Clinical-therapeutic and recuperators complex aspects in a chronic ethanol-consuming patient with incomplete AIS / Frankel D tetraplegia after cervical SCI on the background of ankylosing spondylitis, with C6 transosus fracture operated (by mixed osteosynthesis) and immobilized in the halo-vest by falling from its own level and the posterior cerebral artery ischemic stroke (possibly by vertebral artery dissection)

Stoica Simona-Isabelle1,2, Carmen Chişmăuş1, Magdalena Lapadat1, Dumitraşcu Andreiţa1, Nohai Iulia-Maria1, Gelu Onose1,2

Abstract
Spinal cord injuries and strokes are frequent causes of motor deficit in patients of all ages, with complex family and social consequences (through sensitivity and movement disorders). On the other hand, toxic-nutritional abuses (especially ethanolic) cause morpho-physiological changes throughout the body, with frequent consecutive neuro-psychic manifestations, followed by (potentially) various traumatic injuries. Therefore, the biological and scientific clinical follow-up of traumatized vertebro-medullary patients is of particular importance. With the approval of the Bioethics Commission of the Bagdasar Arseni Emergency Clinical Hospital (TEHBA) Bucharest (number 9181 dated April 11, 2018), we will present the special case of a patient admitted to the Neuromuscular Recovery Clinic of THEBA for incomplete tetraplegia motor deficit AIS/Frankel D, with neurologic level C6 after a vertebral-medullary trauma (produced in conditions of ethanol abuse) and with parieto-occipital ischemic vascular accident produced simultaneously. The peculiarities of this case are the possible (but less common) immediate consequences of vertebral-medullary traumas: paravertebral ganglion lesions; arterial (carotid / vertebral) dissections, which can cause ischemic lesions, all requiring appropriate clinical and therapeutic management. Spinal cord injuries can be favored by toxic-nutritional abuse and may have immediate, late, and permanent morpho-physiological consequences. However, sometimes the clinical evolution and prognosis are surprisingly positive.

Key words: Traumatic brain injuries, spinal cord injury Polytraumatism

Introduction
Spinal cord injury (SCI) is the cause of a plegic deficiency, with temporary or permanent loss of nervous control over a somatic and vegetative territory. Each year in Europe, about 11,000 patients - and in the US - about 12,000 patients suffer posttraumatic paralysis. The majority of SCI sufferers is represented by young men (75% in Europe and 77.8% in the US) of about 33.4 years (in Europe) and 39.5 years (in the US). In rural areas, elderly people are most affected by SCI through accidental falls. The causes of SCI are: road accidents (55%), work accidents, sports accidents (8%), drops from height (22%), aggression (4%). The most affected in the SCI are the vertebrae: C5, C6, C7 and the T12-L1 junction, with variable myelosuppression, clinically manifested by tetraplegia / paraplegia (1)

For severity assessment of the SCI we use American Spinal Injuy Impairement Scale (AIS) with the following grade: A (if there is no sensitivity or motor control in the S4-S5 territory or inferior to lesion level); B (if the sensitivity, including at S4-S5 level, is below the affected area, without the motricity); C (if the voluntary movement is present lower than the lesion level, but at least half of the key muscles do not have antigravity action, they have a force of <3 on the MRC scale); D (if the movement is below the lesion level, and at least half of the key muscles act antigravitatively, they have a force> 3 on the MRC scale); E (if motricity and sensitivity is normal to a patient who has suffered a SCI).(2)

For the SCI, treatment in the acute phase is represented by: first aid on the spot (hemodynamic and respiratory balancing, immobilization of the spine), target imaging investigations, neurosurgical intervention (centromedular drainage and spine stabilization with metal osteosynthesis and / or bone grafting).

The stroke is: „the rapid development of localized or global clinical signs of cerebral dysfunction with symptoms exceeding 24 hours, leading to death, without any other cause, except for vascular origin.” Stroke are 80-85% ischemic (thromboembolic: atrial fibrillation, acute myocardial infarction, valvulopathy, congenital heart disease, atherosclerosis, hypercoagulability, arteriopathy) and 10-15% haemorrhage (after traumatic brain injury, broken arterio-venous malformations, hypertensive encephalopathy, coagulopathies).(3)

Vertebral artery dissection may becaused by: SCI, respiratoy infection, arteriopathies and the symptomatology may consist of: local pain, headache, Wallenberg’s syndrome. The diagnosis can be made with: Doppler ultrasound, angiography (CT,
IRM). The treatment is made with: anticoagulants and antiplatelets. The prognostic is good (in most cases).

(4) Ankylosing spondylitis "it is a chronic inflammatory disease, usually evolutionary, that affects both the axial skeleton and the peripheral joints." The diagnostic criteria are: inflammatory lumbosacral pain and stiffness > 3 months, limiting the lumbar spine motion in the sagittal and frontal plane, limiting chest expansion, radiological: bilateral / unilateral sacroilitis. There are also rapidly evolving forms, affecting the whole spine in 1-2 years. Possible comorbidities are: Crohn's disease, ulcerative colitis, anterior uveitis, cardiovascular disorder (aortic regurgitation, atrio-ventricular blocks), pleuropulmonary damage (pulmonary fibrosis, sleep apnea syndrome), osteoporosis, depression.(5,6)

CASE PRESENTATION
We present the case of a 62-year-old patient, hypertensive, smoker, chronically ethanol-consuming, with clinical and paraclinic ankylosing spondylitis appearance, operated by intestinal occlusion (in 2014). He has been hospitalized from 21.02.2018 to 06.06.2018 in Spinal Neurosurgery and Neuromotorious Recovery Clinics of TEHBA for: spastic incomplete quadriplegia, neurogenic bladder, sensitivity disorders, neuromuscular recovery.

