



CLINICAL GROUP

Observational Study

Relationship between Atherosclerosis and Cancer: An observational outcome study

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Abstract

Background and aims: It was investigated whether there is a relationship between advanced atherosclerosis of the carotid artery and cancer.

Method: The carotid Total Plaque Area (TPA), the maximum plaque thickness, and the cardiovascular risk based on Framingham calculator using body-mass-index were determined in healthy subjects using ultrasound.

We compared the outcome in subjects with advanced atherosclerosis of the carotid artery (type III-IV b finding) with and without statin treatment. The follow-up was collected as part of occupational health check-ups.

Result: In 4207 healthy men aged 35-65 years (50 ± 8 years) we found 578 subjects with advanced atherosclerosis of the carotid artery (type III-IV b finding). A follow-up was available for 566 (97.9%) subjects. The average follow-up time was 48 months (range 0 to 139 months). Ten subjects were excluded because of missing data on treatment. A total number of 230 subjects was treated with a statin, 326 received no statin. Within the group of these subjects (54 ± 6 years), 33 men died (15 cancer, 4 strokes, 11 heart attacks, 1 COPD, 1 sepsis, 1 cirrhosis of the liver).

In the group with CVD death, two subjects were treated with a statin, 13 were untreated. In the group with cancer death, two subjects were treated with a statin, 13 were untreated. The death rate because of cancer and CVD was 1.7% for the treated subjects and 8.0% for the untreated subjects.

In the group with low risk (n= 1890), six men died (2 traffic accidents, 1 aneurysm bleeding, 2 cancer, 1 unclear, a follow-up examination has been completed for 43.9% (n= 830) of the subjects, mean follow-up time of 65 months (0-137 months). One man died (Parkinson) within the group with intermediate risk. In this group (n= 482) a follow-up examination has been completed for 55% (n= 265) with a mean follow-up time of 55 months (total range: 0-130 months). In the reference group (no atherosclerosis; n= 1257) follow-up has been completed in 38.3% (n= 482) with a mean follow-up time of 60 (0-145) months and nobody died.

In 3203 healthy women aged 35-65 years nobody died of cancer.

Conclusion: The development of cancer in men with advanced atherosclerosis of the carotid artery appears to be noticeable. The question of whether there is a causal relationship requires further investigations.

Abbreviation

TPA: Total Plaque Area (carotid plaque)

Introduction

Atherosclerosis imaging of the carotid artery can improve the risk prediction for cardiovascular disease [1-11] and statin

therapy in the subclinical stage improves the prognosis [11-17]. Cancer and atherosclerosis share risk factors such as diabetes, smoking, and obesity. This can be an explanation for the association of increased plaque burden on the carotid artery and cancer [18]. Smoking is an important risk factor for advanced atherosclerosis. In our cohort, the mean TPA in all age groups for smokers is about twice as high as that one for those who never smoked.



Materials and methods

In 4207 healthy men aged 35–65 years, the Total Plaque Area (TPA), the maximum plaque thickness, and the cardiovascular risk based on Framingham calculator using body-mass-index [19] were determined in an occupational medical setting using ultrasound. The carotid plaque surface was imaged with a high-resolution ultrasound linear transducer probe (10.0MHz). The measuring method was described in earlier publications. Four plaque types were classified, with type I and II a representing low cardiovascular risk, type II b and IV a representing intermediate risk, and type III and IV b representing high risk [7,8]. We defined the following categories based on the TPA and maximum plaque thickness: no measurable atherosclerosis and increasing tertiles for those with atherosclerosis according to the risk stratification [9].

We compared causes of death for subjects with advanced atherosclerosis of the carotid artery (type III-IV b finding) with and without statin treatment. The follow-up was collected as part of occupational health check-ups. The evaluation of all investigations was carried out with the approval of the

responsible ethics committee. This study was conducted in compliance with the ethical standards of the responsible institution on human subjects as well as with the Helsinki Declaration. Furthermore, we obtained a follow-up for some subjects without high-risk carotid plaque.

Result

In total, of the 4207 men who were included in the study were 2064 excluded due to missing follow-up data, resulting in several 2143 adults for the final study. Figure 1 presents the results of carotid artery screening with ultrasound.

