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# Cognitive Impairment in Patients with Arterial Hypertension as a Predictor of Dementia

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**Abstract**
Arterial hypertension (AH) is considered as one of the causes of vascular dementia. Brief Montreal Cognitive Assessment Scale (MoCA) can be sensitive to the detection of cognitive impairment (CI) in AH.

**Introduction:**
Dementia is one of the major causes of loss of autonomy, and the main reason for the Institutionalization of the elderly. Epidemiological studies conducted in the last 10 years have shown that the prevalence of dementia is close to 5% in the population over 65 years of age. These studies have also shown that its prevalence increases sharply with age, and as a result of the expected shift in population demographics, the incidence and prevalence of dementia are expected to increase dramatically over the coming decades. The number of demented patients worldwide is projected to increase from 243 million in 2001 to 81.1 million in 2040. Significantly the vast majority of new cases are expected to appear in developing countries. For example, the number of demented persons in China and India will increase by 300% during this period.1 Prevention and management of dementia are therefore a major public health challenge in the majority of countries around the world.

As a general rule, the occurrence of dementia is not a sudden phenomenon. It is the final stage of cognitive deterioration, the speed of which varies from one individual to the other. However, even in cases where its development is rapid, the process is measured in terms of months. Taking into account the life expectancy of individuals at risk, retarding the development of dementia for a few months may have important consequences on the prevalence of dementia.

Such expectations have been raised in recent years with the discovery of a relationship between hypertension and dementia. Overall, published studies suggest that high blood pressure increases the risk of cognitive decline and dementia, and therefore, that lowering blood pressure might reduce this risk.

**Objective:**
To evaluate the validity of the use of MoCA test for the detection of CI in patients with AH lasting no more than 10 years.

**Material & Methods:**
Observed 60 patients with a higher education (females -24, males -36) aged 40-60 years with AH lasting no more than 10 years. To determine the degree of CI, MMSE and MoCA were used.

**Hypertension and Cognitive Decline:**
It has been known for decades that there is a direct causal relationship between high blood pressure and the risk of stroke and therefore the risk of dementia. It is common knowledge that large strokes or multiple strokes contribute directly to cognitive decline and to the risk of dementia, consequently called vascular dementia. However, it is only in the past 10 years that studies have reported that hypertension may be related to cognitive decline and dementia without the occurrence of a stroke.

**Hypertension and Stroke Related Dementia:**
Hospital and population-based studies have firmly established that dementia is more frequent in patients with stroke than in patients without. Cognitive assessment performed 3 months after stroke revealed that 20% to 30% of patients are demented. In one of the largest clinical series of 453 patients who were examined 3 months after their stroke, 26% were demented. It is estimated that stroke multiplies the risk of dementia by a factor of two to five, thus constituting one of the strongest risk factors for dementia. The strength of this association suggests a causal link between stroke and dementia, although numerous other factors influence this relationship, some pertaining to the patient such as age, level of education, cognitive level before stroke, white matter lesions on magnetic resonance imaging (MRI), Apolipoprotein E4 (ApoE4) allele, etc - and others to the stroke itself mainly its size, severity, and location. Interestingly, in the few studies that have included a classification of dementia, typical vascular dementia represented only 57 %11 to 64 %7 of all dementias with stroke, thus suggesting that a significant proportion of stroke-associated dementias may be classified as Alzheimer's disease (AD) or mixed dementia. This was confirmed in population-based studies in Rochester and New York, where a 50% to 60% increase in AD in individuals with stroke compared with those without was observed. These data were interpreted as meaning that the occurrence of a stroke may actually unmask ongoing AD. This hypothesis was also lent support by studies showing that prestroke cognition is altered in 15% to 20% of patients with a poststroke dementia. The effect of this interaction between neurodegenerative factors or lesions and stroke on the

risk of dementia has been demonstrated in the Nun study. In this autopsy study, participants who had the neuropathological hallmarks of AD and at least one lacunar stroke had a risk of clinical dementia multiplied by a factor of about 20 compared with those with the hallmarks of AD but no lacunar stroke.

