CASE REPORT

Motor improvement in cerebellar ataxia after integral rehabilitation

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Abstract: Cerebellar ataxia is a complex motor impairment arising from neurologic disease. The dysfunction significantly impairs quality of movement, equilibrium, and gait. In this study, we evaluated motor function in 20 patients with acquired or degenerative ataxia undergoing rehabilitation using a program developed at the International Center for Restorative Neurology in Havana. All patients were evaluated before and 28 and 42 days after treatment using the International Cooperative Ataxia Rating Scale (ICARS) and measurement of maximal strength. Analysis of variance demonstrated a significant reduction in ICARS score, indicating reduction in the severity of ataxia. A highly significant correlation was found between change in ICARS score and increase in lower limb strength. The program developed at the International Center for Restorative Neurology in Havana for rehabilitation of ataxia was shown to be effective for ameliorating motor impairment in patients with ataxia, independent of its etiology.

Keywords: ataxia, rehabilitation, motor recovery, strength, plasticity

Introduction

Cerebellar ataxia is a common consequence of neurologic disease, with more than 400 types having been described.¹ The disease leads to progressive impairment, primarily affecting posture and equilibrium, along with impaired limb movement (both superior and inferior), oculomotor control, and gait. These difficulties affect the life of the patient and that of relatives, constituting a huge economic burden.²⁻⁴

There are at present no effective drugs available to treat ataxia.^{5,6} Rehabilitation is, therefore, an essential tool when aiming to recover motor function or to reduce the level of impairment.^{3,7–9} Several programs have been designed to rehabilitate patients with ataxia, based on neural plasticity and the adaptive abilities of the adult nervous system.1,8,10

The International Center for Neurological Restoration (CIREN) in Havana has implemented an intensive multidisciplinary program for the treatment of neurologic sequelae, including ataxia. It consists of a group of training and fitness activities under the control of different therapists and physicians aimed at reacquisition of motor abilities.10

The results of a pilot study in a small group of patients with ataxia suggested a positive impact of our restorative program on motor control. The present study was performed to confirm or refute those results in a larger group of patients who came to our institution seeking to improve their neurologic status and living conditions.

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Subjects and methods

Twenty patients diagnosed with acquired or degenerative cerebellar ataxia and treated at CIREN from September 2009 to April 2010 were included in the study. All patients were evaluated and diagnosed by a neurologic team (Table 1) and entered in the study after signing their agreement. Patients with concomitant brain lesions, terminal chronic disease, severe cognitive impairment (Mini-Mental State Examination score <14), or mental retardation were excluded, along with those who were illiterate or had clinical signs of aphasia, apraxia, agnosia, severe depression, or any other psychiatric condition precluding effective interaction with the therapist.

All patients underwent 42 days of neurorestorative treatment following the steps defined in the program, ie, general physical condition, specific physical condition, prefunctional, training and functional training (Table 2). The treatment program is based on a multifactorial, intensive, and personalized strategy. The daily schedule typically includes 5 hours of physical therapy, one hour of occupational therapy, and one hour of speech therapy, comprising 7 hours of guided activity per day. Importantly, our intention was to evaluate the efficacy of the program and that of any particular component of it.

No control group was included. One reason for this is that patients come to our institution for a limited time and pay for

Table I Description of the patient sample included in the study

Patient	Age	Sex	Etiology	Classification
	(years)			
I	21	М	Medulloblastoma	Acquired
2	26	F	TBI	Acquired
3	23	М	TBI	Acquired
4	31	М	TBI	Acquired
5	31	М	TBI	Acquired
6	29	М	TBI	Acquired
7	51	М	Medulloblastoma	Acquired
8	27	F	Hypoxia	Acquired
9	26	М	TBI	Acquired
10	45	F	TBI	Acquired
11	24	М	Cerebral palsy	Acquired
12	24	М	TBI	Acquired
13	25	М	Angioma	Acquired
14	21	М	TBI	Acquired
15	19	F	Friedreich's ataxia	Hereditary
16	23	М	SCA	Hereditary
17	16	М	Friedreich's ataxia	Hereditary
18	30	F	Friedreich's ataxia	Hereditary
19	22	М	Friedreich's ataxia	Hereditary
20	36	Μ	SCA7	Hereditary

Abbreviations: TBI, traumatic brain injury; SCA, spinocerebellar ataxia; M, male; F, female.