Type III and IV b atherosclerosis were found in 578 subjects. A follow-up was available for 566 (97.9%) subjects. The average follow-up time was 48 months (range 0 to 139 months). In total, 230 subjects were treated with a statin, 326 received no statin. Ten subjects were excluded because of missing data on treatment. Within the group of these subjects (54±6 years), 33 men died (15 cancer lung, stomach, pancreas, larynx, gallbladder, 4 strokes, 11 heart attacks, 1 COPD, 1 sepsis, 1 cirrhosis of the liver).

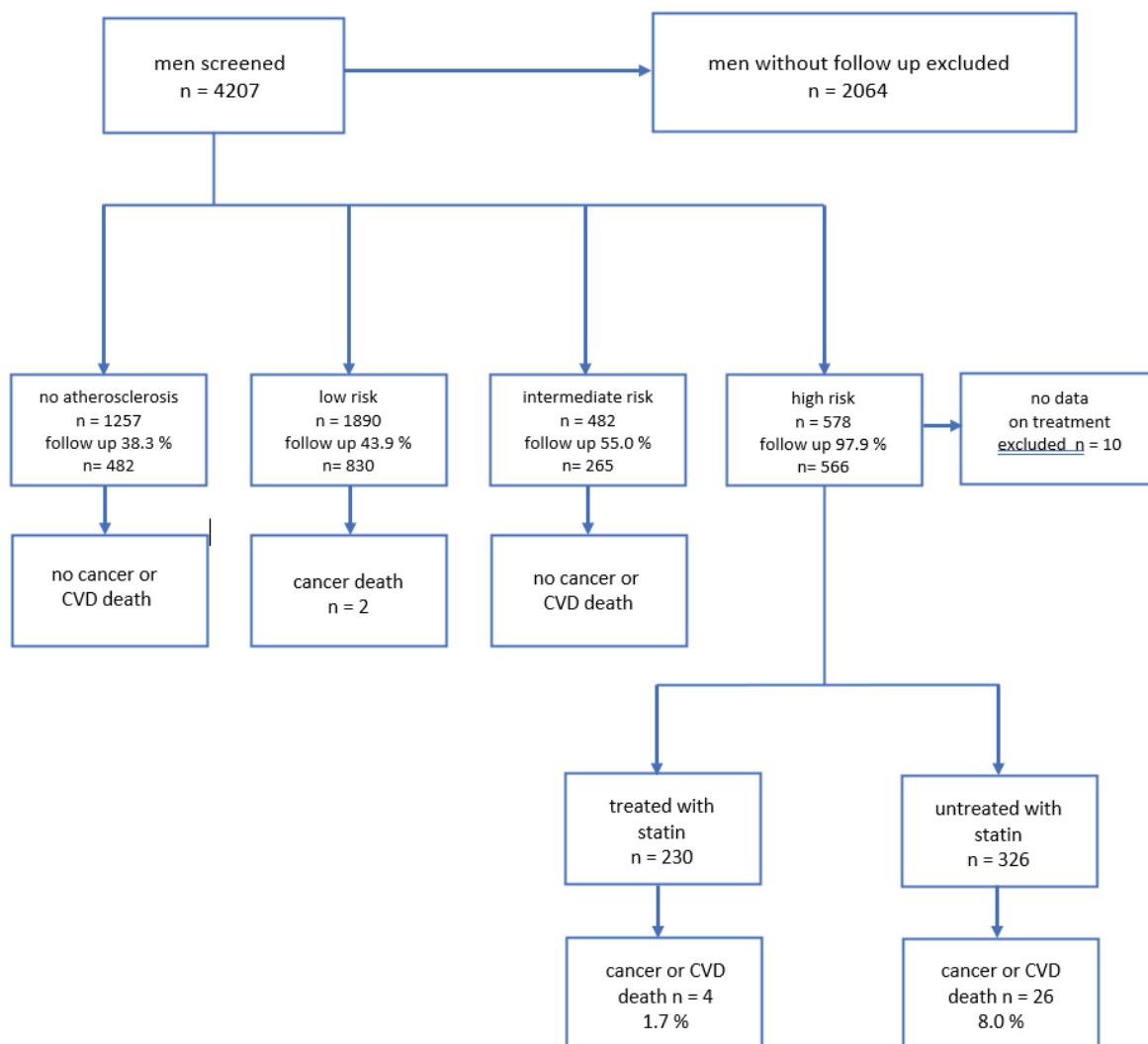


Figure 1: Results of carotid artery screening with ultrasound for men.



