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ABSTRACT

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SINDROME SHOSHINA-VASSILIEV NELLA PRATICA NEUROLOGICA: VENTUNESIMO ANNO DALLA SCOPERTA.

Riassunto

La nuova sindrome Shoshina-Vassiliev, descritta nel 1976, approvata ufficialmente e brevettata nel 1985, si riscontra più frequentemente in malati di paralisi cerebrale ed è caratterizzata da una specifica alterazione del ricambio di dopamina. La scoperta di questo meccanismo ha consentito di elaborare una cura efficace al 100%, con l'assunzione di microdosi di preparato contenente L-DOPA, tipo Nakom (Sinemet). La sospensione della cura, indipendentemente dalla sua durata, provoca il ritorno della malattia nel giro di 1-2 giorni (tetraplegia, anartria, strabismo, ecc.). Con la ripresa della cura, già dopo 1-3 ore si verifica il completo ristabilimento clinico. Il numero di questi malati non è inferiore all'1-2% soltanto dell'intero numero di pazienti affetti da paralisi cerebrale. L'autore ha individuato e curato più di trenta di tali pazienti, in età da 3 a 45 anni.

E' stato elaborato il metodo degli adrenogrammi secondo Vassiliev che permette di fare lo screening della sindrome Shoshina-Vassiliev; è stata elaborata la biocorrezione che dà risultati nel 100% dei casi, a volte già nel giro di poche ore dall'assunzione di microdosi del preparato (in particolare Nakom, Sinemet). Si è così ottenuta la triade: scoperta della nuova sindrome, conoscenza del suo meccanismo, efficacia di cura al 100%. La scoperta della nuova sindrome ha consentito all'autore di elaborare il metodo di biocorrezione di vari tipi di paralisi ad eziologia dopaminica: paralisi cerebrali, encefalopatie, malattie demielinizzanti, compresa la sindrome Vassiliev e la sclerosi laterale amiotrofica , le miopatie, le paralisi post-traumatiche, ecc.. Il numero dei pazienti guariti supera gli 800.

Parole chiave: sindrome Shoshina-Vassiliev - tests con 0,1 g. e 0,5 g. di L-DOPA secondo Vassiliev - adrenogrammi secondo Vassiliev - ricambio della dopamina - biocorrezione secondo Vassiliev - microdosi di preparato contenente L-DOPA - nuova triade: sindrome, meccanismo, cura.

The Shoshina-Vassiliev syndrome, described by the authors in 1976 and officially recognised in 1985, most frequently occurs in patients suffering from cerebral palsy[15-18,20]. It is of fundamental importance in the diagnosis and cure of a wide range of paralyses with dopamine aetiology, among which various diseases, thought to be incurable, may be included such as multiple sclerosis, myopathy, post-traumatic paralysis and epilepsy[2,3,5-13,19].

The syndrome is characterised by a specific abnormality of dopamine (DA) metabolism within the organism and, particularly important, by the surprising substitutive effect of some specific preparations which act upon dopaminergic neurone function (for example: Larodopa, Roche; Nakom, Merck; Sinemet, Du Pont).

To this end, we ourselves have elaborated, experimented and patented the Vassiliev biocorrection method[2-8] in which adrenograms are employed to define reactivity and functional and synthetic potential of the sympathetic adrenergic system (SAS) through a test with 0.1g of L-DOPA. Subsequently, a rapid clinical test was developed with 0.5 g of L-DOPA to screen patients with DA deficit and to assess the possibility of a cure with biocorrection and provide a clinical assessment of the data obtained (hypothalamic syndrome, attacks and paroxysms)[2,3,5-18,12]. Adrenograms provide a graphic representation of the dynamics of catecholamine urine excretion: adrenaline (A), noradrenaline (NA) and dopamine (DA) and their common precursor DOPA with regard to circadian rhythm along with the 0.1g L-DOPA test and a comparison with healthy subjects of the same age and sex. It is then possible to

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calculate strictly personalised minidoses of L-DOPA preparation. This calculation forms the basis of the Vassiliev biocorrection method and, in the case of the Shoshina-Vassiliev syndrome, is 100% effective in a very short time span (hours,days)[1,4-14].