Table 2 Overview of th	e physical rehabilitation	program for		
ataxia at the International Center for Neurological Restoration				

Step	Goals	Activities
General	Reduction of	Thermotherapy
physical	osteomyoarticular retractions	Therapeutic massage
condition	Improve muscle tone	Breathing exercises
	Increase respiratory capacity Increase general fitness and working capacity	Passive, active, and resisted mobilizations Ideomotor training Exercises to reduce
		rigidity
		Exercises for general physical condition
Specific	Consolidate achievements	Coordination exercise
physical	of the previous step	Exercises for posture
condition	Improve coordination	Exercises with weight
	(proprioception, equilibrium,	Mechanotherapy
	rhythm, and precision)	(step simulator, static
	Improve posture Increase force	bicycle)
Prefunctional	Consolidate previous	Training of gait
training	achievements	Body transfers
0	Training in static and	Static equilibrium
	dynamic gait patterns Increase resistance	Mechanotherapy
Functional	Consolidate previous	Dynamic equilibrium
training	achievements	Coordination training
-	Improve equilibrium,	(presport activities
	coordination, and rhythm	with balls)
	Improve gait	Walking, trotting, and running

their treatment. Another aspect to consider is that all patients included in the study had a long history of ataxia without signs of spontaneous recovery, making it plausible to consider any improvement a consequence of the treatment.

We used the International Cooperative Ataxia Rating Scale (ICARS, Table 3) to evaluate cerebellar dysfunction. The ICARS scale is useful for evaluation of severity and temporal changes in patients with ataxia.^{11,12} Two external evaluators (both experienced neurologists) not involved in administration of therapy applied the ICARS scale before and at two time points (28 and 42 days) after starting rehabilitation. The validity of the results was confirmed using the Cronbach's alpha coefficient.

A maximum strength score was also used for the lower limbs. Measurements were carried out by an external independent expert in physical therapy following the method described by Kraemer and Fry.¹¹ After general and lower limb warm-up, external weights were applied during hip flexion, extension, abduction, and adduction, and during knee extension on the horizontal press and on the quadriceps bench. The weight was steadily increased until the patient

Table 3 International Cooperative Ataxia Rating Scale (2001)

