revascularization mainly rely on degree of stenosis. However, other plaque characteristics might be better determinants of recurrent stroke, allowing more targeted intervention. The Plaque At RISK (PARISK)-study aimed to improve identification of patients at increased risk of recurrent stroke using multimodality carotid imaging.

Methods: We included 240 patients with symptomatic <70% ipsilateral carotid stenosis in a prospective multicenter cohort study. MRI (carotid and brain) and MDCTA (carotid) were performed at baseline and after 2 years. The clinical endpoint was a recurrent ipsilateral ischemic stroke or TIA. Cox-proportional hazard models were used to assess whether intraplaque hemorrhage (IPH), ulceration, proportion of calcifications, and total plaque volume in ipsilateral carotid plaques were associated with the endpoint. Next, we investigated the predictive performance of these imaging biomarkers by adding (combinations of) these markers to the ECST-risk score.
Results: During 5.1 years follow-up 37 patients reached the clinical endpoint. IPH presence and total plaque volume were associated with recurrent ipsilateral ischemic stroke or TIA (HR=2.12, 95%CI: 1.02-4.44 for IPH; HR=1.07, 95%CI: 1.00-1.15 for total plaque volume per 100 mm^3 increase). Ulcerations and proportion of calcifications were not statistically significant determinants. Addition of IPH and total plaque volume to the ECST-risk score improved the model performance (C-statistics increased from 0.67 to 0.75-0.78).

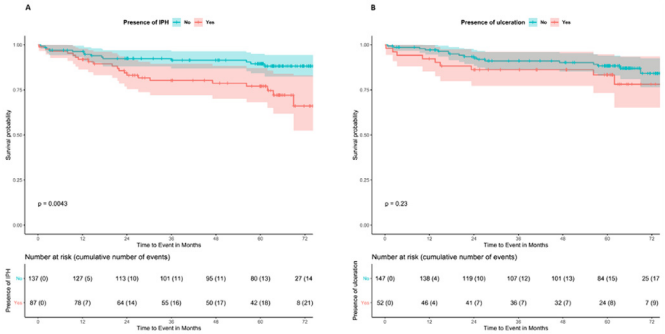

Figure. Kaplan-Meier curves showing the survival probability for the clinical endpoint, defined as a clinical ipsilateral recurrent ischemic stroke or TIA, regarding IPH (A) and ulcerations (B). The tables show the according number at risk and cumulative number of events per year. IPH = intraplaque hemorrhage.

Conclusions: IPH and total plaque volume are independent risk factors of recurrent ipsilateral ischemic stroke or TIA in patients with mild-to-moderate carotid stenosis. These plaque characteristics improve current decision making.

O076 / #775, AGING, DEMENTIA AND STROKE, 25-05-2022 11:00 AM - 12:30 PM.
CLONAL HEMATOPOIESIS ARE NOT ASSOCIATED WITH AN INCREASED SYSTEMIC INFLAMMATION, ATHEROSCLEROSIS NOR INCIDENCE OF ATHEROTHROMBOSIS: RESULTS FROM THE 3-CITY STUDY (CHIP-3C)

S. Marti [1], M. Dufossée [2], S. Fawaz [3], D.-A. Tregouet [4], C. James [5], T. Couffinhal [6], O. Mansier [5]. [1] *Laboratory Of Hematology, University hospital of Bordeaux, PESSAC, France;* [2] *Inserm U1034, INSERM, PESSAC, France;* [3] *Coronary And Vascular Diseases, University hospital of Bordeaux, PESSAC, France;* [4] *Inserm U1219, INSERM, BORDEAUX, France;* [5] *Inserm U1034 And Laboratory Of Hematology, INSERM and University hospital of Bordeaux, PESSAC, France;* [6] *Inserm U1034 And Coronary And Vacular Diseases, INSERM and University hospital of Bordeaux, PESSAC, France*

Background and Aims : Clonal hematopoiesis of indeterminate Potential (CHIP) is defined by the detection of leukemia-associated mutations in the absence of hematological malignancy. This condition is associated with an increased mortality mainly driven by athero-thrombotic complications. Previous studies in mouse models demonstrated that CHIP increase atherosclerosis development. However the association between CHIP, atherosclerosis and athero-thrombosis remains poorly evaluated in patients.