In the group with CVD death, two subjects were treated with a statin, 13 were untreated. In the group with cancer death, two subjects were treated with a statin, 13 were untreated. The death rate because of cancer and CVD was 1.7% for the treated group and 8.0% for the untreated group.

In the group with low risk (n = 1890), six men died (2 traffic accidents, 1 aneurysm bleeding, 2 cancer, 1 unclear), a follow-up examination has been completed for 43.9% of the subjects. The average follow-up time was 65 months (range 0 to 137 months). One man died (Parkinson) within the group with intermediate risk. In this group (n= 482) a follow-up examination has been completed for 55% of the subjects. The average follow-up time was 55 months (range 0 to 130 months). In the reference group (no atherosclerosis; n= 1257)

follow-up has been completed in 38.3% (n= 482) with a mean follow-up time of 60 (0-145) months and nobody died. In the groups without or low carotid atherosclerosis (Table 1; n= 1577) two men (0.1%) died of cancer. In the group with advanced carotid atherosclerosis (n= 566) 15 men (2.7%) died of cancer.

In the high-risk group, 227 subjects (40.1%) were smokers. In total, 11 (4.8%) smokers died from cancer. In the group with low and intermediate-risk, 376 subjects (23.9%) were smokers. One (0.3%) smoker died from cancer.

Table 1 presents clinical baseline characteristics for men with no carotid plaque (no atherosclerosis) and tertiles of carotid atherosclerosis. Carotid plaque was found in 1661 (77.5%) individuals.

Table 1: Baseline characteristics for men with no carotid plaque (no atherosclerosis) and tertiles of carotid atherosclerosis.

Maximum plaque thickness (cPT max)	No atherosclerosis (ref)	Tertile 1	Tertile 2	Tertile 3	P value ref vs. Tertile 3
Age SD	43±6	49±7	53±7	54±6	<0.001
BMI, weight SD	27.05±4.51	27.41±3.91	28.32±4.15	27.79±4.15	0.006
Smoker, n (%)	129 (26.8%)	179 (21.6%)	68 (25.7%)	227 (40.1%)	<0.001
Systolic blood pressure SD	123±14	127±15	131±16	134±18	<0.001
Diastolic blood pressure SD	80±8	82±9	83±9	83±9	<0.001
Diabetes, n (%)	10 (2.1%)	32 (3.9%)	15 (5.7%)	53 (9.4%)	<0.001
Treated blood pressure, n (%)	58 (12.0%)	164 (19.8%)	102 (38.5%)	201 (35.5%)	<0.001
Positive family history, n (%)	87 (18.04%)	209 (25.18%)	59 (22.3%)	163 (28.8%)	<0.001
Tot Plaque area mm ² (TPA) SD	0	31±23	83±40	143±61	
Max. plaque thickness mm SD	0.0	1.5±0.3	2.2±0.5	3.0±0.7	
Framingham-BMI % SD	8.95%±6.85%	13.72%±8.70%	19.18%±10.54%	23.36%±13.19%	<0.001
Cardiovasc/cancer Death, n (%)	0 (0%)	2 (0.24%)	0 (0%)	30 (5.3%)	<0.001
Average follow-up time (months) min, max	60 (0.145)	65 (0.137)	55 (0.130)	48 (0.139)	
Follow-up, n (%)	482 (38.3%)	830 (43.9%)	265 (55%)	566 (97.9%)	
Values are represented as mean±SD					

TPA/ max. Plaque thickness: 1. tertile (Type I, II a, TPA < 25mm²), 2. tertile (Type II b, IV a); 3. tertile (Type III, IV b).

Table 2: Baseline characteristics of a live subjects and patients with cancer or CVD death.

