

#### Abstract

**Introduction:** Anoxic encephalopathy is one of the hardest rehabilitation condition which can generate cognitive dysfunction and tetraparesis. The multimodal/ pleiotropic therapeutical approach by influencing endogenous defense activity fundamental biological processes of neurogenesis, neuroprotection, neuroplasticity and neurotrophicity and counteracting neurogenic inflammation and the secondary phenomenoms according "tooth paste theory" combined with a specific rehabilitation program could be a benefit therapeutic association.

**Matherial and methods:** A 42 years old patients without any prevous medical conditions was admitted in our Neural-muscular clinic division with cognitive impairment and spastic tetraparesis after anoxic encephalopathy. The case reposrt presentation was approved by THEBA Bioethics Commission (No.9181/11.04.2018). The patient was clinically, paraclinically and functionally assessed according to the standardized protocols implemented in our clinic through the assessment scales (AIS, FIM, QoL-Quality of Life, Asworth, Penn, FAC, WISCI II).

**Results:** The combined and complex rehabilitation program lead in this specific case to a complete cognition remission and substancial locomotor regaining: at discharge the patients having independence of walking

**Conclusions:** The modern ambivalent approach of spastic tetraparesis and cognitive impairment emerging from anoxic encephalopathy could be a succeful therapeutical management leading in some cases to a complete recovery.

Key words: Anoxic encephalopathy, spastic tetraparesis, rehabilitation program

#### Introduction

Traumatic brain injury (TBI) consist in two main categories of lesions: primary injuries, that occur at the moment of trauma - most difficult, from medical point of view, to be limited and/ or treated and secondary injuries, which occur after the initial trauma (brain tissue swelling hemorrhage, loss of the local/ regional blood flow –generating ischemia) as a consequence of a specific Central Nervous System (CNS) patho-physiological events "cascade" and produce effects/damages that may continue for a long time - worsening the evolution and the prognosis.<sup>[1]</sup>

Neuroprotection is a neurobiological type process being part of endogenous defense activity (EDA) of the nervous system: a polychronic continuous process that simultaneously performs activities of neurotrophicity, neuroprotection, neuroplasticity and neurogenesis <sup>[2,6]</sup>.

Neurotrophicity is the natural biological process by which the continuous effort of the cell maintains correct DNA expression, thus maintaining a normal phenotype. Neuroprotection is the sum of all mechanisms directed against harmful factors. Neuroplasticity is the brain ability to change/ adapt already existing structures to response to the environmental stimuli (such learning, new experience, injury, etc). Neurogenesis is the process by which new nervous tissue cells are created from stem cells (astrocytes, oligodendrocytes)<sup>[4]</sup>.

The mechanisms of EDA are divided into two categories: (1) the primary mechanisms which are controlled by *neurotrophic factors and neurotrophic like molecules and (2) the second* relative mechanisms which involve some other processes including channel blockers like antagonists, receptors antagonists, chelator of certain metals, etc.<sup>[3,4]</sup>

The aim of neuroprotection is to prevent the death of neurons, glial cells and endothelial cells (neurovascular unit). The primary goal of neuroprotection in TBI is to prevent and reduce secondary /injuries lesions<sup>[5]</sup>.

Anoxic encephalopathy is one of the hardest rehabilitation condition which can generate cognitive dysfunction and tetraparesis. The multimodal/ pleiotropic therapeutical approach by influencing endogenous defense activity fundamental biological processes of neurogenesis, neuroprotection, neuroplasticity and neurotrophicity and counteracting inflammation and neurogenic the secondary phenomenoms according "tooth paste theory" combined with a specific rehabilitation program could be a benefit therapeutic association.<sup>1-2</sup>

## Matherial and methods:

This paper presents the case of a patient having The Teaching Emergency Hospital "Bagdasar-Arseni", TEHBA, Bioethics Committee approval no 9181/11.04.2018.

A 42 years old male without any previous medical conditions was admitted in our Neuro-muscular clinic division with cognitive impairment and spastic tetraparesis after anoxic encephalopathy. psycho-cognitive status.

Family history was insignificant. From personal medical history we underline the patient was intoxicated with carbon monoxide, and suffered anoxic encephalopathy on December 15, 2017.

Clinical examination at admission revealed: patient conscious, relative cooperant, temporo-spatial dezorientated, left central facial paresis, third degree left calcaneal pressure sore. Cardiovascular system had normal heart sound, no added sounds or murmur, blood pressure 115/70 mmHg, heart rate 108. Respiratory system: bilateral equal air entry, normal vesicular breathing, peripheral oxygen saturation (SpO<sub>2</sub>) 98%. urogenital apparatus: urinary catheter at admission.