Methods: The 3-city study is a population-based longitudinal study that enrolled individuals aged ≥65 years. In this cohort, we selected 322 persons who had no cardiovascular event before inclusion. Eighty five of them suffered from a myocardial infarction or stroke during the 12-year follow-up. We searched for CHIP by a targeted NGS strategy on DNA collected at inclusion. Anthropomorphic, cardiovascular (risk factors, diet, atherosclerosis) and biological (CRP level) data at inclusion as well as incidence of athero-thrombotic events were compared between patients with or without CHIP.
Results: A CHIP was detected in 41% of patients. As described, most patients presented mutations in *DNMT3A* (46%) and *TET2* (30%). Patients with CHIP were slightly older than patients without CHIP (74.2 years VS 73 years, p=0.03). Neither the cardiovascular risk profile, nor the CRP levels (1.66 VS 1.75), nor the number of atheromatous plaques nor the intima-media thickness (0.67 VS 0.68) were different between patients with and without CHIP. The incidence of athero-thrombotic complications (myocardial infarction or stroke) was similar between patients with a CHIP and patients without.
Conclusions: In conclusion, CHIP, in particular involving *DNMT3A* mutations, are not strongly associated with systemic inflammation, atherosclerosis or athero-thrombotic events.

O077 / #273, AGING, DEMENTIA AND STROKE, 25-05-2022 11:00 AM - 12:30 PM.
BODY MASS INDEX AND RISK OF DEMENTIA

L.T. Nordestgaard [1,2], B.G. Nordestgaard [2,3], A. Tybjaerg-Hansen [1,2], R. Frikke-Schmidt [1,2]. [1] *Department Of Clinical Biochemistry, Rigshospitalet, Copenhagen University Hospital, Copenhagen, Denmark;* [2] *Department Of Clinical Medicine, Faculty of Health and Medical Science, University of Copenhagen, Copenhagen N, Denmark;* [3] *Department Of Clinical Biochemistry, Herlev and Gentofte Hospital, Copenhagen University Hospital, Herlev, Denmark*

Background and Aims : Midlife obesity and underweight in the elderly have been associated with a high risk of dementia. However, the association between body mass (BMI) and risk of dementia depends on the type of dementia investigated. Whether there is a causal association between BMI and the vascular part of dementia called “non-Alzheimer dementia” remains unknown. We aimed to investigate the association between BMI and risk of “non-Alzheimer dementia”, Alzheimer’s disease, and all-cause dementia.

Methods: In a prospective cohort of the Danish general population including 95,000 individuals we investigated the observational and genetically determined association between BMI and risk of “non-Alzheimer dementia”, Alzheimer’s disease, and all-cause dementia. For the genetic analyses we created a weighted allele score and divided it into four groups from lowest to highest BMI.
Results: The observational association between BMI and risk of “non-Alzheimer dementia” and all-cause dementia was u-shaped with nadir at a BMI of 26 kg/m^2. For Alzheimer’s disease the association was linear with low BMI associated with high risk. Comparing the group with the lowest genetically determined BMI to the group with the highest genetically determined BMI the hazard ratio (95% confidence interval) was 1.22 (1.01-1.47) for “non-Alzheimer dementia”, 1.04 (0.90-1.20) for Alzheimer’s disease, and 1.10 (0.98-1.23) for all-cause dementia.
Conclusions: Genetically determined high BMI is associated with high risk of the vascular part of dementia in the general population. BMI is thus a potentially modifiable risk factor for dementia that could be targeted in the strive to prevent this devastating disease.

O078 / #808, IMAGING GUIDED MANAGEMENT, 25-05-2022 11:00 AM - 12:30 PM.
NON-CALCIFIC ATHEROSCLEROTIC BURDEN IN HEFH: THE FH-CALC STUDY

A. Gallo [1,2], V. Carreau [2], A. Mondelli [2], M. Zarai [3], E. Charpentier [3], N. Kachenoura [4], A. Redheuil [3], E. Bruckert [2]. [1] *Unité Détroi, Université de La*