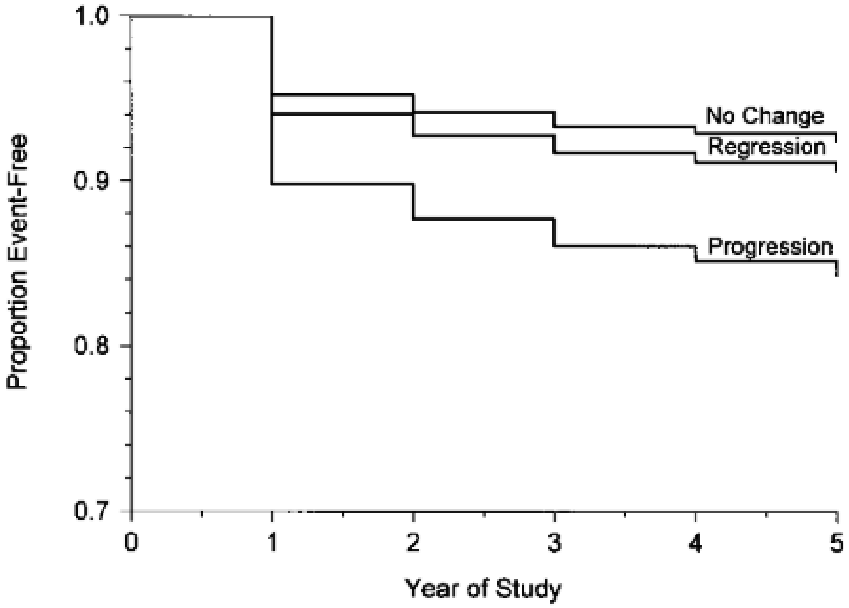


Figure 1. Measurement of carotid plaque area. Each plaque was measured in a longitudinal view in the plane in which the plaque is maximal. Image was frozen and magnified on the screen, and a cursor was traced around the perimeter of the cross section. Microprocessor in the duplex scanner displays cross-sectional area of plaque (cm²). Plaque shown is in the right common carotid artery and measures 0.55 cm².

In 1,686 patients with a mean observation period of 2.5 years, cardiovascular events occurred in 9.4% of patients with plaque regression and in 15.7% of patients without plaque regression (Figure 11), which corresponds to a relative risk reduction of around 40%.

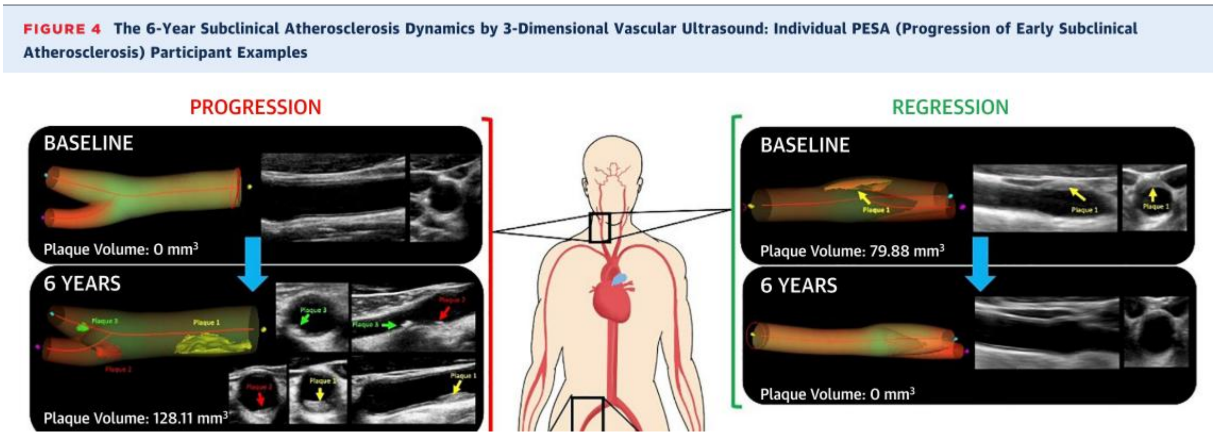
Figure 11: Original work by Spence [16]**showing Kaplan-Meier survival curves for patients with plaque regression or plaque stabilization and plaque progression**