	Alive	Dead all cause	Cancer/cvd dead	Cancer dead	cvd dead	P Value alive vs. cancer/ CVD dead
Age SD	50±7	56±6	57±5	56±5	57±4	<0.001
BMI, Weight SD	27.57±4.16	27.87±4.35	28±4.28	26.85±2.98	29.31±5.08	0.585
Smoker, n (%)	584 (27.8%)	19 (47.5%)	17 (53.1%)	12 (70.6%)	5 (33.3%)	0.005
Systolic blood pressure SD	128±16	134±23	135±24	134±24	137±25	0.112
Diastolic blood pressure SD	82±9	84±12	85±13	83±8	88±16	0.158
Diabetes, n (%)	105 (5%)	5 (12.5%)	4 (12.5%)	2 (11.8%)	2 (13.3%)	0.077
Treated blood pressure, n (%)	507 (24.1%)	18 (45%)	15 (46.9%)	7 (41.2%)	8 (53.3%)	0.006
Positive family history, n (%)	512 (24.3%)	8 (20%)	6 (18.8%)	4 (23.5%)	2 (13.3%)	0.54
Tot Plaque area mm ² (TPA) SD	59±65	144±76	155±67	135±80	154±49	<0.001
Max. plaque thickness mm SD	2.1±1.2	2.9±0.9	2.9±0.8	2.8±0.9	3.1±0.8	<0.001
Framingham-BMI % SD	15.65%±11.16%	27.04%±13.87%	28.74%±13.05%	30.79%±16.34%	26.42%±7.13%	<0.001
Death	0 (0%)	40	32	17	15	<0.001
Average follow-up time (months) min, max	58 (0.145)	59(0.137)	56 (3.139)	51 (0.130)	61 (0.139)	
Follow-up,n	2103	40	32	17	15	

Values are represented as mean±SD

Table 2 presents clinical baseline characteristics for patients with cancer and CVD death. The risk factors for cancer and CVD death are advanced carotid atherosclerosis, age, smoking, diabetes, hypertension.

Figure 2 shows the unadjusted event rates for cancer/CVD death as a function of plaque-type in ultrasound. High risk was observed with type III-IV b in ultrasound $P < 0.0001$.

Table 3 shows an unadjusted Cox proportional hazards model survival analysis for cancer/CVD death. For the outcome, Type III-IV b in ultrasound and FRAM-BMI were significant predictors.

Figure 3 shows unadjusted HRs for plaque-type and FRAM-BMI. Both plaque-type in ultrasound and FRAM-BMI had strong predictions for cancer/CVD death (endpoint P -value < 0.001).

Table 4 presents the baseline data for men with low and intermediate-risk in ultrasound with and without follow-up. The baseline data are very similar. One can assume that the outcome for cancer and CVD death would be similar.

In 3203 healthy women aged 35–65 years nobody died of cancer.

Discussion

According to research in PubMed and google scholar, there are only a few studies that report a relationship between atherosclerosis and cancer [18,20–28], in this case specifically on colorectal cancer. Kim et al. retrospectively evaluated 4871 subjects who underwent an ultrasound examination of the carotid artery and a colonoscopy. This showed that

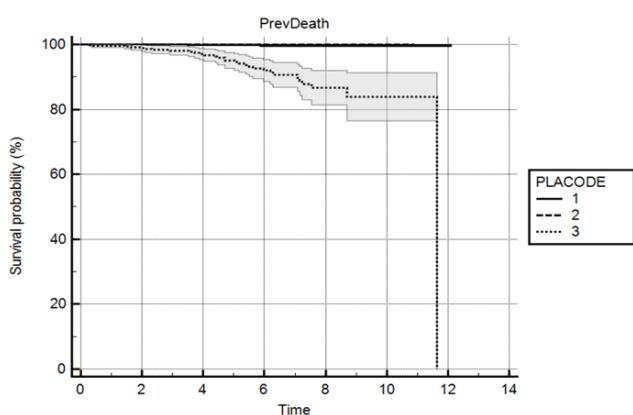


Figure 2: Kaplan–Meier survival curves as a function of plaque-type (tertile 1-3) in ultrasound.
Significance $P < 0.0001$.

the presence of plaques on the carotid artery was correlated with an increased risk of high-risk colorectal adenomas [20]. Some publications describe a correlation between common risk factors such as smoking, diabetes, obesity, chronic inflammation, and the development of atherosclerosis and cancer [23–28]. Dobrzycka, et al. found, after a systematic review on statins and colorectal cancer, suggested evidence that statins have a preventive effect both for prevention and for therapy [21]. Smoking is an important risk factor for advanced atherosclerosis. In our cohort, the mean TPA in all age groups for smokers is about twice as high as the one for those who never smoked. In the reference, low and intermediate-risk group (low atherosclerosis) there are 376 smokers with follow-up. One of whom died of cancer. In the group with advanced atherosclerosis, 15 died of cancer, eleven of whom were smokers. It appears that smoking combined with advanced atherosclerosis is associated with a high risk of cardiovascular and cancer death, but smoking with low atherosclerosis may not. Whether there really is a relationship should be further investigated by appropriate investigations.

