MOTOR RECUPERATION OF A PATIENT WITH BASAL GANGLIA ISCHEMIA- CASE REPORT

DOCU-AXELERAD DAN[1], DOCU-AXELERAD ANY[2], DAMIAN MIRELA[1]

Abstract

Purpose. This case presentation was meant to underline the importance of clinic diagnosis and correlation of symptomatology with imagistic findings, without minimizing the role of imagistic examination, so necessary for an accurate diagnosis.

Methods. This case study relates the relationship between imagists and neurologists and recuperation team can have difficulties in establishing an accurate diagnosis. The same importance has the early initiation of a recuperation programme and shows once again the benefits of team work.

Results. The particularity of the presented case released in the pseudotumoral imagistic aspect, even though the symptoms, the neurological signs and evolution of symptomatology were more suggestive for an ischemic stroke. We underline the importance of complete imagistic examination, in our case the absence of the contrast substance made it impossible to perform a contrast MRI, examination necessary for a more accurate diagnosis and a correct design of passive and active recuperation which has a good outcome.

Conclusions. This case presentation was meant to underline the importance of clinic diagnosis and correlation of symptomatology with imagistic findings, without minimizing the role of imagistic examination, so necessary for an accurate diagnosis. Another important aspect is that sometimes even experienced imagists and neurologists can have difficulties in establishing an accurate diagnosis, and this once again shows the benefits of team work.

Key words: basal ganglia ischemia, imagistic examination, kinetic programme.

Introduction

The basal ganglia are the masses of gray matter deep within the cerebral hemispheres. The basal ganglia include the caudate nucleus, amygdala, claustrum, internal capsule, external capsule, extreme capsule, and lentiform nucleus (*A. Osborn, K. Tong, 1996)*.

The lentiform nucleus comprises the globus pallidus and putamen. The caudate nucleus, globus pallidus, and putamen are collectively referred to as the corpora striatum (N. *Beauchamp, et al., 1999)*

The basal ganglia derive their blood supply from small arteries (medial lenticulostriate arteries, which originate from the A1 segment of the anterior cerebral artery and lateral lenticulostriate arteries which originate from the superior portion of the M1 segment of the MCA) and that is why they are susceptible to ischemic injury. (*N. Tomura et al.,* 1988).

Early CT signs of MCA infarction include hyperattenuation of the MCA, an obscured lentiform nucleus, an obscured sylvian fissure, loss of the gray-white matter junction, and loss of the delineation of the basal ganglia. (*R. Von Kummer, H. Bourquain, S. Bastianello, et al., 2001)*

To underline the importance of clinic diagnosis and correlation of symptomatology with imagistic findings, without minimizing the role of imagistic examination, so necessary for an accurate diagnosis. (*W. Harris, M. Castillo, 2000; A. Osborn, 1980)*

Another important aspect is that sometimes even experienced imagists and neurologists can have difficulties in establishing an accurate diagnosis, and

Another important aspect is that sometimes even experienced imagists and neurologists can have difficulties in establishing an accurate diagnosis, and that shows once again the benefits of team work. (*R. Von Kummer, U. Meyding-Lamede, M. Forsting, et al., 1994)*

Von Kummer, U. Meyding-Lamede, M. Forsting, et al., 1994)

[1]University Ovidius Constanta, Physical Education Faculty, ROMANIA
[2]University Ovidiu" Constanta, Medical School Faculty, ROMANIA
e-mail: docuaxi@yahoo.com
Received 13.03.2011 / Accepted 15.04.2011

Method

It is a case study realized in a hospital in Constanta, department of neurology in cooperation with the Faculty of Physical Education.

Results

This case study refers to a male patient, aged 56, hypertensive, obese, without any treatment undergoing, with a recent carotic TIA, who was admitted in our clinic accusing weakness of left limbs, visual troubles and sfyncterial troubles.

The neurological exam at admission revealed left hemiplegia, altered superficial, profound and cortical sensibility, left equilateral hemianopsia, incontinence.

After 24 hours from the onset we performed a brain CT scan (fig 1, 2) that did not show the presence of ischemia or hemorrhage. After two weeks of treatment the patient is discharged with partial relieved symptomatology and with recommendations for diet, kynetotherapy and clinical and therapeutical reexamination after 3 weeks.

