oncologist. Median survival was 13.2 months (range 1–96 months). Participants who died in the 6 months after their first consultation were more malnourished ($P = .05$) than survivors. Univariate logistic regression showed that malnutrition (odds ratio (OR) = 6.86, $P = .04$) and recommendation of optimal or adapted treatment by the oncogeriatric team (OR = 0.18, $P = .04$) were significantly associated with early death (Table 1).

These findings showed that malnutrition, measured using the MNA, was associated with early death, consistent with previous studies that showed an association between malnutrition and death in individuals with cancer.[6,7] The current study found an association between optimal or adapted treatment given by the oncogeriatric team and absence of early death, which greater tolerability of treatment in older adults may explain. Various publications have shown that the CGA can identity prognostic factors of early death and severe treatment toxicity.[6,8,9] Recommendation of optimal or adapted treatment by the oncogeriatric team was associated with no high risk of early death.

The main limitation of the current study was the small number of participants, which limited statistical power. Further research is needed to corroborate this finding.

Eloise Lefur, MD
Department of Geriatrics, Nantes University Hospital, Nantes, France

Dominique Berton-Rigaud, MD
Department of Oncology, Centre René Gauducheau, Nantes, France

Anne S. Boureau, MD
Guillaume Chapelet, MD
Gilles Berrut, MD, PhD
Laure de Decker, MD, PhD
Department of Geriatrics, Nantes University Hospital, Nantes, France

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REFERENCES

1. Ries LA. Ovarian cancer survival and treatment differences by age. Cancer 1993;71:524–529.
2. Chan JK, Loizzi V, Lin YG et al. Stages III and IV invasive epithelial ovarian carcinoma in younger versus older women: What prognostic factors are important? Obstet Gynecol 2003;102:156–161.
3. Maas H, Kruitwagen R, Lemmens V et al. The influence of age and co-morbidity on treatment and prognosis of ovarian cancer: A population-based study. Gynecol Oncol 2005;97:104–109.
4. Puts MT, Hardt J, Monette J et al. Use of geriatric assessment for older adults in the oncology setting: A systematic review. J Natl Cancer Inst 2012;104:1133–1163.
5. Miller MD, Paradis CF, Houck PR et al. Rating chronic medical illness burden in geropsychiatric practice and application of the cumulative illness rating scale. Psychiatry Res 1992;41:237–248.
6. Soubeyran P, Fonck M, Blanc-Bisson C et al. Predictors of early death risk in older patients treated with first-line chemotherapy for cancer. J Clin Oncol 2012;30:1829–1834.
7. Aaldriks AA, Maartense E, le Cessie S et al. Predictive value of geriatric assessment for patients older than 70 years, treated with chemotherapy. Crit Rev Oncol Hematol 2011;79:205–212.
8. Freyer G, Geay JF, Touzet S et al. Comprehensive geriatric assessment predicts tolerance to chemotherapy and survival in elderly patients with advanced ovarian carcinoma: A GINECO study. Ann Oncol 2005;16:1795–1800.
9. Repetto L, Fratino L, Audisio RA et al. Comprehensive geriatric assessment adds information to eastern cooperative oncology group performance status in elderly cancer patients: An Italian group for geriatric oncology study. J Clin Oncol 2002;20:494–502.

VASCULAR RETINOPATHY IN RELATION TO COGNITIVE FUNCTIONING IN AN OLDER POPULATION—THE HOORN STUDY

To the Editor: Cognitive impairment and dementia are important health problems that may be caused by vascular damage in the brain.[1] The cerebral vasculature is anatomically, embryologically, and physiologically related to that of the retina, and both are sensitive to exposure to vascular risk factors.[2,3] Vascular damage to the retina is easy to measure noninvasively. Hence, visualization of retinal vessels may offer insight into the status of the vessels in the brain and thus provide insight into vascular causes of late-life cognitive impairment.

A recent systematic review found variable associations between retinal vascular changes and performance on various cognitive domains in persons without dementia.[4,5] The goal of the current study was to extend these findings by assessing these associations in a population-based cohort using a detailed neuropsychological examination.

METHODOLOGY

Participants (N = 313, mean age 72.9 ± 5.6, 52% male) from the population-based Hoorn cohort underwent fundus photography and neuropsychological examination. Details about the design of the study have been described previously.[6–8] None of the participants had cognitive dysfunction severe enough to disturb day-to-day functioning.