Subscale	ltem	Score range
Posture and	Walking capacities: patient's gait is observed during a 10 m walking test, including a half turn, near a wall at	0–8
gait disturbances	about 1.5 m Gait speed: patients who score 1–3 on the preceding test are observed for gait speed; those who scored 4 or	0-4
	more on the preceding test automatically score 4 in this test	
	Standing capacities, eyes open: the patient is asked first to try to stand on one foot; if impossible, to stand	0–6
	with feet in the tandem position; if impossible, to stand with feet together; for the natural position, the patient	
	is asked to find a comfortable standing position	
	Spread of feet in natural position without support and eyes open: the patient is asked to find a comfortable	0-4
	standing position, and the distance between the medial malleoli is then measured	
	Body sways with feet together, eyes open	0-4
	Body sways with feet together, eyes closed	0-4
	Quality of sitting position (thighs together, on a hard surface, arms folded)	0-4
Total category		34
Kinetic functions	Knee-tibia test (decomposition of movement and intention tremor): the test is performed in the supine	0-4
	position so that visual control is possible; the patient is requested to raise one leg and place the heel on the	
	opposite knee, and then slide the heel down the anterior tibial surface toward the ankle; on reaching the	
	ankle joint, the leg is raised again to a height of approximately 40 cm and the action is repeated; at least three	
	movements of each limb must be performed for proper assessment	
	Action tremor in the heel-to-knee test: during the same test as the preceding one, the action tremor of the	0-4
	heel on the knee is observed specifically while the patient holds the heel on the knee for a few seconds before	
	sliding it down the anterior tibial surface; visual control is required	
	Finger-to-nose test (decomposition and dysmetria): the subject sits in a chair; the hand is resting on the knee	0-4
	before the beginning of the movement; visual control is required; three movements of each limb must be	
	performed for proper assessment	
	Finger-to-nose test (intention tremor of the finger): the studied tremor is that appearing during the ballistic	0-4
	phase of the movement; the patient is sitting comfortably with the hand resting on his/her thigh; visual control	
	is required; three movements of each limb must be performed for proper assessment	
	Finger-finger test (action tremor and/or instability): the seated patient is asked to maintain his/her index	0-4
	fingers pointing at each other for about 10 seconds, at a distance of 1 cm, at the level of the thorax, under visual control	
	Pronation-supination alternating movements: the subject, comfortably seated on a chair, is asked to raise	0-4
	his/her forearm vertically and to make alternating pronating and supinating movements of the hand; each	
	hand is moved and assessed separately	
	Drawing of the Archimedes spiral on a predrawn pattern: the subject is comfortably settled in front of a	0-4
	table, with a sheet of paper fixed to avoid artifacts; the subject is asked to perform the task without timing	
	requirements; the same conditions of examination must be used at each examination (eg, same table, same	
	pen); the dominant hand is examined	
Total category		52
Speech	Dysarthria (fluency of speech): the patient is asked to repeat a standard sentence several times, always the	
disorders	same, eg, "A mischievous spectacle in Czechoslovakia" Dysarthria (clarity of speech)	
Total category		8
Oculomotor	Gaze-evoked nystagmus: the subject is asked to look laterally at the finger of the examiner; the movements	
disorders	assessed are mainly horizontal, but they may be oblique, rotatory, or vertical	
	Abnormalities of ocular pursuit: the subject is asked to follow a slow lateral movement performed by the	
	finger of the examiner	
	Dysmetria of the saccade: two index fingers of the examiner are placed in each temporal visual field of the	
	patient, whose eyes are in the primary position; the patient is then asked to look laterally at the finger,	
	on the right and on the left; the average overshoot or undershoot of the two sides is then estimated	
Total category		6
Total scale		100

was unable to perform the task. The final score was the sum of all weights reached for each muscle measured.

Results

Significant changes on ICARS scale

Statistical data processing was performed by analysis of variance and regression analysis. P < 0.05 was considered to be statistically significant.

Table 4 summarizes the scores on the ICARS scale for each of the time points of evaluation (before treatment, and 28 days and 42 days after initiation of rehabilitation). Repeated-measures

Table 4 ICARS score before and after treatment

Scale (maximum	Before treatment	After 28 days	After 42 days	
score)	Mean ± SEM (CI)	Mean ± SEM (CI)	Mean ± SEM (CI)	
Posture	22.4 ± 1.59	19.35 ± 1.73	18.8 ± 1.91	
and gait	(19.27–25.52)	(15.95–22.74)*	(15.06–22.53)*	
disturbances (34)				
Kinetic	$\textbf{18.8} \pm \textbf{2.03}$	14.75 ± 1.87	13.15 ± 1.41	
function (52)	(14.80-22.79)	(11.07–18.42)*	(10.39–15.90)*	
Speech	$\textbf{2.35} \pm \textbf{0.27}$	2 ± 0.21	$\textbf{1.9}\pm\textbf{0.20}$	
disorders (8)	(1.81–2.88)	(1.59–2.40)	(1.50–2.29)*	
Oculomotor	$\textbf{1.85}\pm\textbf{0.35}$	1.6 ± 0.31	$\textbf{1.6}\pm0.30$	
disorders (6)	(1.16–2.53)	(0.99–2.20)	(1.01-2.19)	
Total ICARS	$\textbf{45.4} \pm \textbf{3.43}$	$\textbf{37.7} \pm \textbf{3.16}$	$\textbf{35.45} \pm \textbf{2.93}$	
score (100)	(38.66–52.14)	(31.50–43.89)*	(29.70–41.19)*	

Notes: *p < 0.05 Tukey's (honest significant difference) test (horizontal comparisons); alpha = 0.05.

