Available online at www.medicinescience.org

CASE REPORT

Medicine Science International Medical Journal

Medicine Science 2024;13(4):1020-3

Rehabilitation of a patient with late motor neuron syndrome after electric shock

Tuba Tulay Koca, Nurhayat Koca

Sütçü İmam University, Faculty of Medicine, Department of Physical Medicine and Rehabilitation, Kahramanmaraş, Türkiye

Received 09 October 2024; Accepted 23 November 2024
Available online 01 December 2024 with doi: 10.5455/medscience.2024.10.119

Abstract

The development of motor neuron disease has been reported in a small number of cases after electric shock trauma. Our case is a 61-year-old male telecomunication worker who presents with complaints of progressive loss of strength in his right upper and lower extremities 22 years after an electric shock. It is accompanied by unsteadiness in walking, difficulty in speaking and swallowing. In electroneurophysiological examination; chronic changes compatible with preganglionic involvement were observed in the lower and upper extremities. On physical examination, the patient speaks dysarthric. Mobilized with walker, functional ambulation scale (FAS) is stage 2. There is 3/5 strength in the right upper extremity and 2-2+/5 strength in the right lower extremity. Although rare, neurological complications may occur decades later after electric shock. There are a limited number of case reports and observational meta-analytic studies in this field in the literature.

Keywords: Electric shock, motor neurone disease, occupational disease

Introduction

Electric shock can damage many organs. Although it can result in death in the acute period, we also see its effects in the short and long term. Electric shock may cause complications in the central and peripheral nervous systems by causing demyelination and axonal changes [1]. However, case reports regarding late complications are limited. Bulbar dysfunction has also been reported in a small number of cases in these individuals [2]. Hydrocephalus, cerebral venous thrombosis and amniotrophic lateral sclerosis are identified among the long-term sequelae [3]. Although electrical injuries are mostly seen in young men working in the industry, they also occur in home accidents. While neurological or neuropsychological complaints may develop in these people within hours or days, findings are observed after many years in a small number of cases [4]. Neurological complications occurring decades after electric shock have been reported in a small number of case reports in the literature.

Motor neuron diseases are neurodegenerative diseases in which upper and or lower motor involvement and bulbar involvement are observed, and they present with specific symptoms that vary depending on which part of the motor neuron system is affected. In the most common form of the disease, Amyotrophic lateral sclerosis, upper and lower motor neurons are involved. When only upper motor neuron involvement is prominent, Primary Lateral Sclerosis (PLS) and Hereditary Spastic Paraparesis (HSP) come to mind; when only lower motor neuron involvement occurs, Progressive Muscular Atrophy (PMA), Spinal Muscular Atrophy (SMA), Hirayama Disease, Kennedy Syndrome, Poliomyelitis and Postpolio syndrome (PPS) should be considered [5].

In addition to genetic causes, various molecular mechanisms such as excitotoxicity, mitochondrial disorder, changes in axonal transport, oxidative stress, accumulation of folded proteins, and neuroinflammation are also held responsible for the pathophysiology of the disease [6].

Here, we present our 61-year-old patient with a prediagnosis of motor neuron disease, who we took to 3-week neurorehabilitation program.

CITATION
Koca TT, Koca N. Rehabilitation of a patient with late motor neuron syndrome after electric shock. Med Science. 2024;13(4):1020-3.

Corresponding Author: Tuba Tulay Koca, Sütçü İmam University, Faculty of Medicine, Department of Physical Medicine and Rehabilitation, Kahramanmaraş, Türkiye
Email: tuba_baglan@yahoo.com

DOI: 10.5455/medscience.2024.10.119

Case Presentation

A 61-year-old male patient was admitted with complaints of loss of strength that started in the right lower extremity, accompanied by loss of strength in the right upper extremity over time, and progressed for 10 years, accompanied by difficulty in speaking and swallowing, and inbalance. The patient was taken to an inpatient neurorehabilitation program.

The patient's medical history includes a history of electric shock at the level of his right wrist when he was 29 years old. He was working as a worker in a telecommunications company and was electrocuted while on duty. He uses Maraş herb, has no known systemic disease, uses acetyl salicylic acid 1x100 mg po daily.

Diffusion cranial magnetic resonance imaging (MRI) was consistent with nonspecific ischemic foci. Cervical, thoracic and lumbar MRI findings include nonspecific spondylolytic degenerative changes consistent with age. Routine laboratory tests are within normal limits. There are no findings supporting malignancy in the abdomen and thorax tomography examined for malignancy. There are pleural plaques in the lungs that support asbestos exposure.

Neuroelectrophysiological examination findings; Sensory conduction study, right ulnar, and sural sensory nerve action potential are normal. Motor conduction study, right ulnar, and right tibial compound muscle action potential are normal. Left peroneal nerve compound muscle action potential could not be obtained. Right ulnar, sural, tibial sensory and motor nerve conduction studies are normal. Needle electroneuromyography: Mild twitching in the right extensor digitorum brevis muscle. Chronic neurogenic changes in muscles. Chronic changes compatible with preganglionic involvement were observed in the lower and upper extremities.