Any vascular retinopathy was defined as presence of hypertensive or sclerotic vessel changes; hemorrhages and microaneurysms; preretinal, vitreous, or flame-shaped hemorrhages; hard exudates; cotton wool spots; intraretinal microvascular abnormalities; venous beading; areas of neovascularization; fibrous proliferation; laser coagulation scars; focal arteriolar narrowing; arteriovenous nicking; venous or arteriolar occlusion; arterial narrowing; retinal

Table 1. Comparison of Cognitive Functioning Between Participants with Any or Severe Vascular Retinopathy and Those without Vascular Retinopathy (Reference No Retinopathy)

	Any (n = 74) Versus No (n = 239) Vascular Retinopathy	Severe (n = 6) Versus No (n = 239) Vascular Retinopathy
Measure	Mean Difference in *Z*-Score, Beta (95% Confidence Interval) *P*-Value	
Abstract reasoning	0.07 (−0.18–0.32) .58	−0.02 (−0.77–0.81) .95
Memory	0.09 (−0.03–0.21) .13	0.12 (−0.24–0.47) .52
Information processing speed	−0.02 (−0.21–0.17) .84	−0.10 (−0.68–0.49) .75
Attention and executive functioning	0.02 (−0.13–0.17) .75	0.01 (−0.42–0.45) .95
Visuoconstruction	0.10 (−0.15–0.36) .42	−0.24 (−1.05–0.57) .56
Language	0.13 (−0.09–0.35) .24	0.01 (−0.68–0.71) .97
Sum score	0.07 (−0.05–0.18) .25	−0.01 (−0.37–0.34) .94

Negative values reflect worse performance in the retinopathy group.

vasculopathy; or atherosclerosis or a clinical diagnosis of hypertensive retinopathy or diabetic retinopathy (based on the Europe and Diabetes Study (EURODIAB)[9]). Signs of severe vascular retinopathy were presence of hemorrhages or microaneurysms, cotton wool spots, intraretinal microvascular abnormalities, areas of neovascularization, fibrous proliferation, laser coagulation scars, or a clinical diagnosis of severe diabetic retinopathy (EURODIAB[9] Grade 3–5).

Raw test scores in six cognitive domains (abstract reasoning, memory, information processing speed, attention and executive functioning, visuoconstruction, language) were standardized into *z*-scores per test and subsequently averaged per domain. Global cognitive functioning was calculated by averaging *z*-scores of the different domains. An estimation of premorbid intelligence and an assessment of depressive symptoms were included.[7]

Participants underwent vascular risk factor assessment, including measurements of body mass index, blood pressure, hypertension, glycosylated hemoglobin (HbA1c,%), total cholesterol, presence of type 2 diabetes mellitus, and self-reported history of cardiovascular disease.[6,7]

Between-group differences were analyzed using analysis of (co)variance for continuous variables, Mann–Whitney *U* tests for nonparametric data, and chi-square tests for proportions.

Covariates in cognitive functioning analyses were sex, age, estimated premorbid intelligence, and in separate analyses, vascular risk factors or depression. The study sample allowed group differences with effect sizes as small as ~0.30 (power = 0.80, alpha = 0.05) to be detected. Thus, statistical power was sufficient to detect decrements in cognitive performance relevant for daily functioning.[10] Sensitivity analyses were performed by comparing persons with and without severe vascular retinopathy.

RESULTS

Any vascular retinopathy was present in 74 participants (23.6%). Participants with any vascular retinopathy were older than those without (74.1 ± 5.6 vs 72.5 ± 5.6, $P = .03$), were less likely to be male (43% vs 57%, $P = .05$), and had higher HbA1c levels (6.0 ± 0.6% vs 5.7 ± 0.6%, $P = .001$). No other differences in risk factor profile were found.

There were no differences in cognitive functioning between participants with and without any vascular retinopathy (Table 1). Mean differences in *z*-scores ranged from –0.02 to 0.13 (all $P > .05$). Additional adjustment for depressive symptoms or vascular risk factors did not change the results (data not shown). Six participants (1.9%) had severe vascular retinopathy. They were slightly older than participants without vascular retinopathy (77.0 ± 7.1 vs 72.5 ± 5.6, $P = .05$). Cognitive functioning did not differ significantly from that of participants without vascular retinopathy. Mean differences in *z*-scores ranged from −0.24 to 0.12 (all $P > .05$).

DISCUSSION

In a population-based sample of older persons participating in the Hoorn Study, presence of any or severe vascular retinopathy was unrelated to cognitive functioning.

A limited number of studies have investigated the relationship between vascular retinopathy and cognitive functioning in the general population.[4] Generally, neuropsychological examinations in these studies were less elaborate than in the current study. Effect sizes were in the range of −0.25 to 0.03, which is in line with the present results.

Strengths of the present study include the detailed recording of retinal determinants and extensive neuropsychological assessment in a well-defined population-based cohort of older participants. A limitation is that only cross-sectional data on retinal changes and cognitive functioning were available.

In conclusion, in individuals without dementia in the general older population, there is no clinically relevant relationship between the presence of any vascular retinopathy and cognitive performance.