It is also noticeable that in the group with advanced atherosclerosis treated with a statin only two subjects died of cancer, while in the untreated group 13 subjects died of cancer. Statin treatment for advanced atherosclerosis can reduce cardiovascular events [13]. Whether this also applies to cancer cannot be deduced from this observational study, hence further investigation seems reasonable. Carotid screening of cancer patients would be useful in this context. A new approach to the prevention of the leading causes of death could emerge.

Conclusion

The development of cancer for men with advanced atherosclerosis of the carotid artery appears noticeable. Whether there is a causal relationship requires further investigation.

There is an initial suspicion that statins could have a preventive effect.

Limitations

The sample size of the study is too small, the outcome events were too few, and the follow-up time was not long enough. All this greatly limit the reliability of the conclusions. Therefore, only a reasonable suspicion can be formulated.

A new analysis should be carried out after a longer follow-up period.

Informed consent

All patients provided informed consent.

Table 3: Cox proportional hazards model survival analysis for cancer/CVD death using the covariates FRAM-BMI and Type III-IV b in ultrasound.

Covariate	b	SE	Wald	P	Exp(b)	95% CI of Exp(b)
FRAMBMI	0.03113	0.01118	7. 7469	0.0054	1. 0316	1. 0092 to 1. 0545
PLACODE=3	3. 6844	0.7492	24. 1848	<0.0001	39. 8195	9. 1701 to 172. 9089

Significance $P < 0.0001$

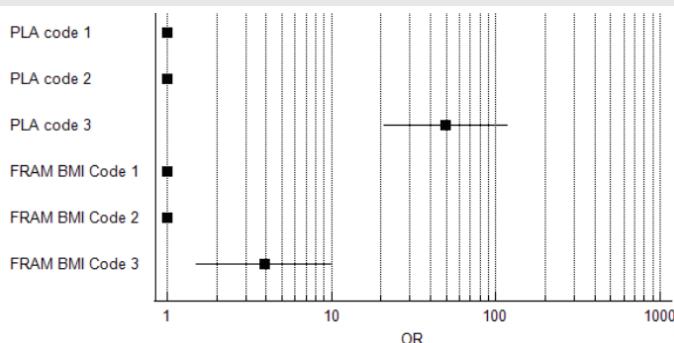


Figure 3: HRs (95% CI) for cancer/CVD death associated with plaque-type and FRAM-BMI.

P-values for trend < 0.0001.

Table 4: Baseline characteristics for men with no atherosclerosis, low and intermediate-risk with and without follow up.

Risk group in ultrasound finding	0-2 follow up	0-2 without follow up	P Value
Age SD	48±7	48±8	0.6
BMI, weight SD	27.49±4.16	27.29±4.08	0.107
Smoker, n (%)	376 (23.8%)	442 (21.5%)	0.014
Systolic blood pressure SD	127±15	127±16	0.212
Diastolic blood pressure SD	81±9	82±9	0.13
Diabetes, n (%)	57 (3.6%)	59 (2.9%)	0.256
Treated blood pressure, n (%)	324 (20.5%)	412 (20.1%)	0.771
Positive family history, n (%)	354 (22.4%)	443 (21.6%)	0.493
Tot Plaque area mm ² (TPA) SD	30±35	22±26	<0.001
Max. plaque thickness mm SD	1.7±0.4	1.6±0.4	0.315
Framingham-BMI % SD	13.18%±9.19%	12.98%±9.46%	0.078
Cardiovasc./cancer Death. n (%)	2 (0.1%)		
follow-up time (months) min, max	62 (0. 145)		
n (%)	1577 (41.5%)	2052 (58.5%)	
Valves are represented as mean±SD			

Author contributions

WB and MR discussed the approach and the findings of this study with AA intensively and gave AA valuable feedback.

MR supported the statistics.

All examinations have been done by AA.

Data availability

Any inquiries regarding supporting data availability of this study should be directed to the corresponding author.

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