On physical examination of the patient: Dysarthric speech is present. Right hand and foot muscles are atrophic (Figure 1). There is a scar on the left shoulder belonging to the electrical outlet. There are also scar marks on the right hand and front of the right thigh due to electric shock. Right upper extremity proximal muscle strength was 4/5; distal muscle strength was 3/5, right lower extremity muscle strength common was 2-2+/5 according to Medical Research Council (MRC) Muscle Strength Scale.

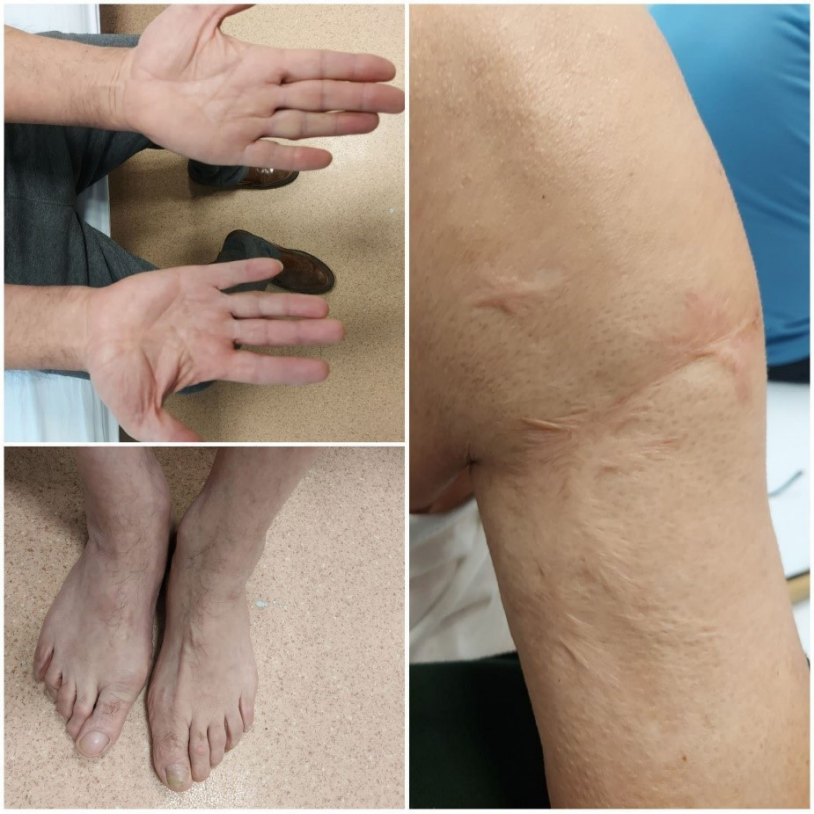

Figure 1. The patient has muscle atrophy in the right hand and foot. Scar (exit site) on the left shoulder due to electric shock

Functional ambulation scale (FAS) was stage 3, mobilized with walker. He has no spasticity. Sitting balance is available. Fasciculation, tandem walk, Romberg test, Babinsky reflex are positive. Deep tendon reflexes were hyperactive bilaterally. An informed consent form was taken from the patient.

Rehabilitation Program

Our patient benefited from 3-week neurorehabilitation treatment. Transcutaneous electrical stimulation therapy was applied to the right hand dorsiflexor, hand interosseous muscles, flexor digitorum profundus and superficialis, finger adductor and

DOI: 10.5455/medscience.2024.10.119

abductor muscles, knee stabilizer, extensor, fleksor muscle group, right foot dorsiflexor and evertor group muscles for 20 minutes a day. 30-40 minutes daily strengthening, joint range of motion and moderate stretching exercise program for the distal muscle groups of the right upper and lower extremities was applied. In the control examination at the end of the program, the right hand distal muscle strength was 4/5; right lower extremity muscle strength increased to 3+/5.

For loss of balance, proprioception, core stabilization and strengthening of gluteal/quadriceps muscle groups and walking exercise training program were applied 3 days a week for 45 minutes under the guidance of a physiotherapist. The patient, who was mobilized with a walker when he first arrived, was able to mobilized with canedien at the end of the program. Ankle foot orthosis (AFO) was prescribed to prevent deformity development in the right foot and to provide optimalization in walking. No orthosis prescription was required for the right hand and wrist.

Discussion

Motor neuron diseases are progressive, terminal neurodegenerative conditions, and amyotrophic lateral sclerosis is the most common form. Amyotrophic lateral sclerosis is mostly limited to anterior horn motor cells and pyramidal tract involvement. The average life expectancy is 3.5 years, but some cases live longer than 5 years. Advanced age and bulbar involvement are characterized by poor prognosis [7,8]. It is thought that this disease is more common in men working in manual labor and that chronic trauma may play a role in the etiology at rates ranging from 10 to 30%. In the current literature, a significantly lower ulnar/median distal motor latency ratio and a considerably higher compound muscle action potential ratio are detected in ALS patients. In some atypical cases, a slowdown in conduction velocity and a decrease in compound muscle action potential amplitudes have been reported in the affected extremity [9-11]. While the electrophysiological data of our case were compatible with the literature, nerve conductions were normal. In the study conducted by Bailey B et al. [12], neurological or neuropsychological symptoms developed after electric shock with a frequency of one in four, and the median duration of these symptoms was found to be 52 days.