**Prevention of Dementia in Stroke Patients:**

Blood pressure-lowering therapy with the long-acting ACE inhibitor perindopril combined with the diuretic indapamide reduces the risk of poststroke dementia by one third and the risk of severe cognitive decline by nearly one half, according to the results from the PROGRESS study (Perindopril pROtection aGainst REcurrent Stroke Study).35 PROGRESS was a randomized, double-blind, placebo-controlled trial that enrolled 6105 men and women, with a mean age of 64 years, with prior stroke or transient ischemic attack (TIA), from 172 institutions in 10 countries in Asia, Australia, and Europe. Participants were randomized to active treatment (n=3051) or placebo (n=3054).36,37 Active treatment was comprised of perindopril 4 mg daily for all participants, along with 2.5 mg daily of the diuretic indapamide (2 mg in Japan) in patients in whom a diuretic was neither specifically indicated nor contraindicated. The main results of PROGRESS38 were that active treatment with perindopril alone or with indapamide reduced blood pressure by 9/4 mm Hg over 4 years of follow-up, and was associated with an overall reduction of 28% in the risk of recurrent strokes (the primary outcome of the study) compared with placebo (P<.0001 among hypertensive and non-hypertensive patients with a history of stroke or TIA). Active treatment also reduced the risk of total major vascular events by 26%. Combination therapy with perindopril plus indapamide reduced blood pressure by 12/5 mm Hg and stroke risk by 43%.

One of the secondary outcomes of PROGRESS was dementia and severe cognitive decline. During the follow-up period of 4 years dementia diagnosed according to DSMIV criteria and severe cognitive decline a drop of ≥3 points in the Mini Mental State Examination [MMSE] were assessed. Median MMSE score at baseline was 29 (range, 27 to 30); a large proportion of patients 41%) had good cognitive function (MMSE =30, but 16% had cognitive impairment (MMSE <26). Over 25% of patients screened positive for dementia 768 and 812 in the active treatment and placebo groups, respectively. After independent assessment by an expert in dementia, 410 patients were identified as having dementia (equivalent to an incidence of 17 per 1000 patient-years), of whom 108 had dementia preceded by a stroke. Cognitive decline was identified in 610 patients (incidence of 25 per 1000 patient-years), of whom 134 had had a previous stroke. Overall there was a non-significant (12% (range, -8% to 28%]) reduction in the risk of dementia in the active treatment group. Evaluation within the two dementia subgroups (with or without prior stroke), however, showed a significant reduction of 34% (P=.03) in the risk of dementia with active treatment in patients with prior stroke and a 1% reduction in patients without prior stroke.

A similar pattern was observed for cognitive decline, with an overall risk reduction of 19% (P=0.01) with active treatment overall, but a significant risk reduction of 45% (P<.001) with active treatment in patients with prior stroke and a 9% reduction in patients without stroke. Combination therapy was more effective in reducing the risk of dementia (23%) than monotherapy (-8%), although there was no statistical difference between regimens (P for homogeneity, 0.1) In patients with no cognitive impairment at baseline (84%), active treatment reduced the risk of dementia by 31%, but there was no effect in patients with cognitive impairment at baseline (-3%). Among the patients without cognitive impairment at baseline, a 50% reduction in the risk of dementia was observed in those with prior stroke, compared with a 16% reduction in those without stroke.

**Results:**

80% of patients with AH are pre-demention CI which is identified in the application of MoCA-test, whereas MMSE was not informative. According to MoCA average value was 19.5±2.5 points and according to the MMSE - 30 points. At the same time moderate CI (22.5±2.5 points) detected in 56.6% of cases, heavy CI (16.5±2.5) was diagnosed in 23.3%. Among patients with a duration of up to 5 years of AH is well coped with the tasks of MoCA40%, 60% of detected light CI. In patients with AH from 5 to 10 years, MCI identified in 53.4% of cases (21±1.0 points), 46.6% of patients had a dementia (14-19 points).

**Conclusions:**

The observed decline in cognitive function in patients with AH that can possibly be regarded as a predictor of dementia. These results make it necessary and justified to make use of MoCA-test for the universal screening for CI.