The story of the discovery of the Shoshina-Vassiliev syndrome is particularly interesting considering the fact that the first attempts at treatment were carried out at a time when the exact pharmacokinetics of DA had not been sufficiently studied and in medical practice only preparations of pure L-DOPA were utilised (the use of L-DOPA with a prolonged effect in the form of Nakom and others was introduced at a later date).

Among the first cases, three examples may be cited which go back to 1976, with a detailed description by Shoshina of the clinico-neurological state revealed over a long observation period and neuro-hormonal test data which highlighted abnormal DA metabolism. In all three cases the patients presented with serious motor dysfunction before the age of 12 months. Intellectual development was excellent and language had developed adequately. As far as neurological state was concerned no abnormalities were revealed at the level of cervical innervation. Various pathologies were observed, however, in the motor sphere.

One of the three cases suffered from rigid hypokinetic syndrome (patient K), the second case from hypotonic hyperkinesis (patient M) and the third presented with a mixed syndrome (patient A). Thus we were faced with a typical clinico-neurological picture: intellect intact, impossibility in keeping the body upright and pyramidal and extra-pyramidal symptomatology.

Patient K had begun to show signs of reduced motor activity at the age of 7 months. Nevertheless, at 8 months she could sit and at 15 months she began to walk, though unstable, bending forwards and dragging the left leg, with the knee joint flexed.

At 2 years she was diagnosed as suffering from infant cerebral palsy (ICP) and appropriate therapy began, without success. At 5 years she could no longer walk, sit or keep her head upright. She was admitted to the clinic in May, 1976 at the age of 6 years. The cllinical features from a motor aspect included: unable to hold her head up, head falling forwards; unable to sit independently; unable to rotate the trunk; in passive movements upper and lower limbs were rigid like a "waxwork doll"; she lifted the arm with a series of jerks. The feet, clubbed, were heavily deformed, of a "Friedreich foot" type. She had muscular contractions of the tarsal joints (especially on the left), pronounced reflexes of the knee and extension; bilateral Babinski symptom; no dysfunction of internal organs; frequent respiratory problems.

Patient M showed the first symptoms of motor disorder at 6 months. At 9 months muscular weakness was evident: she could neither sit nor remain standing. The neurologist examined her at 14 months and diagnosed: myopathy, myasthenia and later, ICP. She was treated without success. The patient could neither sit nor walk.

She was admitted to the clinic in September, 1978 at the age of 6 years. Neurologically, she presented with the following symptomatology: unable to keep the head upright; muscular hypotonia of the legs and arms; reduced muscular strength proximally; limited movement of all joints, able to touch nose and eyes only with right hand, slight contraction of the left tibiotarsal joint; reduced tendon reflex; non-violent hyperkinesis, athetosis of the fingers (especially of the left hand), in the face muscles, particularly of the lips, opening and closing continuously and often wet from the tongue. She could not sit by herself nor remain standing. The internal organs were not affected but frequent respiratory illness was recorded.

Patient A was admitted to the clinic at 8 years with a diagnosis of ICP and double hemiplegia. the history of infancy was good with normal intellectual and language development for her age. She had begun to walk at 15 months but with a noticeable defect: the right leg dragged and the right hand was contracted and held close to the body. Neurological symptomatology remained unchanged up to the age of five and a half years. She was treated adequately. At five and a half years walking worsened and hand movements decreased. The feet were turned inwards and movement of the right tibiotarsal joint proved difficult. Tendon reflex was lively. Reflexes were pathological on both sides, particularly on the right. From the age of 6 she was completely immobile.

