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# Paraplegia due to Anterior Spinal Artery Stroke: Rehabilitative Program on Lower Extremity Weakness and Locomotor Function

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#### Abstract

**Background:** Spinal cord ischemia in the absence of trauma with a stroke in the territories of anterior spinal artery (ASA) attributed to paradoxical embolism through patent foramen ovale (PFO) is a rare event. Rehabilitative treatment is focused on the improvement of lower limbs muscle torque to consent a recovery of balance and gait.

Methods: Case report of a 43-year-old woman with paraplegia after ASA stroke due to PFO

**Results:** A 43-year-old woman was hospitalized for sudden and progressive increase of weakness of lower limbs few hours after evacuation effort by using the Valsalva maneuver. Neurological and physiatric examination revealed paraplegia, lower limbs dysesthesia, and hesitancy with delayed bowel emptying. Spine-MRI showed D12–L1 anterolateral cord ischemia. Transcranial doppler sonography examination disclosed a PFO with moderate right-left shunt. During hospitalization, the patient was treated with steroids and acetylsalicylic acid drugs. Then, she was transferred to the Department of Physical Medicine and Rehabilitation and submitted to intensive rehabilitation of balance and gait. First, the patient was treated to increase the lower limbs torque and trunk control using also functional electrical stimulation (FES) cycling. Then, she performed an aquatic treadmill increasing contemporaneously both the weight support on the paretic legs than gait velocity. Motor and urinary symptoms disappeared in 30 days.

**Conclusion:** After diagnostic work-up, PFO was considered the only cause of disease, suggesting that this was a case of ASA due to probable paradoxical embolism. The patient was treated with pharmacological therapy and a rehabilitative protocol with good recovery of locomotor function and muscle strength.

**Keywords:** Spinal cord ischemia; Patent foramen ovale; Anterior spinal artery; Rehabilitative treatment

### Introduction

Spinal cord ischemia is a rare disease, and the natural history and pathogenesis remain largely unknown, even if in the absence of trauma, sudden development of symptoms referable to lesions of the spinal tract suggests an infarct or hemorrhagic lesion of the spinal cord [1]. Anterior spinal artery (ASA) thrombosis, spinal cord angioma, and aortic lesion are considered the more possible cause of this disease [2]. It is known the possibility that the spinal cord infarct in the anterior spinal artery distribution is attributed to paradoxical embolism through a patent foramen ovale (PFO) [3].

The clinical picture of ASA syndrome varies with the level of ischemia causing muscle weakness, grading from paresis to tetra- or paraplegia with loss of the sensation of pain, temperature, touch and loss of sphincter control. Pharmacological and rehabilitative treatment are necessary to improve the clinical picture consenting a good recovery of walking and activities of daily living.

Physical rehabilitation needs to move beyond the goal of maximizing independence to focus on maintenance of optimum health and fitness as well as maintenance of target system function below the level of injury. The main objective of the rehabilitation treatment is to succeed in initiating an early, customized healthcare, and lifestyle plan based on three major principles: prevent and treat impairments and medical complications from the first few days, provide physical therapy and rehabilitation to promote optimal neurological recovery and functional independence, and provide social and professional rehabilitation. In the present report, we described a case of paraplegia after ASA stroke due to PFO submitted to rehabilitative treatment with good recovery of lower extremity weakness and locomotor function.

#### **Case Report**

A 43-year-old woman with no significant medical history was hospitalized at the Department of Physical Medicine and Rehabilitation, University of Foggia, Italy, for sudden and progressive increase of weakness of lower limbs. She referred onset of sudden low back pain during evacuation effort by using the Valsalva maneuver and forced post-expiratory apnea and a progressive increase of lowerextremity weakness few hours later. The pain was associated with mild paresthesia, and the patient had difficulty walking. She denied any history of diabetes, hypertension, cardiovascular disease, tobacco, drug use, or surgery. The patient was afebrile. Cranial nerves were intact. Examination of the head, neck, and back revealed no evidence of trauma. There was no erythema or warmth, point tenderness,