There is no cure for the disease yet, neurological rehabilitation practices aim to improve the current functional status of patients and thus increase their quality of life. There is a significant relationship between occupational trauma and electrical exposure in the development of motor neuron diseases. There are a limited number of case reports and observational meta-analyses supporting the relationship in the current literature [13,14].

As in our case, a small number of cases were observed in which neurological complications occurred after 2 decades. In the case reports of Gallagner et al. [15] from 30 years ago, neurological complications were observed within 2-3 years in 5 of 17 electric shock cases; In the rest, no findings were observed before 10 years. In all cases, motor neuron disease developed under the age of 45. In our case, findings of motor neuron disease appear at the age of 51, and the way they appear differs, with the loss of muscle strength being more prevalent in the right half of the body where the electric shock occurred. Findings appear as long as 22 years after electric shock. Here, it is difficult to definitively attribute the patient's motor neuron disease to electric shock; this clinic may also have occurred coincidentally. It is necessary to make a good differential diagnosis.

Neurorehabilitation plays a critical role in the treatment of motor neuron diseases by improving quality of life, ensuring functional independence and symptom management. The multidisciplinary approach, which includes physical, occupational, speech therapy and psychological support, reduces the individual's disability, pain and optimizes motor function. Studies support that neurorehabilitation improves mobility, communication, respiratory functions and contributes positively to general well-being [16,17].

Conclusion

In conclusion, despite clinical advances, motor neuron diseases remain fatal, and current clinical treatments provide limited benefit. All people presenting with findings of motor neuron disease should be questioned about their history of electric shock, and care should be taken in this regard in the long-term follow-up of these people.

Conflict of Interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Patient Informed Consent
An informed consent form was taken from the patient.

References

1. Fu PK, Hsu HY, Wang PY. Delayed reversible motor neuronopathy caused by electrical injury. J Chin Med Assoc. 2008;71:152-4.
2. Parvathy G, Shaji CV, Kabeer KA, Prasanth SR. High-voltage electrocution causing bulbar dysfunction. J Neurosci Rural Pract. 2016;7:453-5.
3. Yiannopoulou KG, Papagiannis GI, Triantafyllou AI, et al. Neurological and neurourological complications of electrical injuries. Neurol Neurochir Pol. 2021;55:12-23.
4. Peters S, Visser AE, D'Ovidio F, et al. Associations of electric shock and extremely low-frequency magnetic field exposure with the risk of amyotrophic lateral sclerosis. Am J Epidemiol. 2019;188:796-805.
5. Masrori P, Van Damme P. Amyotrophic lateral sclerosis: a clinical review. Eur J Neurol. 2020;27:1918-29.
6. Rojas P, Ramírez AI, Fernández-Albarral JA, et al. Amyotrophic lateral sclerosis: a neurodegenerative motor neuron disease with ocular involvement. Front Neurosci. 2020;14:566858.
7. Chen GX, Mannetje A', Douwes J, et al. Associations of occupational exposures to electric shocks and extremely low-frequency magnetic fields with motor neurone disease. Am J Epidemiol. 2021;190:393-402.
8. Brown RH, Al-Chalabi A. Amyotrophic lateral sclerosis. N Engl J Med. 2017;377:162-72.

DOI: 10.5455/medscience.2024.10.119

9. Bracco L, Antuono P, Amaducci L. Study of epidemiological and etiological factors of amyotrophic lateral sclerosis in the province of Florence, Italy. Acta Neurol Scand. 1979;60:112-24.

10. Milanese C, Martin L. Amyotropic lateral sclerosis and trauma. Acta Neurol Belg. 1970;70:482-91.

11. Rosati G, Pinna L, Granieri E, et al. Studies on epidemiological, clinical, and etiological aspects of ALS disease in Sardinia, Southern Italy. Acta Neurol Scand. 1977;55:231-44.

12. Bailey B, Gaudreault P, Thivierge RL. Neurologic and neuropsychological symptoms during the first year after an electric shock: results of a prospective multicenter study. Am J Emerg Med. 2008;26:413-8.

13. Gallagher JP, Sanders M. Trauma and amyotrophic lateral sclerosis: a report of 78 patients. Acta Neurol Scand. 1987;75:145-50.

14. Tashiro K, Inoue I, Ohyagi Y, et al. A case of mortor neuron syndrome with onset 9 months after electrical injury. Rinsho Shinkeigaku. 2000;40:732-5.

15. Gallagher JP, Talbert OR. Motor neuron syndrome after electric shock. Acta Neurol Scand. 1991;83:79-82.

16. Orient-López F, Terré-Boliart R, Guevara-Espinosa D, Bernabeu-Guitart M. Neurorehabilitation treatment of amyotrophic lateral sclerosis. Rev Neurol. 2006;43:549-55.

17. Sathasivam S. Managing patients with amyotrophic lateral sclerosis. Eur J Intern Med. 2009;20:355-8.