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A case report series study for the effects of Policosanol on Post-Stroke Cognitive Impairment

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Ischemic stroke may be a leading explanation for disability, including post-stroke cognitive impairment (CI), that no effective therapy is out there. Post-stroke patients are managed with antiplatelet drugs, and antihypertensive, hypoglycemic and/or cholesterol-lowering drugs, as needed. Policosanol is an antiplatelet and cholesterol-lowering agent. Studies done before are demonstrated that adding policosanol to aspirin (AS) therapy improves post-stroke neurological recovery compared to placebo + AS.

According to Nys et al., a high proportion of stroke survivors dealt with cognitive impairment within 3 months after stroke. Although the prevalence of post stroke cognitive impairment is extremely high consistent with this data, there's still evidence showing that this criteria may underestimate the frequency of the dementia and therefore the cognitive decline in stroke survivors. These patients with the cognitive impairment might be divided consistent with the degree of the cognitive decline into the mild cognitive impairment and dementia.

Policosanol effects on post-stroke cognitive impairment have not been investigated until. Policosanol is likely safe for most people when taken by mouth for up to 3 years in doses of 5-80 mg per day. Policosanol's side effects are usually mild and can include headaches, sleeping difficulty, dizziness, stomach discomfort, skin redness or weight loss. Yet certain side effects are relatively uncommon. The study was undertaken to evaluate the evolution of recent post-stroke (≤30 days from onset) survivors with CI treated with policosanol + AS for 12 months. Recent patients with post- stroke CI untreated with policosanol were enrolled, managed according to guidelines and started on AS (125 mg/day) and policosanol (20 mg/day) for 12 months. Routine neurological examinations; control of therapy compliance and adverse experiences (AE) were done.

CI was assessed with the Luria-Nebraska test at baseline and at 12 months. Ischemic stroke is characterized by the sudden loss of blood circulation to the brain, leading to a corresponding loss of neurologic function. Acute ischaemic stroke is caused by thrombotic or embolic occlusion of an arteria cerebri and is more common than haemorrhagic stroke. Acute ischemic stroke (AIS) is characterized by the sudden loss of blood supply to a brain region, usually in a vascular zone, which results in a corresponding loss of neurological function.

Interestingly, in several studies, the dementia ratio within 3 months after stroke varies from 6% to 27%. The variability of the conclusion could also be thanks to the various application of the standards of the dementia or the cognitive impairment. this standard criteria of the dementia include the diagnostic and statistical manual of mental disorders IV (DSM IV), international classification of disease-10 (ICD-10) and national institute of neurological and communicative disorders and AD and the stroke also related disorders (NINCDS-ADRDA) association criteria.

Besides the demented patients, the degree of the cognitive decline of other cognition-impaired patients who fail to satisfy the above criteria might be measured like the mini-mental state examination (MMSE) score, Montreal cognitive assessment scale (MoCA) score, the abbreviated test, AD assessment scale-cognitive (ADAS-Cog) then on. Of course, there are another measures which are mainly originated from the above. for instance , the six-item screener (SIS) may be a brief cognitive function test which springs from the MMSE and designed for either in-person or telephone administration. What's more, multiple neuropsychological test batteries are wont to examine not only the entire cognitive function but also the extent of impairment on every single cognitive domain like memory, language, visuoconstruction, executive function, calculation, comprehension and judgment.

As no change on test results stable condition was pre-defined, improvement as changes to better levels and deterioration as changes to worst levels. Patients (56) (37 men, 19 women) (73 years) exhibited vascular risk factors: hypertension (76.8%); dyslipidemia (50.0%); smoking (26.8%); and diabetes (17.9%). All patients completed the follow-up and none had a recurrent vascular event. Only 1 patient (1.8%) had further CI deterioration; 27 (48.2%) remained stable, and 29 (51.8%) exhibited mild or moderate improvement. Treatment was well tolerated. Only three patients reported mild AE. It is concluded that the patients with CI post-ischemic stroke treated with policosanol + AS for 12 months had good evolution since none died, none had recurrent events, and most experienced CI improvement or remained stable.

This study has limitations for stronger conclusions since it is a case report series, but our results encourage to investigate such effects in randomized, double-blind, placebo-controlled studies.

The prevalence studies specialise in the entire population who show the cognitive impairment after stroke. Although these studies in hospital or community settings always fail to exclude the patients who have suffered the cognitive decline before the stroke and they should be known the seriousness of the matter. The cross-sectional study widely proceeded in ten countries suggests that about 30% ischaemic stroke survivors show a cognitive impairment which is decided by the MMSE score is less than 27.

What's more, in Caribbean, Chausson et al. examined the cognitive function of 293 stroke patients from the cohort of ERMANCIA study in Martinique 5 years after the first-ever stroke and suggested that 58.9% patients suffered from the cognitive impairment. In Asia, the study conducted by Yu et al. in South Korea suggested the very best results of all.

Proceeding in 12 hospitals in South Korea which enrolled 620 patients with ischemic stroke, it proposed that the prevalence reached up to 69.8% 3 months after stroke as measured by Korea MMSE. The study on 252 Singaporean patients within 6 months post-stroke showed that 44% patients suffered from the cognitive decline, while the prevalence declined to 34% in 1-year follow-up. The later study in India showed the prevalence of cognitive impairment was about 20% in total stroke survivors. Zhou et al. examined the cognitive function of 434 patients with stroke by 1-year follow-up in Chongqing. The study suggested a 37.1% of cognitive impairment prevalence 3 months after stroke.

What's more, one recent study proceeding in Changsha including 689 ischaemic stroke patients detected that the prevalence of post stroke cognitive impairment was 41.8%.

Recently, the cohort of first-ever stroke patients without pre-stroke dementia which is the first cohort study in France suggested that the frequency of the cognitive impairment 3-month after stroke was 47.3%.