The adrenograms of these patients showed

up specific abnormalities in DA, NA and DOPA synthesis, in both free and bound forms. This prompted treatment with L-DOPA (Levodopum), commencing with a non-optimal dose taken at various times (tab.1, fig1,2). An increase in L-DOPA up to 500mg brought DA and NA synthesis back to within physiological limits and it was thus possible to include a preparation of L-DOPA in the treatment of patient K in December, 1976 and of patient M in September 1978. Patient K began with 125mg daily of a preparation of L-DOPA which was increased over 1 month to 750mg daily taken in three doses of 250mg and treatment continued for 1 year and 8 months. Patient M took 500mg of L-DOPA twice a day for 9 months. When patient A was admitted, the results of the first two patients had been analysed and Nakom had been introduced in pharmacotherapy so that on the basis of adrenogram data an optimal dose of 90mg of Nakom could be administered immediately (fig.3).

In each case, treatment with a preparation of L-DOPA gave surprising results: patient M, with hyperkinetic and hypotonic syndrome, actually began to move more rapidly; on the third day she began to keep her head upright, on the fifth day she began to sit and on the sixth day to remain standing with support. She exhibited a great desire to move about. In the first week athetosic hyperkinesis did worsen but disappeared later. Patient K, with rigid hypokinesis, began to move more gradually. Within 7 days of treatment with a preparation of L-DOPA the muscles became less rigid and the number of hand movements increased. After 20 days she began to hold her head up and to sit and after 40 days she could walk with support. The difficulty in movement of the tibiotarsal joint and the foot deformity completely disappeared after 12 months of treatment with a preparation of L-DOPA. Thus, neurological symptomatology was definitively removed in patients M (in 8 months) and K (in 12 months).

Patient A began to keep the head upright after 3 days, to sit after 4 days and to walk after 3 weeks.

After 18 months of treatment with 750mg daily of a preparation of L-DOPA, patient K began to show signs of the "on-off" syndrome so that for 3-4 hours she was unable to move. In this period her general condition deteriorated: she became irritable and it was thus decided to substitute the preparation of L-DOPA with Nakom. Patient M took 500mg of L-DOPA for only 8 months without any side effects.

The patients currently take Nakom once a day after breakfast (patient K has been taking 90mg since June,1978; patient M, 95mg since June, 1979 and patient A, 90mg) and their condition is satisfactory. They do not suffer from any neurological symptomatology or any pathology of the internal organs or

Tab.1 - Sympathetic-adrenergic activity in early patients with Shoshina-Vassiliev syndrome, in the control period, before introduction of preparation.

Patients		9 - 12				12 - 16				16 - 22				22 - 9			
		A	NA	DA	DOPA	A	NA	DA	DOPA	A	NA	DA	DOPA	A	NA	DA	DOPA
K	Free form	7.8	15.6	8.4	3.0	10.0	4.0	0	0	24.8	20.8	0	11.6	2.1	0	0	2.4
	Bound form	8.6	0	-	-	0	0	-	-	2.8	1.0	-	-	1.4	0	-	-
	Sum	16.4	15.6	-	-	10.0	4.0	-	-	27.6	21.8	-	-	3.5	0	-	-
M	Free form	10.8	15.0	0	0	7.2	4.8	80	5.6	4.4	0	48	0	1.1	2.4	39.2	* 0
	Bound form	10.8	0	-	-	0	0	-	-	1.6	0	-	-	2.4	0	-	-
	Sum	21.6	15.0	-	-	7.2	4.8	-	-	6.0	0	-	-	3.5	2.4	-	-
A	Free form	4.2	0	84	24.0	5.6	7.2	41	3.0	3.9	0	112	18.0	2.1	0	40	1.8
	Bound form	3.6	1.0	-	-	0	0.5	-	-	1.9	0	-	-	1.3	0	-	-
	Sum	7.8	1.0	-	-	5.6	7.7	-	-	5.8	0	-	-	3.4	0	-	-
Healthy subjects 5 - 10 years N 12	Free form	3.3	9.8	190	62	2.8	8.1	172	38	3.8	11.4	200	38	1.8	2.0	114	30
		±0.3	±1.2	±14	±8.0	±0.2	±0.9	±12	±7.3	±0.6	±0.1	±32	±6.8	±0.2	±0.4	±10	±6.0
	Bound form	2.4	4.3	-	-	4.7	3.2	-	-	4.0	6.7	-	-	2.4	0.7	-	-
		±0.3	±0.8			±1.4	±1.1			±1.3	±2.0			±0.8	±0.3		
	Sum	5.7	14.1	-	-	7.5	11.3	-	-	7.8	18.1	-	-	4.2	2.7	-	-
		±0.2	±0.7			±0.8	±0.6			±1.0	±1.6			±0.6	±0.2		

