A Preliminary Retrospective Analysis of the Effects of Policosanol on Ischemic Stroke Patients

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Abstract—Introduction: Stroke is one of the leading causes of mortality and disability. Clinical studies conducted in patients with a recent ischemic stroke and treated with policosanol (20 mg/day) + standard aspirin (AS) (125 mg/day) therapy have shown benefits versus placebo + AS to patients with recent ischemic stroke. The objective of the present paper is to a preliminary retrospective analysis of the policosanol treatment effects in the patients included in ischemic stroke recovery trials.

Methods: This report analysed the records of all patients included in four ischemic stroke recovery studies. Patients with a modified Rankin Scale score (mRSs) 2 to 4 were randomized, within 30 days of onset, to policosanol+AS or placebo+AS, for 6 and 12 months. The primary outcome was mRSs reduction. Decreases on low-density lipoprotein-cholesterol (LDL-C), total cholesterol and increases on high-density lipoprotein-cholesterol (HDLC) were secondary outcomes.

Results: Two hundred and seventy one patients (mean age: 67 years) were included in the analysis. At the six months more policosanol+AS (117/136, 86 %) than placebo+AS patients (10/135, 7.3 %) achieved mRSs goals. In correspondence, at the 12 months of the study more policosanol+AS (50/59, 84.7 %) than placebo+AS patients (5/59, 8.5 %) achieved mRSs goals. Treatment with policosanol+AS significantly decreased mean mRSs from the first interim check-up. The treatment effect did not wear off, even improved, after 6 and 12 months therapy when the net decrease versus placebo+AS was 56 % and 70.8 %, respectively. In addition, policosanol+AS reduced significantly LDL-C (21.6 %) and total cholesterol (12.5 %), and increased HDLC (6.3 %). Treatments were safe and well tolerated. Eight patients reported serious adverse events (6 placebo+AS, 2 policosanol+AS) and other 13 patients (8 placebo+AS, 5 policosanol+AS) reported moderate or mild adverse events.

Conclusions: The preliminary retrospective analysis of the effects of policosanol+AS on ischemic stroke patients indicate that this treatment for 6 and 12 months proved to be more effective than the placebo+AS treatment in the functional recovery of these patients.

Keywords—Aspirin, policosanol, ischemic stroke, recovery, modified Rankin Scale score.

I. INTRODUCTION

Ischemic stroke is the second leading cause of death worldwide as well as the leading cause of long-term disability.¹,² About half of stroke survivors remain with physical or cognitive impairment that severely affect their physical and social functions. Also, stroke implies a high cost to patients, families and health systems.³

Stroke occurs when blood flow to the brain is interrupted, without oxygen-rich blood, brain cells die. Most strokes (87 % of cases) are classified as ischemic (a clot or a mass blocks a blood vessel, cutting off blood flow to a part of the brain).¹

It is important to identify risk factors and sources of stroke in order to take steps towards preventing stroke. Primary prevention addresses all measures for avoiding a stroke or transient ischemic attack. Secondary prevention addresses all measures for avoiding recurrences after a first transient ischemic attack or stroke manifestation, which is becoming more frequent in an increasingly ageing population.⁴,⁶

Control of modifiable ischemic stroke risk factors, such as hypertension, diabetes, dyslipidemia, cigarette smoking and obesity are key measures to prevent recurrent strokes.⁵

Aspirin (AS) remains the gold standard of antiplatelet therapy for stroke recovery and prevention, and several studies and meta-analyses support the merits of antiplatelet drugs in stroke prevention by lowering platelet function, which reduces thrombotic complications of atherosclerosis.⁷-¹⁰

Reduction of low-density lipoprotein-cholesterol (LDL-C) levels has been shown to be relevant not only for stroke prevention,¹¹,¹² but also for improving functional outcomes after stroke, a key matter for reducing the disability after stroke.¹³-¹⁵

Policosanol, a mixture of 8 high molecular weight sugarcane wax alcohols, has shown protective effects in experimental brain ischemia,¹⁶-¹⁸ and clinical studies have found coherent results.¹⁹-²⁵

In light of these facts, a preliminary retrospective analysis was undertaken to verify whether policosanol added to AS within 30 days of stroke onset, is better than placebo + AS for the six and 12 months recovery of ischemic stroke patients included.