Spence et al. showed in 4,378 patients observed over a period of 10 years that intensified drug prophylaxis could reduce cTPA, with plaque regression occurring in up to 50% of patients and plaque stabilization in 31% of patients, while in patients receiving the usual treatment, 62% of patients experienced plaque progression [[17].

Mendieta et al. measured the global carotid plaque volume in 3,471 patients over a period of 6 years and found plaque regression in 8% of patients, with the extent of regression in global plaque volume over time reaching 100% [18]. The authors stated: "Over a period of 6 years, subclinical atherosclerosis progressed in one-third of asymptomatic middle-aged subjects. Regression of atherosclerosis is possible in the early stages of the disease. The influence of LDL-C and SBP on the progression of subclinical atherosclerosis was more pronounced in younger participants, suggesting that the prevention of atherosclerosis and its progression could be improved by stricter control of risk factors at a younger age, which would likely have a long-term effect on reducing the risk of clinical events."

Figure 12 (from [18]**): Imaging examples of plaque progression and plaque regression in the carotid arteries over 6 years.**



Fuster et al. found significantly lower overall mortality in 732 patients aged 68 years with a mean follow-up of 8.9 years when plaque regression was observed (log-rank p=0.04) [19]. They also showed that carotid artery ultrasound is better than coronary calcification at predicting cardiovascular events.

In a **Korean cohort study** of 650 patients (47% with plaque progression), the incidence of heart attack or stroke over 4 years was significantly higher (26.6%) in the group with carotid plaque progression than in the group without progression (13.0%, HR 2.19, (95% CI: 1.52-3.15; p<0.001), which again corresponds to an approximately 50% lower risk with plaque regression [[20]

Conclusion: As early as 2002, Spence showed in a groundbreaking study that regression of carotid atherosclerosis is associated with an approximately 40% lower risk of cardiovascular events. Later observations, in line with our practice-based cohort study, show that a roughly 40% to 50% risk reduction can be expected with plaque stabilization and plaque regression. The paradigm shift toward intensified preventive treatment for plaque progression ("treating arteries instead of risk factors" [[21] **has been confirmed by several studies over the past 20 years.**

Discussion

The research results of the Vascular Risk Foundation over the last 23 years are based on consistent data collection at the Study and Cardiology Center in Olten and our partner center in Koblenz, Germany (Dr. med. Ansgar Adams):

1. We have demonstrated that the prognostic significance of plaque burden in the carotid arteries (cTPA method) is comparable to the results of coronary calcification in our cohort study [2]. A direct comparison of the prognostic significance of carotid atherosclerosis and coronary calcification also showed comparable results in our study and other studies.
2. A direct comparison of imaging of atherosclerosis in the carotid arteries using ultrasound and in the coronary arteries using computed tomography has shown, in agreement with our [5], [15]and other studies, that atherosclerosis occurs first in the carotid arteries, which means that atherosclerosis can be identified and treated earlier.
3. Also innovative are the studies on the regression of atherosclerosis in the carotid arteries, where, in accordance with the literature, we found a roughly 50% lower cardiovascular risk in patients with plaque regression [15].

In line with the prevailing paradigm, which advocates the earliest possible start of preventive treatment [22], the presence of carotid plaques in the absence of coronary calcification offers a valuable opportunity to initiate treatment earlier. In terms of cardiovascular consequences, the long-term hazard ratio (HR) for patients with carotid plaque but without coronary calcification is 1.66 (95% CI: 1.04-2.66), which is prognostically relevant [[9].