Local clinical examination at admission revealed: left central facial paresis, increased upper left limb muscular tonus at first admission - 2 Ashworth (left elbow flexion reducible) and 1 Ashworth at upper right limb. As for lower limb, muscular tonus was increased at 4 on Ashworth at left lower limb (with knee flexion irreducible on the last 30 degrees of extension) and 2 on Ashworth at right lower limb. Patient had lower and upper limbs motor control present at all levels, Cutaneous plantar reflex: indifferentt at left side, and flexion on right side

Functional: at first admission the patient was bedridden.

As for FIM, cognitive subtotal score was assessed with a result of 7/35 points. Mini mental state examination (MMSE) could not be tested.

The patient was paraclinical examined in order to evaluate his biological reserve and his availability in bearing the recovery program. To this purpose, both laboratory and imaging investigations have been used. The laboratory investigations revealed: severe hypoalbuminemia, marked hyposideremia, moderate *normocytic* and *normochromic anaemia*, severe inflammatory biological syndrome. Urinary tract infection with Pseudomonas spp treated with the help of the drug sensitivity test.

During the hospitalization the patient was evaluated by doctors from several specialties: (1) plastic surgery, for the pressure sore; they recommended local rigorous hygiene and avoiding decubitus on the affected area. (2) infectious diseases, for the urinary tract infection; recommended antibiotic etiology according to the antibiotic sensitivity.

The main diagnostic at discharge was: Psychocognitive and dysfunctional status with spastic tetra paresis due to anoxic encephalopathy. Vertiginous syndrome. Urinary tract infection with Pseudomonas spp, treated. Third degree left calcaneal pressure sore, cured.

For the rehabilitation program we had the main general objectives: (1) Bring the patient in state that allows self-care and movement. (2) Improving communication disorders. (3) Improving psychocognitive / mental and emotional status. (4) Comorbidity therapy. (5) Social (family) and professional reinsertion of the patient.

Rehabilitation program specific first mean is hygienic-dietary regime consisting in suitable hydration (1,5-2l/ day), rich regime that includes vitamins, minerals, and should avoid fermenting foods (beans, peas) but with a proper fibres intake for maintaining a healthy gut transit.

Should avoid skin contact with liquids/objects that are too cold or hot. Avoid cold, moisture, cold air currents, sudden movements, respectively trauma at any level of the spinal cord.

During admission the patient was assessed and treated with the following drugs: anticoagulant, pain killer, gastric protector hydro electrolytic rebalancing antispasmodic treatment, antiarrhythmic treatment, urinary disinfectant, antibiotic, iron supplement, neuroprotective drug.

Physiotherapy administrated were: LASER for the improvement of the knee pain and Shock Wave for reduce knee flexion.

Kinetic objectives followed were:

- 1. Combating spasticity
- 2. Restore / maintain joint limb mobility
- 3. Re-engineer motor control at all levels
- 4. Re-education of verticalization / orthostatic position
- 5. Re-training transitions and practicing walking on a physiological basis
- 6. Engaging control coordination and balance on walking

Several kinetic means were encountered in the rehabilitation program.

To combat spasticity it is recommended dorsal decubitus position with the lower limbs stretched at 90 degrees on the bed sheet, prolonged stretching. To restore and maintain joint mobility: (1) Passive mobilizations that are gently executed at the maximum stretching point/ prolonged stretching (2) Passive-active and active-passive mobilizations (3) Kinetic bed side program: passive mobilizations (4) Kinetic program at the gym: pulley, bicycle (4) Main role: to maintain articular function and to prevent heterotypic ossification with secondary ankylosis. To increase muscle force were performed active mobilizations with resistance: pulley and Helcometru. For the lower limbs training: exercises at the ergo metric bicycle and Motomed. Transfer training followed was: changing and maintaining position from dorsal decubitus to shortened seat and transfer from dorsal decubitus to the wheel chair and from the wheel chair to ortostatic position. To maintain verticalization trellis exercises with orthostatic maintenance were done. For practicing walking progressively, initially walking at parallel bars was performed and subsequent walking with supporting frame, respectively walking for short distances with one crutch in the left hand. Occupational therapy targets the re-expression of locomotion function and independence through the upper limbs training.

Training the diaphragmatic breathing – in order to relax and bring a proper oxygen intake that is vital for the rehabilitation program and furthermore for successful results.

Patient's evolution was favourable: functionally improved, the patient performs walking on the flat terrain for average distances under supervision. At discharge patient had bladder and bowel control recovery. Psycho-cognitive status improved following neuroprotective treatment (MMSE score 28/30 and FIM score 30/35).

Prognosis ad vitam and ad functionem, were favourable. At laborem prognosis doesn't matter because the patient did not have an occupation.

As complications we mentioned urinary tract infection with Pseudomonas spp, treated and third degree left calcaneal pressure sore, cured.

The combined and complex rehabilitation program lead in this specific case to a complete cognition remission and substantial locomotor regaining: at discharge the patients having independence of walking.

## Conclusions

The modern ambivalent approach of spastic tetra paresis and cognitive impairment emerging from anoxic encephalopathy could be a successful therapeutically management leading in some cases to a complete recovery.

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