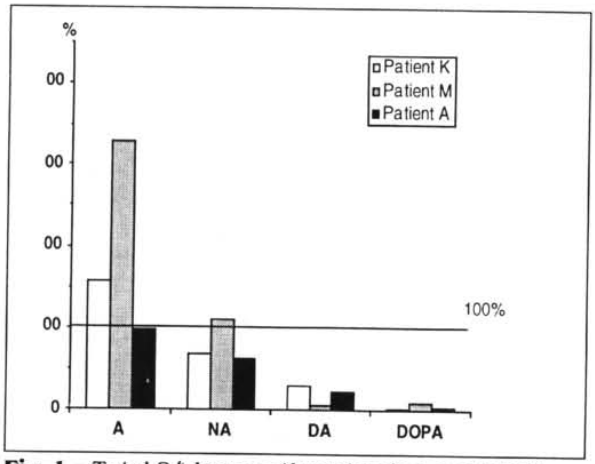

Fig.1 - Total 24-hour cathecolamine and DOPA excretion in patients with Shoshina-Vassiliev syndrome, compared with healthy subjects of the same age.

endocrine system. It is not possible to suspend treatment.

In September, 1982, during the dopamine turnover test, Nakom was suspended for 2 days. During the course of the first day, at about 10.00h, patient K began to feel weak in the legs and walking was difficult. She could not keep her head upright or remain seated on the bed or raise her legs. On clinical examination, muscular tone was unchanged, as was cervical innervation.On the third day, 90mg of Nakom were administered at 9.00h. Within an hour, movement completely recovered, without any neurological symptomatology. On the first day without Nakom, around 12.00h, patient M suffered weakness of the legs and arms and could hardly walk unaided. In the second half of the day she could not hold her head up and was able to walk only when supported. On the second day