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paravertebral spasms, or palpable mass. The upper extremities were without deformity, with normal findings on motor, sensory, and reflex examinations. Paresis grade T9C (motor score: 84) according to American Spinal Injury Association (ASIA) impairment scale [4,5] was present in the legs. Although, several studies used the ASIA examination both in case of traumatic or nontraumatic spinal cord injuries, initial neurological assessment following ASIA classification proved to be the best predictor of prognosis in these patients [6]. There was no patellar, Achilles, and plantar reflexes bilaterally and sensation to pinprick was lost throughout the genital area and the legs up to the D10 level. Vibratory sensation, light touch, and proprioception were preserved. She referred to have urinary incontinence and hesitancy with delayed bowel emptying. The patient was submitted to selfcatheterization and subsequently to intermittent catheterization post-voiding for the assessment of post-void residual. Spine-magnetic resonance imaging (MRI) showed D12-L1 antero-lateral cord ischemia, with hyperintense signal abnormalities on T2 weighted MRI scans (Figure 1). Specific clinical features of patients with typical sudden onset of neurological deficits caused by spinal cord ischemia, and axial or sagittal T2 weighted MRI images, showing hypertensities in the ASA territory, are very useful in detecting spinal cord infarction [7,8]. Thyroid hormones, serum folate, homocysteine, and vitamine B12 levels were within normal limits. No coagulation disorders were present. Echocardiographic evaluation showed a floppy image of atrial septum. Transcranial doppler sonography examination confirmed the suspect of the PFO presence with the shunt of pulmonary emboli from right to left atrium both spontaneously than during Valsalva maneuver (Figure 2). The patient was also evaluated using the Medical Research Council (MRC) Scale for lower limbs muscle strength (grades 0-5) [9], Functional Independence Measure (FIM) [10], and Barthel



**Figure 1:** Sagittal image of the spinal cord at magnetic resonance imaging (MRI) of the 43-years-old woman showing D12–L1 antero-lateral cord ischemia (red arrow) with hyperintense signal abnormalities on T2 weighted MRI scans.



**Figure 2:** Transcranial doppler sonography examination showing the patent foramen ovale presence. During subsequent Valsalva maneuver, the presence of a Doppler signal noise is evident with the passage of many micro-embolic signals (MES) (red circles), a sign of severe right-left shunt.

Index (BI) [11]. Table 1 showed the clinical picture of the patient at the admission and after discharge. The patient, after the discharge from the Department of Physical Medicine and Rehabilitation, University of Foggia, Italy, was hospitalized at the Cardiovascular Surgical Unit, IRCCS Policlinico San Donato Milanese, Milan, Italy, where transesophageal echocardiogram was performed and she was undergone to PFO repair with an endoscopic, closed chest approach.

The patient was submitted to intensive rehabilitation program composed by passive joint extension and flexion to increase the lower limbs torque and trunk mobilization for 1 hour, two times/day. Then, she started with a training on functional electrical stimulation (FES) cycling for extensor muscle, 30 minutes/day, 5 days/week for 4 weeks. Passive exercises and FES lasted about one month, and when she was able to stand up independently and to walk with two crutches, she started to perform an aquatic treadmill from 20 minutes/day at the beginning, increasing progressively the time of training to 45 minutes/day, 5 times/week for 4 weeks. An aquatic treadmill system was used using the water level at umbelicus as a body weight support (Figure 3). In the first phase (15'), the patient was submitted to trunk stability and proprioceptive exercises increasing the weight support on the paretic legs. Then she performed gait rehabilitation exercises (30') and the velocity of the treadmill was set to the maximum speed tolerated by the patients beginning approximately from 0.3 km/h. During the following walking sessions, the water level was reduced and consequently the body weight on legs increased. Two months later she was able to walk more quickly and at the end of the training period her Walking Index for Spinal Cord Injury (WISCI) [12] level was 8 respect to 0 at the admission. MRC score improved of about 2 points for leg extensor muscle and anterior tibial muscle and about of 1 point for other muscles involved. Finally, FIM score became 111/126 and BI score ranged from 35 to 85/100. At the discharge the patient became L2D on the ASIA scale (motor score: 92).

## Discussion

In the present case report, we described the effect on lower extremity weakness and locomotor function of a rehabilitative combined treatment in a rare case of paraplegia due to ASA stroke. ASA syndrome is an extremely rare cause of acute ischemic cord infarction. It is caused by occlusion or hypoperfusion of the anterior spinal artery, which supplies the anterior two thirds of the spinal cord.

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	At admission	At discharge
Motor function	Paraplegia	Paraparesis
MRC leg extensor muscles MRC leg flexor muscles MRC adductors muscles MRC ankle dorsiflexors muscles MRC plantar flexor muscles Walking Index for Spinal Cord Injury Sensory function (pinprick)	2/5 3/5 3/5 2/5 3/5 0 Reduced	4/5 4/5 4/5 4/5 4/5 8 Normal
Urinary function	Incontinence	Normal
Voluntary anal contraction	Yes	Yes
Deep anal pressure	Yes	Yes
Bowel function	Delayed emptying	Normal
FIM scale	80/126 Motor function: 45/91 Cognitive function: 35/35	111/126 Motor function: 76/91 Cognitive function: 35/35
ADL score	1/6	6/6
Barthel Index	35/100	85/100
ASIA	Motor score: 84 Pin prick score: 86 Light touch score: 86 Sensory neurological level: T9 C	Motor score: 92 Pin prick score: 106 Light touch score: 106 Sensory neurological level: L2 D

MRC: Medical Research Council; WISCI: Walking Index for Spinal Cord Injury; FIM: Functional Independence Measure (FIM); ADL: Activities of Daily Living; ASIA: American Spinal Injury Association

 Table 1: Clinical picture of the 43-years-old woman with paraplegia due to anterior spinal artery stroke before and after the rehabilitation treatment.