II. MATERIALS AND METHODS

The present analysis includes the data of all patients included in ischemic stroke recovery studies.

Studies Design

Patients who suffered recent ischemic stroke (≤ 30 days before recruitment) and gave their informed written consent enrolled at external visits of the Institute of Neurology and Neurosurgery (Havana, Cuba). The independent Ethics Committee approved the studies protocols and the studies were registered in the Cuban Public Registry of Clinical Studies.
All participants underwent clinical history and full clinical examination. All the patients included in the study were indicated and received rehabilitation in their Polyclinics of residence and were recommended to follow a healthy lifestyle, with control of blood pressure, smoking cessation, low fat and calorie diet, systematic physical activity and eliminate alcohol consumption.

Eligible patients were randomized to policosanol + AS or placebo + AS for 6 and 12 months and attended to control visits at 3; 6, 9 and 12 months on treatment. Patients underwent general examination and neurological assessment at each visit, laboratory analyses at baseline and at 6 and 12 months on therapy, meanwhile we controlled treatment compliance and adverse events at each visit post randomization.

Studies Patients

Enrolled patients were ambulatory men and women over 40 years of age who had ischemic stroke (diagnosed by a neurologist) within the 30 days prior to enrolment.

The studies protocol defined stroke as the occurrence of focal clinical signs of central nervous system dysfunction of vascular origin that lasted for at least 24 hours. Ischemic stroke confirmed through clinical assessment and computerized axial tomography performed within the following 48 hours after stroke onset in patients were eligible for randomization if they had a modified Rankin Scale score (mRSs) of 2, 3 or 4. The exclusion criteria included suspected or confirmed haemorrhagic stroke, atrial fibrillation, other cardiac sources of embolism, subarachnoid haemorrhage, diastolic hypertension ≥ 110 mm Hg, cardiac valve diseases, history of myocardial infarction, instable angina or revacularisation surgery within the six months prior to the trial and previous consumption of policosanol or other lipid-lowering and antiplatelet drugs.

Treatment

Patients consumed policosanol+AS or placebo + AS once daily with the breakfast for 6 and 12 months. Keeping in mind that randomised controlled trials support the use of daily doses of AS (75–150 mg) for the prevention of vascular events in high-risk patients we used 125 mg/day. 

Good treatment compliance, assessed through counts of remainder tablets and patient’s interviews, was to consume at least 85 % of the scheduled tablets per period evaluated. Antiplatelet (different to policosanol and AS) or lipid-lowering drugs were not permit to use during the study.

No patients included in the study received rechannel treatment neither with rTPa nor with mechanical thrombectomy.

Studies Outcomes

Clinical response was defined in terms of stroke functional scale (mRSs), which measure patient disability. The primary outcome of this study was functional outcome measured by the mRSs, which assesses the outcome with scores that range from 0 to 6 (0 no symptoms; 1 no relevant disability despite symptoms, able to conduct all usual activities; 2 slight disability, unable to carry out all previous activities but able to conduct self-assistance; 3 moderate disability requiring some help, but able to walk without assistance; 4 moderate severe disability, unable to walk without assistance, and unable to attend body needs without assistance; 5 serious disability; bedridden, incontinent, and requiring constant care and attention; and 6 death). We assumed to obtain a higher rate of cases with a favourable stroke outcome (mRSs ≤1) than in the placebo/AS group. In addition, reduction of mean mRSs with policosanol/AS should be greater than with placebo/AS. To obtain mRSs ≤ 1 are considered as favourable post stroke outcomes.

The modified Rankin neurological scale (mRSs) was always applied and evaluated by the principal investigator. All the patients included in the study had mRSs before the ischemic stroke of 0.

Decreases on LDL-C, total cholesterol and increases on HDL-C levels were secondary outcomes.