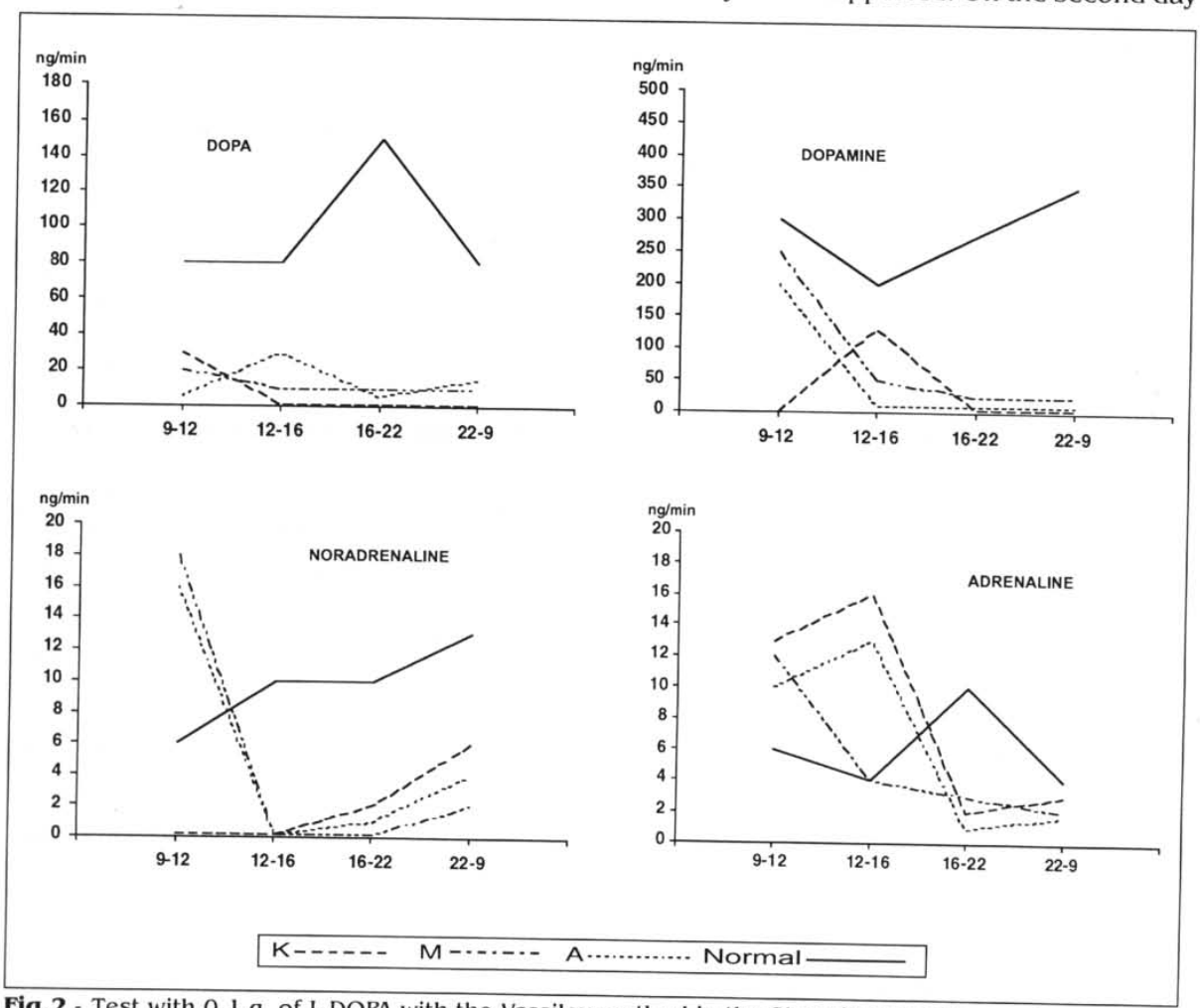

Fig.2 - Test with 0,1 g. of L-DOPA with the Vassilev method in the Shoshina-Vassiliev syndrome.