Figure 3: Patient with paraplegia due to anterior spinal artery stroke on aquatic treadmill system for gait rehabilitation.

It can be associated with aortic surgery [13] atherosclerosis and diabetic arteriopathy, [14] vasculitides, aortic disease, anemia, polycythemia, atlanto-occipital dislocation [15], cervical spondylosis [16], cervical spinal trauma [17], recreational drugs such as cocaine [18], and some infections (tuberculosis, schistosomiasis, and Neisseria meningitidis) [19]. Paradoxical embolism through a patent foramen ovale represents a rare but possible cause of spinal stroke [3].

The clinical picture of ASA syndrome varies with the level of ischemia. There is different degree of muscle weakness and dissociated

sensory loss. Usually, the loss of motor power parallels that of pain because of the anatomical proximity of the pyramidal and spinothalamic tracts in the cord.

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In ASA stroke, there is abrupt onset of symptoms, with deficits usually appearing within several minutes to a few hours of the initial insult. Flaccid motor paralysis and absent deep tendon reflexes may later progress to spasticity and hyperactive tendon reflexes. Bladder and bowel paralysis are often noted with retention or incontinence of urine and feces. MRI may be a sensitive tool for evaluating spontaneous spinal cord infarctions [20].

# Treatment options for this relatively rare condition also remain elusive

However, rehabilitative treatment may consent a complete motor recovery in few months improving gait, balance, muscle torque, and reducing disability assessed with FIM, BI, and gait parameters [21,22]. At present, there is a lack of clinical studies on the duration of rehabilitative treatment in these syndromes. Usually, the spinal cord injury rehabilitation is focused on on the improvement of lower limbs muscle torque, postural control, balance, and gait. A recent review on the objectives of rehabilitation after spinal cord injury showed the utility and effectiveness of physical rehabilitation strategies on health and fitness, and maintenance of target systems below the level of injury (i. e., muscle, bone, and circulation) highlighting areas for future research in this field [23]. Exercising the paralyzed limbs using also FES is a potent means of reducing muscle atrophy, preserving muscle function or to promote regeneration of the damaged peripheral nerve [24,25].

In the present case report, the patients with ASA stroke was submitted to intensive daily rehabilitation program composed by passive joint extension and flexion, trunk mobilization, lower limbs functional electrical stimulation and aquatic treadmill with good recovery of locomotor function and muscle strength. In the first phase of rehabilitative treatment is necessary to increase lower limbs torque to consent the recovery of trunk and postural control; FES represents a useful tool to improve muscle torque and knee mobilization [21]. A successive phase of paraplegia treatment is focused on gait rehabilitation with an aquatic treadmill system that consents to walk reducing the body weight with an high water level. It is known that if the water level increases the body weight reduced differently. In fact, it is possible to reduce the body weight approximately about 70% at shoulder level. Moreover, walking movements may be facilitated on the water treadmill by activating spinal and supraspinal sensorimotor cortical centers, [26] contemporaneously, it is possible increase the lower limbs muscle torque by the resistance of water while patient's walking. The protocol used for this patient yielded significant training effects on the muscle torque, and contractile speed properties of the paralyzed muscles. A combination of higher torque-producing capacity and reduced fatigability in the trained limb enabled it to perform a greater magnitude of contractile work during repetitive activation.

There is a need to determine the optimal training regimens to improve fitness levels. Moreover, for future research, considering that the literature has conflicting reports on this matter, is mandatory to conduct large-scale observational studies to provide evidence for the effectiveness of interventions for spinal cord injury, and to identify the kind of exercise interventions, the best way of measuring the amount and intensity of physical activity after spinal cord injury, so distinguishing the confounding influence of pharmaceutical interventions investigating possible rehabilitation interventions. It is important to develop a more user-friendly method of delivering FES,

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evaluating the effect on bone of lower cycling cadences during FES, or undertaking further studies to examine the feasibility and effectiveness of very early training to prevent muscle atrophy. Finally, it is also mandatory to promote adoption of a standardized classification system for rehabilitation interventions.