Laboratory Analyses

Venous blood samples were taken following a fasting of 12 hours. Plasma was separated from red blood cells by centrifugation at 4°C and 2000 x g for 10 min, and aliquots were immediately taken. Lab analyses were performed within the next 8 hours after blood drawing.

Lipid Profile and Blood Safety Indicators

Serum lipids levels as well as blood biochemistry (alanine amino transferase-ALT; aspartate amino transferase-AST; glucose and creatinine) indicators were determined using reagent kits (Roche, Basel, Switzerland) in a Hitachi 719 autoanalyzer (Tokyo, Japan) of the Clinical Laboratory.

Safety and Tolerability Assessment

Safety and tolerability indicators included laboratory and physical examination data, and adverse events (AE) reports. Study protocol defined an AE as any undesirable experience, absent at hospital discharge or worsened thereafter, happening in a patient, independently if it could be or not related with the therapy. AE were classified as mild, moderate or serious according to their intensity. Mild AE should not require stopping of study medications or specific treatment of the AE, moderate AE should require the withdrawal of study medications and/or treatment of the AE, while serious AE should lead to patient hospitalization and/or to death.

Statistical Analysis

The study was designed to have a statistical power of 80 % to detect a reduction of 30 % in the frequency of policosanol+AS cases with a favourable outcome as compared to the placebo+AS group, with a two-sided significance level of p<0.05. We analyzed the data on an intention-to-treat basis, including those of all patients who underwent randomization. Continuous values were compared with the t test for paired (within group comparisons) and independent (between group comparisons) samples, and the Bonferroni’s test was used to adjust significances from repeat comparisons. Categorical data were compared with the Chi square test. All p values
were two-sided.

III. RESULTS

Population characteristics

Two hundred and seventy one patients enrolled (mean age: 67 years) (135 men, 136 women) were eligible for randomization. One hundred and fifty three patients (77 policosanol and 76 placebo) were included in the six months studies and one hundred eighteen patients (59 policosanol and 59 placebo) were included in the long-term studies (12 months).

Twenty nine patients (16 placebo+AS, 13 policosanol+AS) discontinued prematurely the trials, because of serious adverse events (6 placebo+AS, 2 policosanol+AS), travels abroad (3 placebo+AS group, 5 policosanol+AS), protocols violations (2 placebo+AS, 2 policosanol+AS), unwillingness to follow-up (5 placebo+AS, 3 policosanol+AS) and change of localization (1 policosanol+AS).

Baseline characteristics were well balanced in the two groups (Table I). The most frequent (≥ 20 %) risk factors at baseline were hypertension (93.4 %), over weight + obesity (62.4 %), smoking (39.5 %), hypercholesterolemia (18.8 %) and diabetes (16.6 %). Concomitant therapy was also well matched in both groups, the most frequent being the angiotensin converting enzyme inhibitors (ACEI) (74.9 %).

Effects on Stroke Functional Outcomes

Table II shows the distribution of patients into different mRSs values at baseline, and after 3, 6, 9 and 12 months on treatment. Baseline values were similar in both groups. In all comparisons, more patients treated with policosanol+AS than with placebo+AS achieved mRSs ≤1.

At the six months more policosanol+AS (117/136, 86.0 %) than placebo+AS patients (105/135, 73.7 %) achieved mRSs goals (p<0.001). At the 12 months of the study more policosanol+AS (50/59, 84.7 %) than placebo+AS patients (5/59, 8.5 %) achieved mRSs goals (p<0.001).

Safety and Tolerability

According to the effects on physical and blood safety indicators, treatments were safe and well tolerated (data not shown for simplicity). Systolic and diastolic pressure significantly decreased in the group treated with policosanol+AS decreased persistently and significantly LDL-C, final reduction was 21.6 %, and the same happened with total cholesterol, final decrease was 12.5 %. In turn, the treatment increased HDL-C by 6.3 % (Table IV). Policosanol+AS failed to modify triglycerides.