After two weeks the patient returns, complaining of severe headache, nausea and repetead bilious vomiting. The neurological examination shows left hemiplegia, equilateral reflex Babinski"s,e xagereted left reflex tendon, impaired sensibility, urinary incontinence.

It is decided to perform a brain MRI (fig. 3) with and without contrast (unfortunely our clinic did not have the contrast substance at that time). The imaging investigation proves the presence of a expanded-infiltrated non-specific lesion, localized in the right frontal and parietal lobe and small demyelinating lesion in the left parietal lobe.

The evolution of symptoms and neurological signs correlated with imagistic results rise the suspicion of a right paraventricular tumor, and after consulting the neurosurgeon it is decided that it will be opportune to perform surgery. Unfortunely surgery is delayed for two weeks because the patient developed acute renal insufficiency. The biopsy is performed with good postoperative evolution , without accidents or new neurological impairement. During surgery it is observed the macroscopical aspect of mass tumor that is suggestive for pseudotumoral ischemia of the right basal ganglia.

The hystopathologycal result established the final diagnosis of cerebral ramollissement (cerebral tissue with microlesions of ischemic ramollissement; absence of tumoral aspects of examed fragments).

After surgery the patient underwent an individualized program of rehabilitation using physical exercises adapted to his clinical tolerance.

We initiate **passive and active kinetics therapy program:**

I. The initial phase, immediately after the surgery:

- in the early stages of the disease, the total or partial paralysis of the limbs is lax;

- the objective of the rehabilitation is the maintenance of the articular mobility in complete amplitudes and the prevention of muscular contractions;

- the correct posture of the limbs in functioning position;

- the upper limb is to be kept with the shoulder in abduction (a pillow in the axilla), the forearm in slight flexion on the arm or in extension, semisupination, the fist in slight extension, the finger in semi-flexion and the thumb in abduction;

- the lower limb is to be kept in extension, not allowing for any degree of flexion or rotation of the hip;

- the knee is to be kept in extension, and the lower foot at a right angle with the lower leg with the aid of a posterior splint, well cushioned in order to avoid compression or skin injuries.

II. The functional recovery of the hemiplegic upper limb

The rehabilitation of the hemiplegic fist and arm is the most difficult problem for the rehabilitator. The rehabilitation of the upper limb started early, in our case – on the 7th day since the debut of the neuro-muscular deficit. It is the period of flaccidity when, first of all, we must proceed to correctly posture the entire upper limb:

- the arm in 45˚ abduction, the elbow in slight flexion or extension, the fist in extension, the fingers in slight flexion, the thumb in abduction (it is repeated every 5' with 30" breaks after each minute of maintenance);

- gradual tactile and proprioceptive stimulation, from facilitating positions; (sensory stimulation is used in the direction of increasing the desired responses and inhibiting the unwanted ones);

-muscle tapotement associated with light pressure, joint light compression, in rapid alternation (5-6' with 30" breaks).

- form the seated position, we use the normal reactions of stability and equilibrium, which we challenge through light pushes of the upper body so as to unbalance the patient who, trying to maintain balance, initiates muscle contractions in the upper limb (5' with 30" breaks after each minute).

Methodic indications: the rehabilitation therapy develops in two phases:

A. In the initial phase, when the proximal extremity of the upper limb is taken care of, there must be voluntary control of the shoulder and elbow, if possible, in different plans; all movements should be as far away as possible from the sin kinetic schemes.

In the beginning, there is recommended to accentuate the plasticity of the hand to any movement of the upper limb root. Therefore, during the active mobilization of the proximal extremity, the hand shall be kept in an inhibition position, that is: total extension of the fingers and of the fist with the thumb in abduction.

- the passive mobilization of all of the joints of the affected limb is done gently, but it must be insisted in order to carry on the full amplitude of the movement. Every joint should be separately mobilized,

holding at the extremities of the mobilized segments (a joint is not passively mobilized through another joint);

- the training of the body symmetry is made through bilateral activities, then alternative unilateral ones, and finally through reciprocal activities;

- when the overall condition allowed it, (on the 30th day since the debut), the Kabat technique is applied, the diagonals for the upper limb; once the spasticity is installed, the new conditions of the neuro-muscular deficit forced the adjustment of the therapeutic tactics;

- it is important to know that with some hemiplegics there may reside a lack of usage of the hand, although motility is recovered. This is explained by the profound sensory disorders due to the involvement of the upward sensory paths which are very close on the pyramidal path, at the level of the cortex and the inner capsule.

