Vascular dementia

Vascular dementia (VaD) is characteristic of chronic brain ischemia and progressive memory decline, which has a high incidence in the elderly.

It is the second most common form of dementia but reliable evidence on age-specific associations between blood pressure (BP) and risk of vascular dementia is limited and some studies have reported negative associations at older ages.

In a cohort of 4.28 million individuals, free of known vascular disease and dementia and identified from linked electronic primary care health records in the United Kingdom (Clinical Practice Research Datalink), we related BP to time to physician-diagnosed vascular dementia. We further determined associations between BP and dementia in a prospective population-based cohort of incident transient ischemic attack and stroke (Oxford Vascular Study).

For a median follow-up of 7.0 years, 11 114 initial presentations of vascular dementia were observed in the primary care cohort after exclusion of the first 4 years of follow-up. The association between usual systolic BP and risk of vascular dementia decreased with age (hazard ratio per 20 mm Hg higher systolic BP, 1.62; 95% confidence interval, 1.13-2.35 at 30-50 years; 1.26, 1.18-1.35 at 51-70 years; 0.97, 0.92-1.03 at 71-90 years; P trend=0.006). Usual systolic BP remained predictive of vascular dementia after accounting for effect mediation by stroke and transient ischemic attack. In the population-based cohort, prior systolic BP was predictive of 5-year risk of dementia with no evidence of negative association at older ages. CONCLUSIONS:

BP is positively associated with risk of vascular dementia, irrespective of preceding transient ischemic attack or stroke. Previous reports of inverse associations in old age could not be confirmed [1)].

Types

All patients with idiopathic normal pressure hydrocephalus (INPH) who underwent shunting in Sweden in 2008-2010 were compared to age- and sex-matched population-based controls. Inclusion criteria were age 60-85 years and no dementia. The 10 most important vascular risk factor (VRFs) and cerebrovascular and peripheral vascular disease were prospectively assessed using blood samples, clinical examinations, and standardized questionnaires. Assessed VRFs were hypertension, hyperlipidemia, diabetes, obesity, psychosocial factors, smoking habits, diet, alcohol intake, cardiac disease, and physical activity.

In total, 176 patients with INPH and 368 controls participated. Multivariable logistic regression analysis indicated that hyperlipidemia (odds ratio [OR] 2.380; 95% confidence interval [CI] 1.434-3.950), diabetes (OR 2.169; 95% CI 1.195-3.938), obesity (OR 5.428; 95% CI 2.502-11.772), and psychosocial factors (OR 5.343; 95% CI 3.219-8.868) were independently associated with INPH. Hypertension, physical inactivity, and cerebrovascular and peripheral vascular disease were also overrepresented in INPH. Moderate alcohol intake and physical activity were overrepresented among the controls. The population-attributable risk percentage was 24%.

The findings confirm that patients with INPH have more VRFs and lack the protective factors present in the general population. Almost 25% of cases of INPH may be explained by VRFs. This suggests that INPH may be a subtype of vascular dementia. Targeted interventions against modifiable VRFs are likely to have beneficial effects on INPH [2)].

Etiology

Considerable studies showed that a reduction in cerebral blood flow (CBF) might affect learning and memory processes, resulting in the development, and progression of dementia, such as vascular dementia.

Pathogenesis

The underlying mechanism of its pathogenesis remains unclear.

Treatment

A study investigated the effects of a synthetic cannabinoid receptor agonist WIN55,212-2 (WIN) on VaD, and molecular mechanisms of the effects. VaD model was induced by 2-vessel occlusion (2VO). Spatial reference learning was evaluated by the Morris water maze, and recognition memory was assessed using the novel object recognition test. Autophagy-related proteins [microtubule-associated protein 1 light chain 3 (LC-3) and Beclin-1] were examined by immunohistochemistry and Western blot. Caspase-3 was detected by Western blot. Inflammatory factors, tumor necrosis factor alpha (TNF-α) and interleukin 1 beta (IL-1β), were estimated by reverse transcription-polymerase chain reaction (RT-PCR) and Western blot. VaD increased the levels of LC-3, Beclin-1, and inflammatory factors, which were reversed by chronic treatment with WIN. WIN decreased the expression of Capase-3, and improved the learning and memory impairment of VaD rats. These data indicate that WIN exerts a neuroprotective effect on the cognitive deficits of VaD rats, which may be associated with the suppression of excessive autophagy and inflammation [3].

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