Table II lists the effects on functional stroke scale. Treatment with policosanol+AS significantly decreased mean mRSs from the first interim check-up (p<0.0001 vs placebo+AS). The treatment effect did not wear off, even improved, after 6 and 12 months therapy (p<0.0001 vs placebo+AS) when the net decrease versus placebo+AS was 56 % and 70.8 %, respectively.

Table III lists the effects on functional stroke scale. Treatment with policosanol+AS significantly decreased mean mRSs from the first interim check-up (p<0.0001 vs placebo+AS). The treatment effect did not wear off, even improved, after 6 and 12 months therapy (p<0.0001 vs placebo+AS) when the net decrease versus placebo+AS was 56 % and 70.8 %, respectively.

discussion

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policosanol/AS as compared to placebo/AS throughout the study, but individual values were within normal limits. The treatment did not modify any other physical or blood safety indicator (alanine amino transferase-ALT, aspartate amino transferase-AST, glucose and creatinine) versus placebo/AS. Eight patients reported serious adverse events (6 placebo+AS, 2 policosanol+AS). In addition, other 13 patients (8 placebo+AS, 5 policosanol+AS) they reported moderate or mild adverse events (Table V).

### Table III. Distribution of cases in accordance to the Modified Ranking Scale score (mRSs)

<table>
<thead>
<tr>
<th>mRSs values</th>
<th>Poli+AS (n=136)</th>
<th>Pla+AS (n=135)</th>
<th>Poli+ASA (n=136)</th>
<th>Pla+ASA (n=135)</th>
<th>Poli+AS (n=59)</th>
<th>Pla+AS (n=59)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0</td>
<td>0</td>
<td>11^+</td>
<td>0</td>
<td>3^+</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
<td>106^+</td>
<td>10</td>
<td>40^+</td>
<td>5</td>
</tr>
<tr>
<td>2-3</td>
<td>129</td>
<td>133</td>
<td>17^++</td>
<td>121</td>
<td>0^++</td>
<td>45</td>
</tr>
<tr>
<td>4</td>
<td>7</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Data presented as n (number of cases), Poli policosanol, AS aspirin

^p < 0.01, ^p < 0.001, Comparisons versus placebo/AS (χ² test)

### Table IV. Effects on lipid profile (X±SD)

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Baseline</th>
<th>6 months</th>
<th>12 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo+AS</td>
<td>3.47 ± 1.01</td>
<td>3.60 ± 0.98</td>
<td>3.82 ± 1.15</td>
</tr>
<tr>
<td>Policosanol+AS</td>
<td>3.56 ± 0.94</td>
<td>2.78 ± 1.05^***</td>
<td>2.79 ± 0.81^***</td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo+AS</td>
<td>5.73 ± 1.25</td>
<td>5.80 ± 1.12</td>
<td>5.73 ± 1.16</td>
</tr>
<tr>
<td>Policosanol+AS</td>
<td>5.78 ± 1.12</td>
<td>5.40 ± 1.15^***</td>
<td>5.06 ± 0.96^</td>
</tr>
<tr>
<td>HDL-C (mmol/L)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo+AS</td>
<td>1.42 ± 0.38</td>
<td>1.41 ± 0.34</td>
<td>1.38 ± 0.35</td>
</tr>
<tr>
<td>Policosanol+AS</td>
<td>1.44 ± 0.37</td>
<td>1.50 ± 0.36^</td>
<td>1.53 ± 0.41^</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo+AS</td>
<td>1.78 ± 0.94</td>
<td>1.81 ± 0.89</td>
<td>1.83 ± 0.59</td>
</tr>
<tr>
<td>Policosanol+AS</td>
<td>1.76 ± 0.80</td>
<td>1.74 ± 0.98</td>
<td>1.70 ± 0.97</td>
</tr>
</tbody>
</table>

X mean, SD standard deviation, AS aspirin

^p < 0.05, ^p < 0.01, ^p < 0.001 Comparison vs baseline (t test for paired samples)

*p < 0.05, *p < 0.01 Comparison vs placebo/AS (t test for independent samples)