#### References

- Kim SW, Kim RC, Choi BH, Gordon SK (1988) Non-traumatic ischaemic myelopathy: a review of 25 cases. Paraplegia 26: 262-272.
- Foo D, Rossier AB (1983) Anterior spinal artery syndrome and its natural history. Paraplegia 21: 1-10.
- Mori S, Sadoshima S, Tagawa K, Iino K, Fujishima M (1993) Massive spinal cord infarction with multiple paradoxical embolism: a case report. Angiology 44: 251-256.
- Ditunno JF Jr, Young W, Donovan WH, Creasey G (1994) The international standards booklet for neurological and functional classification of spinal cord injury. American Spinal Injury Association. Paraplegia 32: 70-80.
- Kirshblum SC, Waring W, Biering-Sorensen F, Burns SP, Johansen M, et al. (2011) Reference for the 2011 revision of the International Standards for Neurological Classification of Spinal Cord Injury. J Spinal Cord Med 34: 547-554.
- Salvador de la Barrera S, Barca-Buyo A, Montoto-Marqués A, Ferreiro-Velasco ME, Cidoncha-Dans M, et al. (2001) Spinal cord infarction: prognosis and recovery in a series of 36 patients. Spinal Cord 39: 520-525.
- Weidauer S, Nichtweiss M, Lanfermann H, Zanella FE (2002) Spinal cord infarction: MR imaging and clinical features in 16 cases. Neuroradiology 44: 851-857.
- Masson C, Pruvo JP, Meder JF, Cordonnier C, Touzé E, et al. (2004) Spinal cord infarction: clinical and magnetic resonance imaging findings and short term outcome. J Neurol Neurosurg Psychiatry 75: 1431-1435.
- 9. Medical Research Council (1976) Aids to examination of the peripheral nervous system. Memorandum no. 45, London.
- Hall KM, Cohen ME, Wright J, Call M, Werner P (1999) Characteristics of the Functional Independence Measure in traumatic spinal cord injury. Arch Phys Med Rehabil 80: 1471-1476.
- 11. Mahoney FI, Barthel DW (1965) Functional evaluation: the barthel index. Md State Med J 14: 61-65.
- 12. Ditunno JF Jr, Ditunno PL, Graziani V, Scivoletto G, Bernardi M, et al. (2000)

Walking index for spinal cord injury (WISCI): an international multicenter validity and reliability study. Spinal Cord 38: 234-243.

- Brewer LA 3rd, Fosburg RG, Mulder GA, Verska JJ (1972) Spinal cord complications following surgery for coarctation of the aorta. A study of 66 cases. J Thorac Cardiovasc Surg 64: 368-381.
- 14. Satran R (1988) Spinal cord infarction. Stroke 19: 529-532.
- Lee C, Woodring JH, Walsh JW (1991) Carotid and vertebral artery injury in survivors of atlanto-occipital dislocation: case reports and literature review. J Trauma 31: 401-407.
- Hughes JT, Brownell B (1964) Cervical spondylosis complicated by anterior spinal artery thrombosis. Neurology 14: 1073-1077.
- Foo D, Rossier AB, Cochran TP (1984) Complete sensory and motor recovery from anterior spinal artery syndrome after sprain of the cervical spine. A case report. Eur Neurol 23: 119-123.
- Schreiber AL, Formal CS (2007) Spinal cord infarction secondary to cocaine use. Am J Phys Med Rehabil 86: 158-160.
- O'Farrell R, Thornton J, Brennan P, Brett F, Cunningham AJ (2000) Spinal cord infarction and tetraplegia--rare complications of meningococcal meningitis. Br J Anaesth 84: 514-517.
- Elksnis SM, Hogg JP, Cunningham ME (1991) MR imaging of spontaneous spinal cord infarction. J Comput Assist Tomogr 15: 228-232.
- Barbeau H, Ladouceur M, Mirbagheri MM, Kearney RE (2002) The effect of locomotor training combined with functional electrical stimulation in chronic spinal cord injured subjects: walking and reflex studies. Brain Res Brain Res Rev 40: 274-291.
- McKinley W, Sinha A, Ketchum J, Deng X (2011) Comparison of rehabilitation outcomes following vascular-related and traumatic spinal cord injury. J Spinal Cord Med 34: 410-415.
- Galea MP (2012) Spinal cord injury and physical activity: preservation of the body. Spinal Cord 50: 344-351.
- Baldi JC, Jackson RD, Moraille R, Mysiw WJ (1998) Muscle atrophy is prevented in patients with acute spinal cord injury using functional electrical stimulation. Spinal Cord 36: 463-469.
- 25. Hasnan N, Ektas N, Tanhoffer AI, Tanhoffer R, Fornusek C, et al. (2012) Exercise Responses during FES Cycling in Individuals with Spinal Cord Injury. Med Sci Sports Exerc.
- Miyai I, Suzuki M, Hatakenaka M, Kubota K (2006) Effect of body weight support on cortical activation during gait in patients with stroke. Exp Brain Res 169: 85-91.