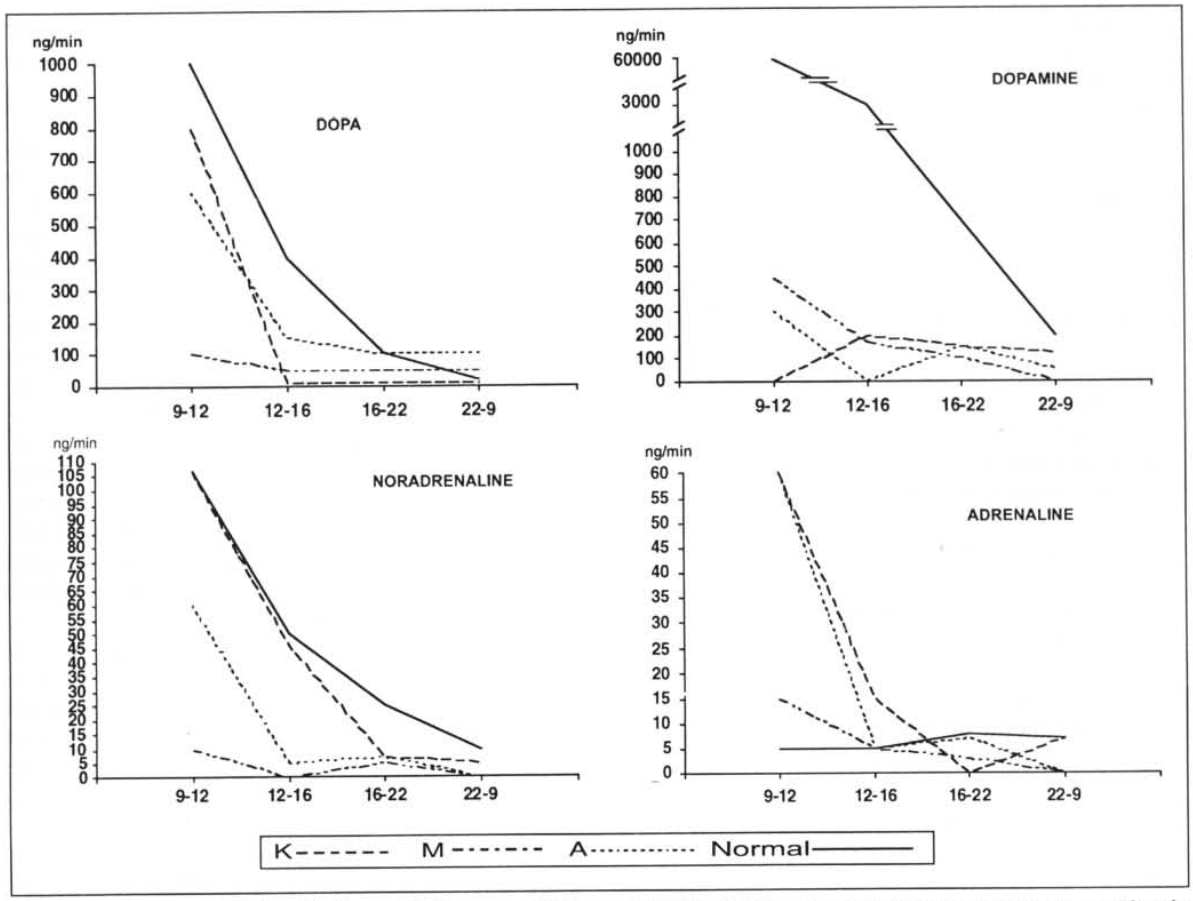

Fig.3 - Influence of introduction of 90 mg and 95 mg of Nakom on cathecolamine and DOPA synthesis in the Shoshina-Vassiliev syndrome.

weakness increased, the head hung down and the patient bent forwards. On getting up the trunk flexed abnormally backwards. There was no pathology at the level of cervical innervation. At 9.00h on the third day she was given 95mg of Nakom and was able to walk after 30 minutes with no weakness present.

The patients take a daily dose of 90mg and 95mg, respectively, of Nakom and report that the preparation begins to work at varying speeds depending on the day: strength and normal movement may return in half an hour, an hour or two hours. In summer and on sunny days the preparation acts more rapidly, within 20-30 minutes, while in the winter or on rainy days it may take 1-2 hours. The action of Nakom is influenced by activity in that when the patient begins to move around then the actual movements return to normal more rapidly. Positive emotions accelerate the action of Nakom while colds reduce its effects. The effects of Nakom initially last 9-10-12 hours and, later, as long as 14 hours. Patients normally take Nakom between 8.00h and 9.00h and, between 19.00h and 21.00h, muscular weakness and immobility may set in. On waking in the morning patients may move normally from half an hour to an hour and a half.

In tab. and fig.1,2 the results of the analysis of catecholamine and DOPA excretion in the patients under observation are reported. A sharp drop in DOPA and DA content may be observed, in both free and bound forms in practically all time intervals; this reduction is particularly evident in the analysis of 24-hour urine which indicates a block in DA and DOPA synthesis unlike NA synthesis and the tendency towards an increase in A synthesis (fig.1). The latter disorder may be due to an irritation of the surrenal glands, probably as a consequence of former drugs. This supposition was actually confirmed following an analysis of

other patients affected by the Shoshina-Vassiliev syndrome.