### Table V. Serious adverse events reported during the study

<table>
<thead>
<tr>
<th>Adverse events (AE)</th>
<th>Placebo (n=135)</th>
<th>Policosanol (n=136)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serious adverse events (SAE)</td>
<td>n %</td>
<td>n %</td>
</tr>
<tr>
<td>Recurrent stroke</td>
<td>3 2.2</td>
<td>2 1.5</td>
</tr>
<tr>
<td>Intestinal occlusion-surgery</td>
<td>1 0.7</td>
<td>0 0.0</td>
</tr>
<tr>
<td>Heart attack</td>
<td>1 0.7</td>
<td>0 0.0</td>
</tr>
<tr>
<td>Respiratory failure</td>
<td>1 0.7</td>
<td>0 0.0</td>
</tr>
<tr>
<td>Total of patients who reported SAE</td>
<td>6 4.4</td>
<td>2 1.5</td>
</tr>
<tr>
<td>Moderate or mild AE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insomnia</td>
<td>1 0.7</td>
<td>4 2.9</td>
</tr>
<tr>
<td>Gastric discomfort</td>
<td>4 3.0</td>
<td>0 0.0</td>
</tr>
<tr>
<td>Chest pain</td>
<td>1 0.7</td>
<td>0 0.0</td>
</tr>
<tr>
<td>Behavioural troubles</td>
<td>1 0.7</td>
<td>0 0.0</td>
</tr>
<tr>
<td>Asthenia</td>
<td>1 0.7</td>
<td>0 0.0</td>
</tr>
<tr>
<td>Fever episodes</td>
<td>0 0.0</td>
<td>1 0.7</td>
</tr>
<tr>
<td>Total of patients who reported moderate or mild AE</td>
<td>8 5.9</td>
<td>5 3.7</td>
</tr>
</tbody>
</table>

n number of patients, no significant (χ² test)

### IV. DISCUSSION

Study patients were randomized within 30 days of the onset of the ischemic stroke, so that the effects of policosanol/AS cannot be interpreted as effects on the acute stroke, but on the further recovery step. Following the recommendations for ischemic stroke management, all patients received AS early on their admission in stroke unit and followed on the thereafter. 7-10 Our study group was restricted to have 2 to 4 mRSs values for lowering the influence of variable stroke severity on the results. Study patients had not been received policosanol before being randomized, so that they were technically virgin to study treatment.

The strength of the study includes that it was randomized, double-blinded and placebo-controlled, with all patients receiving AS, first-line therapy recommended after ischemic stroke. Since both groups were homogeneous at baseline the effects here found can be attributable to policosanol+AS therapy. In particular, the mean mRSs values were comparable in the two groups. Also, the fact that treatment compliance was very good (≥ 85 %) and comparable in both groups.
supports the validity of the present results.

Baseline characteristics of study patients match well with stroke epidemiological data. The mean age of patients, and the high frequency of concomitant morbidities were consistent with common stroke risk factors. In addition to AS, consumed by all patients, the most frequent concomitant drugs were ACEI, but such consumption, coherent with the prevalence of hypertension, was also similar in the two groups, so that we discard the potential influence of concomitant therapy to the present results.

We assessed the effects on stroke outcome by measuring the functional status and degree of functional dependence of the patients with the mRSs, scales used widely to assess post-stroke functional impairment. In particular, mRSs is the clinical outcome tool most widely used for stroke recovery in clinical studies.28,29,32

The present results confirms that the addition of policosanol to conventional AS therapy after hospital discharge should help the neurological recovery post-ischemic stroke. This concept is supported by the proportion of policosanol+AS patients who achieved a good stroke outcome (mRS≤1) at study completion and the mean reduction (56 % and 70.8 % after 6 and 12 months therapy, respectively) of mRSs, the primary study outcome, as compared to placebo+AS. These results are consistent with the efficacy of policosanol+AS demonstrated in previous randomized, double-blind controlled studies in which the control group received placebo+AS and the net decrease of the mean mRSs here seen at month 3 agrees with those found in previous placebo controlled studies.19,20