After an average observation period of 16 years, individuals with carotid plaque had a higher incidence of developing coronary calcification at follow-up examinations [23]. In the long-term follow-up of the Nixdorf Recall Study, women with a baseline CAC=0 had an incidence of cardiovascular events of 21% (men: 9%) [[24]

As a primary imaging method for assessing cardiovascular risk, the cTPA ultrasound method is prognostically equivalent to coronary calcification and diagnostically superior (earlier detection of a cardiovascular risk situation).

Determining cardiovascular risk using the AGLA score or SCORE2/-OP calculator often poses a motivation problem in everyday clinical practice, as in most cases there is a 95% chance that no heart attack or stroke will occur in the next 10 years. In this situation, if there is no evidence of prognostically relevant carotid atherosclerosis (cTPA < 22 mm^2), the cTPA test allows a low cardiovascular risk to be documented, thus limiting prevention recommendations to primary prevention. On the other hand, if the cTPA test shows prognostically relevant carotid atherosclerosis, the patient is in secondary prevention with the necessary adjustment of prevention recommendations.

The cTPA test is particularly cost-effective as additional information in lipid treatment [[25]and increases the correct allocation of lipid-lowering medication.

The decisive advantages of the cTPA method compared to coronary calcification measurements are as follows:

1. **No costs:** only consultation costs are incurred (the test usually takes less than 2 minutes and can be performed in any doctor's office with a linear ultrasound probe).
2. **Two-tier healthcare:** The coronary calcium test must be paid for privately, which creates a two-tier healthcare system.
3. **No radiation exposure:** Coronary calcification is associated with radiation exposure of just under 1 mSv.
4. **cTPA gatekeeper for cardiac CT:** The measurement of coronary calcification using computed tomography should only be performed after the cTPA measurement, i.e., if relevant coronary calcification is suspected despite a normal cTPA measurement.
5. **No environmental impact:** Compared to computed tomography, the cTPA test requires virtually no resources (minimal power consumption, no environmental damage).
6. **Follow-up checks:** cTPA can be used to determine the progression or regression of atherosclerosis after just 3 months [21], which is a strong motivator for adhering to preventive measures. After a short training session, it can be easily performed in a point-of-care situation, i.e., in a general practitioner's office (like blood pressure or blood sugar checks). whereas such observations using computed tomography are prohibitive due to the costs and radiation exposure (at least 4 mSv per computed tomography scan for contrast-enhanced examination of non-calcified plaque load per examination) and, in addition, coronary calcifications increase under preventive treatment [[26]
7. **False sense of security:** around 25% of women in particular have relevant carotid plaques without coronary calcification and are also more likely than men (25% versus 9%) to suffer cardiovascular events (Nixdorf Recall Study [24]).

Outlook

Authorities and healthcare institutions should promote and support the learning of the cTPA method. The Varifo Foundation is working on appropriate concepts and is happy to share its knowledge.

Summary

The need for cardiovascular risk assessment with the detection of patients with atherosclerosis using imaging for the early identification of individuals who are already in secondary prevention is a breakthrough in the management of the number one widespread disease, atherosclerosis.

The paradigm of risk stratification for intermediate risk according to SCORE2/-OP, using computed tomography as the primary test in healthy individuals, which is the goal in cardiology for 2025, is clearly not sensible based on the scientific data and the seven main arguments in the discussion section.

Carotid atherosclerosis, measured with the cTPA test, should be favored as the primary examination method and widely implemented. In unclear situations, computed tomography can be used as a supplementary test if necessary.

This would save massive costs in the detection of prognostically relevant atherosclerosis and limit the diagnostic radiation exposure of the population (including the risk of overdiagnosis due to incidentalomas) to a necessary minimum.

The cTPA test allows the effect of the measures taken, including longevity and rejuvenation treatments, on the biological effects of atherosclerosis to be assessed after only around three months of preventive intervention [27].

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