The 0.1g L-DOPA test confirmed the presence of a defect in DA synthesis along with the inability of the SAS, with the Shoshina-Vassiliev syndrome, to increase DOPA; this also showed that the DOPA administered is not actually eliminated in a non-metabolised form and would seem to suggest that it is deposited in tissues suffering from DA deficit (fig.2). This reaction is typical only of the Shoshina-Vassiliev syndrome and thus may be employed in the screening of these patients. The 0.1g L-DOPA test showed that patients affected by the Shoshina-Vassiliev syndrome must continue to take DOPA which led to the prescription of replacement therapy with a preparation containing L-DOPA. Fig.3 shows the pharmokinetics of the administration of 90mg and 95mg of Nakom at 9.00h. It is this dose which enables patients with the Shoshina-Vassiliev syndrome to increase DA and DOPA to physiological levels. A high dose of the preparation in healthy subjects leads to hypersynthesis of DA and NA but not of A, which is reflected in urine excretion. DA hypersnthesis is dangerous for the patients due to the "on-off" effect accompanied by secondary spasticity, difficult to treat with biocorrection.

More than 20 years have passed and tests have shown that the patients are clinically sound. They have completed university, they work and do sports. Patient K actually gave birth to a son in 1993. No complications were reported during pregnancy and at birth. The child is healthy. As an interesting footnote it is worth mentioning that in 75% of cases in which one or both parents took Nakom, a male child was born, not only in cases of Shoshina-Vassiliev syndrome but also in cases of other paralyses with dopamine aetiology. The positive condition of these patients has been brought about only by the continuous, daily administration of Nakom as prescribed at the beginning of treatment. An insufficient or excessive dose is immediately reflected negatively on the patient's condition.

So far, a total of 30 patients, aged between 3 and 45 years, with the Shoshina-Vassiliev syndrome have been identified and have immediately undergone biocorrection using the method developed by the authors, with a 100% success rate (one 5-year old with tetraplegia actually began to walk within just 8 hours of taking the preparation).

The clinical picture of the Shoshina-Vassiliev syndrome has widened so that often, in practice, the original diagnosis is more than just ICP and may include leukodystrophy, leukoencephalitis, Strumpell's disease and so on, with or without disorders of speech, intellect, etc. Only the 0.1g L-DOPA test, coupled with the use of adrenograms, allows us to arrive at an accurate diagnosis, to calculate a personalised dose and to obtain a rapid clinical effect.

Literature would seem to point to an enzimopathy in the case of the Shoshina-Vassiliev syndrome, at the level of tyrosine-hydroxylase or DOPA-decarboxylase; especially since our own preliminary analyses did not highlight any disorder in amino oxydase activity or in homovanillic or vanillylmandelic acid excretion[11,12].

The number of patients affected by the Shoshina-Vassiliev syndrome constitutes no less than 1-2% of ICP cases. The discovery of the Shoshina-Vassiliev syndrome and its mechanisms has enabled the author to develop and patent the technique of biocorrection, with strictly personalised doses of a preparation containing L-DOPA and some doses differing from those used during biocorrection of the Shoshina-Vassiliev syndrome in a large number of paralyses with a dopamine aetiology and with a success rate of between 50-75% depending on the stage and type of disease. Although some traces of the disease naturally remain, in most cases they do not prevent the patients from leading a normal life (study, work, family, children and so on).

The most successfully treated diseases with biocorrection include cerebral palsy, encephalopathy, demyelinating disease, myopathy, post-traumatic and post-infective paralysis and so on, all united by a common denominator: disorder of DA synthesis, specific for each. It may be safely assumed that the number of these diseases will rise[9,11,19].