In addition, policosanol+AS reduced significantly LDL-C (31.2 %) and total cholesterol (12 %), and increased HDL-C (5.7 %), the lipid-modifying effects here seen are coherent with previous data in post-stroke patients,19-26 and with the general lipid-lowering profile of policosanol.33,39

The mechanism(s) whereby policosanol may help to improve stroke recovery are beyond the objective of this study. Nevertheless, antiplatelet effects of policosanol40-42 should be responsible, at least partly, of the benefits of policosanol/AS therapy on stroke outcomes over the conventional AS therapy. In such regard, a previous 6 months clinical study conducted in patients who had suffered non-cardioembolic ischemic stroke demonstrated that the antiplatelet efficacy of policosanol+AS was better than that of placebo+AS.19 Another study demonstrated that it inhibits cyclooxygenase 1 (COX-1) activity in vitro, which makes rationale that it may inhibit platelet aggregation.43

Lipid lowering drugs lowers the stroke risk.11,22 Greater reductions in stroke risk are associated with higher LDL-C decreases.11 In a large meta-analysis that included data of 113000 patients, statin therapy at stroke onset was associated with improved outcome.44

In this sense also beneficial effects of policosanol on serum lipids (LDL-C and TC decrease, HDL-C increase), may contribute to the benefits of policosanol+AS on stroke outcomes since LDL-C reduction and HDL-C increase are linked to stroke recovery and prevention.11,45

The pretreatment with statins, hypercholesterolemia or both in ischaemic stroke patients could have neuro-protective effects with reduced neurological deficits at presentation, lower early death and dependency rate, thus increasing the chances for good outcome.36

Moreover, Policosanol (20 mg/day) and atorvastatin (20 mg/day), administered for 12 weeks within the next 30 days after stroke onset, were similarly effective for improving the functional outcome in patients with recent ischemic stroke, all treated with AS.21

The cholesterol-lowering activity of policosanol involves the inhibition of cholesterol synthesis by regulating HMG-CoA reductase through activation of AMP-kinase,47,48 the main regulatory kinase for HMG-CoA reductase. Policosanol treatment of hepatoma cells increased AMP-kinase phosphorylation, providing a clue by which it might down-regulate HMG-CoA reductase activity and decrease cholesterol synthesis without directly inhibiting the enzyme, since AMP-kinase.47 Further studies demonstrated that metabolic transformation of very long chain alcohols to fatty acids is needed for the suppression of cholesterol synthesis, presumably by increasing cellular AMP levels.48 In turn, the mechanism(s) responsible of HDL-C elevation by policosanol have not been demonstrated. Recent studies have proven that policosanol enhances HDL functionality improving anti-glycation, anti-apoptosis, and cholesteryl ester transfer inhibition in vitro.49,50

In agreement with previous studies, policosanol+AS was safe and well tolerated. The decrease of systolic and diastolic blood pressure seen in policosanol+AS group is consistent with some previous data,19,26,34 indicating an additional lowering pressure effect of policosanol. Such additive effects on arterial pressure must be in relation with pleiotropic effects of policosanol, mainly those supporting beneficial effects on endothelial function.

Finally, we must highlight the favorable results obtained in neurological recovery and the recurrence of events in a follow-up study that evaluated the benefits of policosanol administered from hospital discharge up to 5 years later in 55 patients of both sexes with an accident ischemic stroke that had previously undergone transient ischemic attacks. The neurological score improved significantly and progressively up to the first year after the stroke, and persisted thereafter up to the 5 years post-stroke. No patient died during the trial. Fifty patients (90.9 %) did not experience a new vascular event, 1/55 (1.8 %) suffered a new stroke and 4 (7.3 %) experienced a new transient ischemic attacks and no other serious AE occurred during the trial.25

V. CONCLUSIONS

The preliminary retrospective analysis of the effects of policosanol on ischemic stroke patients indicate that the treatment for 6 and 12 months with policosanol+aspirin proved to be more effective than the placebo+aspirin treatment in the functional recovery of these patients.

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