The patient was the victim of a politraumatism by falling from his own level (on February 14, 2018), resulting in: minor traumatic brain injury, thoracic-abdominal contusion (4, 5, 6 right ribs fractures, lower left lobe pulmonary atelectasis, bilateral minimal pleurisy), cervical SCI – transverse fracture C6, C6 / C7 anterior dislocation operated (by mixed osteosynthesis, with 2 screws, metal plate and intersomatic graft on 28.02.2018), ischemic stroke in vertebro-basilar territory. Postoperatively, he was immobilized in the halo-vest system.

General clinical examination showed: dehydrated teguments and mucous membranes with numerous abrasions on the limbs, bilateral Dupuytren, hands and feet arthritic nodules, thorax deformation, SaO2= 95%, normal chest area, rhythmic cardiac noises, mitral systolic murmur (grade III / VI), BP = 90/60 mmHg, HR = 102 / min, slow intestinal transit, lower border of the liver at 1 cm under the costal rib, unpalpable kidney, neurogenic bladder - permanent urinary catheter.

At the NMAK examination we discovered: a consciously, cooperative, temporo-spatial orientation patient, with a spastic incomplete quadriplegia with neurological level C6: bilateral hiperreflexia, bilateral Babinsky, bilaterally plantar clonus (exhaustible); anisocoria, photopupillary reflex bilaterally, normal visual field, moderate motor control on the upper and lower limbs.


The paraclinical investigations performed were:

Thoracic CT scan with contrast (21.02.2018)

Chest radiography (21.02.2018)

The thoracic surgery examination (22.02.2018) said: Trauma with IV, V, VI rib fractures, minimal bilateral pleuresia. The CT examination reveals left inferior pulmonary lobar atelectasis probably in the context of thoracic spondylitis and hyperventilation because of the pain caused by the fractures, but also of the post-traumatic neurological deficit.
Head CT (25.04.2018)

Carotid and vertebral vessels Doppler ultrasound (03.05.2018): the significant decrease in arterial distention, intimate thickening up to 1-1.2 mm, ACC bilaterally with numerous hyperecogenic areas of 0.5 mm across the cervical tract, especially in the posterior wall, hyperecogenic plaque 8.5 mm thick anterior right carotid bulb, homogeneous hyperecogenic plaque 3.2 mm at 10 o'clock left carotid bulb.

Cervical IRM (25.02.2018)

Cervical Radiography (01.03.2018)

Pelvic Radiography (15.05.2018)

The established diagnosis was: incomplete spastic Tetraplegia AIS / Frankel D with C7 neurological level post cervical SCI with C6 fracture and anterior C6 / C7 dislocation on background of ankylosing spondylitis by falling from his own level (14.02.2018); parieto-occipital ischemic stroke (probably produced by SCI); chronic obstructive bronchopneumopathy; essential hypertension; neurogenic bladder; post thoracic traumatism status with IV, V, VI right ribs fracture and lower atelectasia left pulmonarylobe; alcoholism.

Clinical-functional evolution of the patient was favorable. The patient received medical treatment (with: injectable anticoagulants, antibiotics, analgesics, urinary antiseptics, neurotrophic, myorelaxants, hydroelectrolytic rebalancing, gastric protection), has practiced ergotherapy and a suitable kinetotherapeutic program (passive and active exercises at the bed level, then at the gym). On March 26, 2018, the patient experienced a scalp and face...
inflammation (by dislodging one of the halo vest pins), being readmitted in the neurosurgical section. After the readmission in Recovery Clinic, the patient was mobilized in the wheelchair and began to practice walking.

The possible complications are: repeat stroke, secondary epilepsy, urinary lithiasis, chronic respiratory failure.

DISCUSSIONS AND CONCLUSIONS

I have presented the complex case of a patient with multiple comorbidities, who presented an SCI in unclear conditions. Although SCI severity was high, neurological deficits were not complete. And, despite the infectious complications of neurosurgical treatment, the patient progressed in the recovery process. This is a contradictory case that raises many etiological and evolutive questions.

Bibliography

1. Prof Dr Gelu Onose, Șef de Lucrări Dr Aurelian Anghelescu -Ghid de diagnostic, tratament și reabilitare în suferințe după traumatisme vertebro-medulare- Ed Universitară Carol Davila, București
4. Thanvi B et al “Carotid and vertebral artery dissection syndromes” Postgraduate Medical Journal, vol81, 2005 http://pmj.bmj.com/content/81/956/383
5. Gherasim L and al “Medicina Interna” edII, vol1- Ed Medicala, Bucuresti 2003