Abbreviations: Cl, confidence interval; ICARS, International Cooperative Ataxia Rating Scale; SEM, standard error of the mean.

analysis of variance confirmed a significant decrease in total ICARS score at 28 and 42 days ($F_{2,36} = 16.9977$, P < 0.05, see Figure 1), indicating a reduction in the severity of ataxia. Tukey's post hoc test confirmed significant differences between the initial score and both post training evaluations, but not between the post training scores.

Most important improvements seen in posture, gait, and kinetic function subscales

A detailed analysis, considering each subscale of the ICARS scale, allowed more precise evaluation of the results. Repeated-measures analysis of variance demonstrated that treatment led to a significant reduction in the subscale measuring posture and gait ($F_{2,38} = 7.9344$, P < 0.05, see Figure 2). Tukey's post hoc test confirmed significant differences between pretreatment evaluation and measurements during and after treatment, but no differences between the two post training scores. Items showing the most improvement were those related to posture and equilibrium, ie, body oscillations with eyes closed or opened and the quality of the sitting position, but no improvements in gait speed were noted.

The subscale for kinetic function also showed significant improvement (repeated-measures analysis of variance, $F_{2,38} = 15.66580$, P < 0.05, see Figure 3). Tukey's post hoc test confirmed significant differences between the before and after treatment evaluations, but not between the two post training scores. Action tremor showed a significant reduction, but dysmetria in the upper limbs showed no improvement.

Average ICARS score

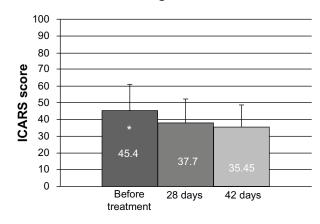


Figure I Modifications in ICARS score after rehabilitation treatment. **Notes:** Mean \pm standard error of the mean (*significant difference, P < 0.05 Tukey's test).

Abbreviation: ICARS, International Cooperative Ataxia Rating Scale.

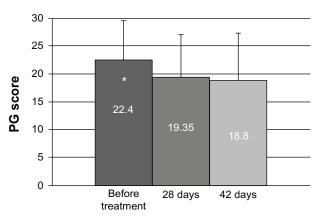


Figure 2 Modifications in the subscale for PG after rehabilitation treatment. Notes: Mean \pm standard error of the mean (*significant difference, P < 0.05, Tukey's test).

Abbreviation: PG, posture and gait.

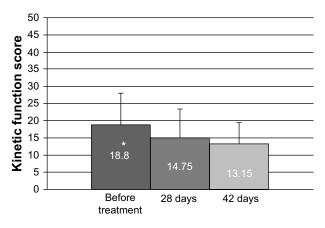


Figure 3 Modifications in the subscale for Kinetic functions after rehabilitation treatment.

Notes: Mean \pm standard error of the mean (*significant difference, P < 0.05, Tukey's test).

Abbreviation: MF, motor function.

Rehabilitation also brought about significant amelioration of language impairment, as shown by a global reduction in scores on this subscale (repeated-measures analysis of variance, $F_{2,38} = 3.6062$), but no significant changes were observed on the oculomotor scale (repeated-measures analysis of variance, $F_{2,38} = 3.51852$).

Correlation analysis

Analyzing the relationship between improvement and other independent variables, no significant relationships were found between global ICARS improvement (final value/ initial value percent) and the etiology of ataxia (hereditary or acquired, one-way analysis of variance, $F_{1,18} = 0.0217$). Regression analysis showed that the initial level of cognitive impairment ($R^2 = 0.13174881$), age ($R^2 = 0.02792980$), and time of development of ataxia ($R^2 = 0.01929383$) had no influence on the outcome of rehabilitation.