The prognosis of the functional rehabilitation of the hand is linked to many aspects, among which we mention some references to the etiology and topography pf the lesion:

- the most serious, from a functional point of view, and, unfortunately, the most frequent, are the cortical or capsular lesions following an ischemia, such as is our case in the ICA territory. If at the debut of the illness, the functional prognosis cannot be determined, two months later it may be known according to: the topography of the lesion, the importance of the sensory and motility disorders.

- the functional prognosis is initially mediocre, and its primarily purpose is that of preventing the elbow-shoulder syndrome and learning how to use the arm as a basic helper, as well as the preservation of the future, in case the rehabilitation should occur (after a year), which is sometimes the case.

The functional rehabilitation was proximally started, then distally.

B. Afterwards, the evolution was the following. During hospitalization, the patient went through:

1. Initially, the hand had no voluntary command or can only flex through stereotype movement.

2. At release, he can actively flex her fingers and thumb, but she cannot extend them except in one position; we explain that it is required to have precision in movement, and not force and execution speed.

The rehabilitator tries the “awakening” of the extensor muscles, with the help of the facilitating techniques, especially those that use the position shifts (Bobath).

III. The functional rehabilitation of the lower hemiplegic limb and walking

The major objective of the lower limb rehabilitation is thus defined: obtaining a balanced command on different antagonist groups and eliminating the sin kinetics in order to recover a walking as close to normal as possible. Most statistics give percentages between 85 and 95 of recovering walking for hemiplegics.

Methodic indications: in order to prevent the typical flexion stiffness and the external rotation of the hip, knee flexion and equinovarus, we install the patient so as to have the basin flat on the bed, with no flexion of the hip and knee, the lower limb totally coupled so as to avoid its fall in external rotation, the feet is maintained at 90˚ on the lower leg.

Spasticity is announced through the exaggeration of the ROT and usually begins with the abductors of the thighs and the quadriceps, in our case, on the 30th day.

For a good rehabilitation of the walk, it is necessary to make a thorough analysis of the muscular deficit, of the repartition and intensity of the spasticity, of the intensity of the sin-kinetics, to sum up, it is necessary to make a functional evaluation of the patient.

The muscular deficit is mostly recorded (the general scheme of hemiplegia) on the following muscles: psoas, abductors and internal rotators of the hip, the knee flexors, and the leg dorso-flexors. The ischio-tibial muscles are partially respected.

During the evolution, the deficit is modified; the first muscles to recover voluntary contraction capacity are the abductors, the quadriceps and then gluteus maximus.

Conclusions:

This case presentation was meant to underline the importance of clinic diagnosis and correlation of symptomatology with imagistic findings, without minimizing the role of imagistic examination, so necessary for an accurate diagnosis. Another important aspect is that sometimes even experienced imagists and neurologists can have difficulties in esthablishing an accurate diagnosis, and that shows once again the benefits of team work.

The muscles that remain most often, deficient, are the common extensor of the fingers, the peroneals, and the middle and small gluteus.

Bearing this in mind, the importance of the correct positioning during the flaccidity period is thoroughly justified.