In 1985, among the demyelinating diseases the new Vassiliev syndrome was discovered which, like the Shoshina-Vassiliev syndrome, may be cured 100% with biocorrection, though over a longer period of time and with eventual suspension of the L-DOPA preparation. The Vassiliev syndrome

differs from the Shoshina-Vassiliev syndrome and other diseases with a dopamine aetiology in the nature of the disorder in DA turnover[5-11,19]. Thus the authors were able to discover three things: a new syndrome, its mechanism and a 100% effective cure.

The Shoshina-Vassiliev syndrome represents an ideal model in the study of a wide range of paralyses; it sheds new light on the role of DA and its function in the aetiology and pathogenesis of diseases previously thought to be incurable and thus enables a cure to be found[9,11,20]. This syndrome deserves to be studied thoroughly and on a worldwide scale by clinicians, genetic researchers and specialists in order to identify any new mechanisms which are valuable at both a theoretical and practical level and to tackle the problem of incurable disease with a new approach.

Conclusions

1) The Shoshina-Vassiliev syndrome, discovered in 1976, is most often found in patients suffering from cerebral palsy, is characterised by a specific abnormality of DA synthesis, may be 100% cured with minidoses of a preparation containing L-DOPA coupled with the Vassiliev biocorrection method which ensures complete clinical recovery. The number of patients, aged between 3 and 45 years, totals more than 30 and constitutes no less than 1-2% of cases of cerebral palsy. We have discovered three things: a new syndrome, its mechanism and complete cure.

2) Any suspension of biocorrection in patients with the Shoshina-Vassiliev syndrome leads to a complete return of the disease (tetraplegia, anarthria, strabismus, etc.). When treatment is resumed, clinical stability is restored within 1-3 hours.

3) The dose required does not depend on the weight or age of the patient nor on the length of time the preparation is administered.

4) Strictly personalised doses are calculated on the basis of adrenograms. A reduction of the dose delays its effects by a few hours while an increase causes secondary spasticity.

5) The preparation is completely physiological, even after 20 years, and patients may have clinically healthy children and can themselves lead a normal, healthy life.

6) The Shoshina-Vassiliev syndrome may only be diagnosed with the aid of adrenograms which allow screening of patients to be made with a high number of diagnoses and their identification.

7) The Shoshina-Vassiliev syndrome represents an ideal model for the study of a large number of paralyses with dopamine aetiology and identification of other functions of DA and needs thorough study on the part of various specialists.

Abstract

The new Shoshina-Vassiliev syndrome, described in 1976 and approved officially and registered in 1985, is most frequently found in cerebral palsy and is characterised by a specific abnormality in dopamine turnover. The discovery of this mechanism has allowed us to develop an effective 100% cure with minidoses of a preparation containing L-DOPA, Nakom type (Sinemet). Any suspension of treatment, regardless of its length, leads to a return of the disease within 1-2 days (tetraplegia, anarthria, strabismus, etc.). When treatment is taken up again the patient recovers within 1-3 hours. This disease accounts for no less than 1-2% of the entire number of patients affected by cerebral palsy. The author has identified and cured more than 30 of these patients, aged between 3 and 45 years.

The Vassiliev adrenogram method has been developed which allows a screening of the Shoshina-Vassiliev syndrome to be made. A biocorrection technique has been elaborated which has led to a 100% success rate, sometimes within just a few hours of administration of minidoses of the preparation (in particular, Nakom and Sinemet). Thus a threesome has been reached: discovery of a new syndrome, knowledge of its workings, 100% effectiveness of treatment.

The discovery of this new syndrome has allowed the author to elaborate a technique of biocorrection of the various types of paralyses resulting from dopamine aetiology: cerebral palsy, encephalopathy, demyelinating disease, including the Vassiliev syndrome and lateral amyotrophic sclerosis, myopathy, post-traumatic paralysis and others. Over 800 patients have been successfully treated.

Key words: Shoshina-Vassiliev syndrome - 0.1g and 0.5g L-DOPA Vassiliev test - Vassiliev adrenograms - dopamine turnover - Vassiliev biocorrection - minidoses of preparation containing L-DOPA - new triad: syndrome, mechanism, cure.

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