On the other hand, one-way analysis of variance showed that treatment significantly modified maximal strength in the lower limbs ($F_{2,38} = 38.40207$, P < 0.05, see Figure 4). These results were confirmed by the highly significant relationship found on regression analysis between the change in ICARS score and maximal strength ($R^2 = 0.99919140$).

In summary, this neurorestorative program brought about improvement in global scores for ataxia in our study patients, which appeared to be independent of the etiology of ataxia. The most important improvements were observed in posture, equilibrium, intention tremor, language, and muscle strength in the lower limbs, while gait, dysmetria, and oculomotor control showed no relevant amelioration.

Discussion

Our results confirm that neurorehabilitation can be a useful tool for treating motor impairment caused by cerebellar ataxia.^{3,10,14,15}

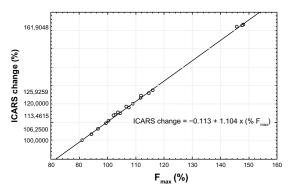


Figure 4 Scatter plot and regression curve for the relationship between change in ICARS score after treatment (% ICARS change) and change in maximal force (% F_{max}). **Abbreviation:** ICARS, International Cooperative Ataxia Rating Scale.

However, these results are difficult to compare with those reported by others because of differences in conception, duration, intensity, patient selection, and outcome measures used, as well as use of combined therapies, among other factors.^{14–20}

The beneficial effect of the program used in this study is reflected in a significant reduction of the global ICARS score. The intensive rehabilitation strategy used in our program induced significant changes after 28 days of treatment, but extending it for 15 additional days did not result in further significant improvement. Short-term improvement after rehabilitation in patients with ataxia has been demonstrated in other studies, and although the benefits decay with time, gains can last for long periods.^{21,22}

All subscales of the ICARS, except for oculomotor control, improved after treatment, while some items showed no significant change. The posture and gait section showed improvement in the posture and equilibrium items, but not for gait speed. On the motor function scale, tremor was improved but not dysmetria.

Our study shows a strong positive relationship between increase in strength and improvement in motor control, in agreement with previous reports.^{23,24} Increased strength in the lower limbs would certainly contribute to increased stability and postural control, as suggested by the significant improvement in the posture and gait subscale.

Gait is a complex motor behavior usually learned during early childhood.²⁵ Reacquisition of gait in older age is extremely complex and difficult.²⁶ Effective treatment of gait disturbance requires development of compensation, strength, and equilibrium, all of which are affected by ataxia and the reduction in motor activity caused by the disease. According to the literature, modest improvements in gait have been obtained only after long-term rehabilitation (6 months or longer),^{25,27} far beyond the time interval tested in the present study. Language alterations are well documented in ataxia, but the effects of rehabilitation have been discrete²⁸ and are consistent with our results.

Neurorehabilitation ameliorates symptoms of ataxia, independent of its etiology,⁹ although less favorable results have been reported for ataxia attributable to degenerative disease, probably because of the progressive nature of the condition.^{21,24} It must be taken into consideration in our study that the number of patients with hereditary ataxia represented only about one third of the total patient group, which may have introduced some bias.

Conclusion

Our results confirm that intense neurorehabilitation is useful for treating and improving motor ability in patients suffering

from ataxia. Although the results point to the major contribution being a gain in muscle strength, intensive training is also likely to assist in relearning of motor abilities, and could also improve sensory feedback, favoring acquisition of motor skills.²⁹ In this regard, a role for neural plasticity mechanisms,^{30–32} which have been well studied and characterized in the cerebellum, ^{26,33} can also be invoked. The specific role of cerebellar plasticity in motor learning and motor control in humans is still being researched.

Disclosure

The authors report no conflicts of interest in this work.

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