Fig 1: FirstCT SCAN

Fig 2: CerebralCT scan

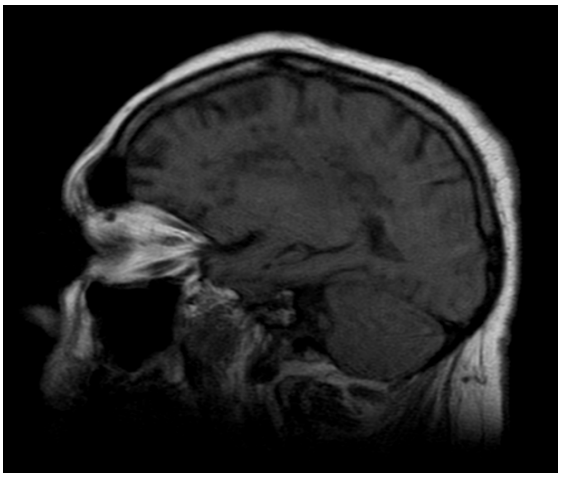

Fig. 3

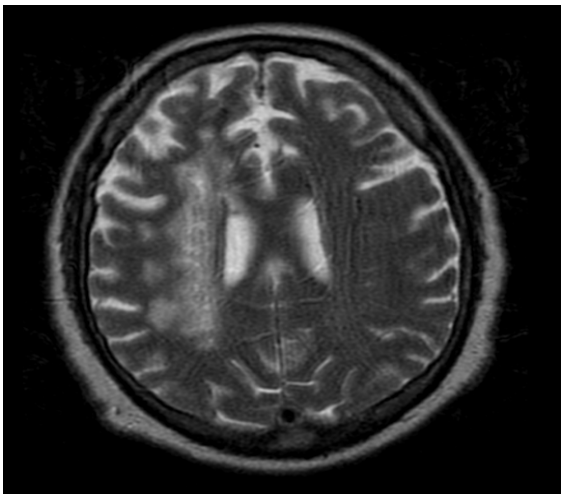

Fig. 4 CEREBRAL RMN

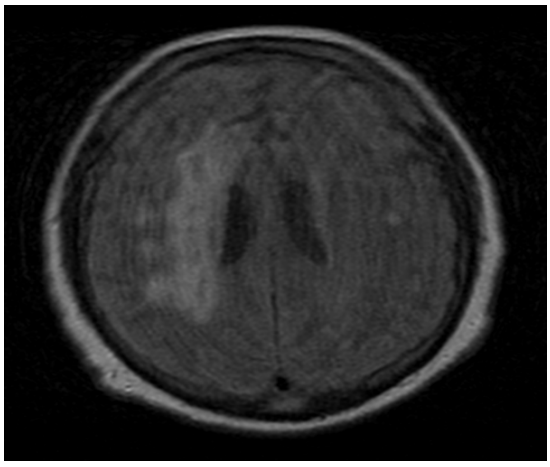

Fig. 5

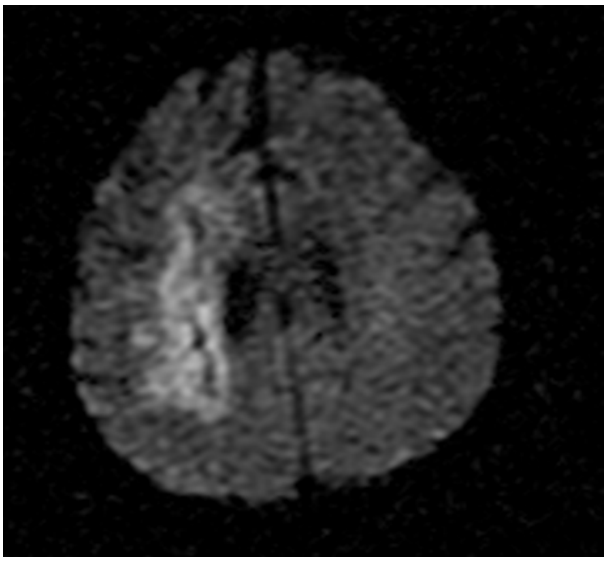

Fig.6

References

BEAUCHAMP, N., BARKER, P., WANG, P., VAN ZIJL, C.M., *1999,* *Imaging of acute cerebral ischemia*. Radiology*;* 212:307-*324*

HARRIS, W., CASTILLO, M., 2000, *The radiology of emergency medicine Baltimore*, Md: Lippincott Williams & Wilkins; *30*-31.

OSBORN, A., TONG, K., 1996, *Handbook of neuroradiology: brain and skull St Louis, Mo:* Mosby, *52*-53, 568–569.

OSBORN, A., 1980, *Introduction to cerebral angiography Philadelphia*, Pa: Harper & Row; *239*-251.

TOMURA, N., UEMURA, K., INUGAMI, A., FUJITA, H., HIGANO, S., SHISHIDO, F., *EARLY, C.T.*, 1988, F*indings in cerebral infarction: obscuration of the lentiform nucleus.* Radiology*;* 168:463-*467.*

VON KUMMER, R., MEYDING-LAMEDE, U., FORSTING, M., et al., 1994, *Sensitivity and prognostic value of early CT in occlusion of the middle cerebral artery trunk.* AJNR Am J Neuroradiol*;* 15:9-*15.*

VON KUMMER, R., BOURQUAIN, H., BASTIANELLO, *S., et al., 2001,* *Early prediction of irreversible brain damage after ischemic stroke at CT.* Radiology 2001*;* 219:95-*100.*