Sophie M. Heringa, PhD
Department of Neurology, Brain Center Rudolf Magnus, University Medical Center Utrecht, Utrecht, the Netherlands

Iris Walraven, MSc
Annette C. Moll, MD, PhD
Department of Ophthalmology, VU University Medical Center, Institute for Research in Extramural Medicine (EMGO Institute), Amsterdam, the Netherlands

Esther van den Berg, PhD
Department of Neurology, Brain Center Rudolf Magnus, University Medical Center Utrecht, Utrecht, the Netherlands
Experimental Psychology, Helmholtz Institute, Utrecht University, Utrecht, the Netherlands

Giel Nijpels, PhD
Department of General Practice, VU University Medical Center, Institute for Research in Extramural Medicine (EMGO Institute), Amsterdam, the Netherlands

Coen D. A. Stehouwer, MD, PhD
Department of Internal Medicine, Cardiovascular Research Institute Maastricht, Maastricht University Medical Center, Maastricht, the Netherlands

Yael D. Reijmer, PhD
J. Philip Kistler Stroke Research Center, Massachusetts General Hospital, Boston, Massachusetts

L. J. Kappelle, MD, PhD
Department of Neurology, Brain Center Rudolf Magnus, University Medical Center Utrecht, Utrecht, the Netherlands

Jacqueline M. Dekker, PhD
Institute for Research in Extramural Medicine (EMGO Institute), VU University Medical Center, Amsterdam, the Netherlands

Geert Jan Biessels, MD, PhD
Department of Neurology, Brain Center Rudolf Magnus, University Medical Center Utrecht, Utrecht, the Netherlands

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Author Contributions: Sophie Heringa performed statistical analyses and wrote the first draft of the manuscript. Esther van den Berg and Yael Reijmer collected cognitive and anthropomorphic data and questionnaires. Iris Walraven collected fundus photographs. Annette C. Moll graded fundus photographs. Giel Nijpels, Coen Stehouwer, and Jacqueline Dekker were principle investigators of the Hoorn Study. Esther van den Berg, Jacqueline Dekker, L. Jaap Kappelle, and Geert Jan Biessels participated in design of the study and interpretation of data and contributed to the discussion. All authors contributed to and approved the final manuscript.

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REFERENCES

1. Gorelick PB, Scuteri A, Black SE et al. Vascular contributions to cognitive impairment and dementia: A statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2011; 42:2672–2713.
2. Wong TY, Klein R, Klein BE et al. Retinal microvascular abnormalities and their relationship with hypertension, cardiovascular disease, and mortality. Surv Ophthalmol 2001;46:59–80.
3. Wong TY, McIntosh R. Systemic associations of retinal microvascular signs: A review of recent population-based studies. Ophthalmic Physiol Opt 2005;25:195–204.
4. Heringa SM, Bouvy WH, van den Berg E et al. Associations between retinal microvascular changes and dementia, cognitive functioning, and brain imaging abnormalities: A systematic review. J Cereb Blood Flow Metab 2013;33:983–995.
5. Schrijvers EMC, Buitendijk GHS, Ikram MK et al. Retinopathy and risk of dementia: The Rotterdam Study. Neurology 2012;79:365–370.
6. Mooy JM, Grootenhuis PA, de Vries H et al. Prevalence and determinants of glucose intolerance in a Dutch Caucasian population. The Hoorn Study. Diabetes Care 1995;18:1270–1273.
7. Van den Berg E, Dekker JM, Nijpels G et al. Cognitive functioning in elderly persons with type 2 diabetes and metabolic syndrome: The Hoorn Study. Dement Geriatr Cogn Disord 2008;26:261–269.
8. Van Leiden HA, Dekker JM, Moll AC et al. Blood pressure, lipids, and obesity are associated with retinopathy: The Hoorn Study. Diabetes Care 2002;25:1320–1325.
9. Aldington SJ, Kohner EM, Meuer S et al. Methodology for retinal photography and assessment of diabetic retinopathy: The EURODIAB IDDM complications study. Diabetologia 1995;38:437–444.
10. Cohen J. Statistical Power Analysis for the Behavioral Sciences, 2nd Ed. New York: Academic Press, 1988.

DENTAL STATUS IS UNRELATED TO RISK OF DEMENTIA: A 20-YEAR PROSPECTIVE STUDY

To the Editor: Good dental status and oral health have been discussed in association with successful aging,[1] cognitive functioning,[2] and dementia.[3] Whether the absence of natural teeth increases the risk of incident dementia was investigated in a large population-based sample of older adults (≥60 at baseline). A variety of demographic factors and apolipoprotein E (APOE) genotype were controlled for in the analyses.

METHOD

The data used in the present study emanate from the Betula prospective cohort study, established in Umeå, Sweden, in 1988.[4] Baseline information about dental status was available for 2,120 participants aged 60 and older. Participants diagnosed with dementia were compared with those without over a follow-up period of up to 20 years. Those with dementia at baseline (n = 19), who were diagnosed with dementia shortly after the latest assessment (n = 10), or who died shortly after (<1 year) inclusion (n = 16) were discarded from the analyses. Thus, 2,075 participants were included in the analyses (917 men, 1,158 women). Their mean baseline age was 71.2 ± 8.2.

Dental status was based on self-reports that divided the participants into three groups: only natural teeth (n = 1,110, mean age 68.5 ± 7.6; 52% female), natural teeth and dentures (n = 439, mean age 73.0 ± 7.7; 57% female), and edentulous and dentures (n = 526, mean age