



Society [and] Swiss Foot and Ankle Society 21 (1): 18-25. PMID 10710257. edit

- Lynch, D., Goforth, W., Martin, J., Odom, R., Preece, C., Kottor M., 1998, Conservative treatment of plantar fasciitis. A prospective study. Journal of the American Podiatric Medical Association 88 (8): 375-80. PMID 9735623.
- Osborne, H.R., Allison, G.T., 2006, Treatment of plantar fasciitis by LowDye taping and iontophoresis: Short term results of a double blinded, randomised, placebo controlled clinical trial of dexamethasone and acetic acid Commentary". British Journal of Sports

 Medicine
 40
 (6):
 545–549.

 doi:10.1136/bjsm.2005.021758.
 PMC
 2465091. PMID 16488901. edit

- Genc, Hakan., Meryem Saracoglu, Bans Nacir, Hatice Rana Erdem, Mahmut Kacar, 2005, Longterm ultrasonographic follow-up of plantar fasciitis patients treated with steroid injection". Joint Bone spine 72 (1): 61–5. doi:10.1016/j.jbspin.2004.03.006. PMID 15681250.
- Haake M, Buch M, Schoellner C, Goebel F, 2003, Extracorporeal shockwave therapy for plantar fasciitis: randomised controlled

multicentre trial. British Medical Journal 327:1-5.

Tsai, Wen-Chung, Chih-Chin Hsu, Carl P. C. Chen, Max J. L. Chen, Tung-Yang Yu, Ying-Jen Chen, 2006, Plantar fasciitis treated with local steroid injection: comparison between sonographic and palpation guidance". Journal of Clinical Ultrasound 34 (1): 12–6. doi:10.1002/jcu.20177. PMID 16353228.

- Rompe JD, Furia J, Weil L, Maffulli N, 2007, Shock wave therapy for chronic plantar fasciopathy. Br. Med. Bull. 81-82: 183–208. doi:10.1093/bmb/ldm005. PMID 17456546.
- Rompe JD, et al., 2003, Shockwave application for chronic plantar fasciitis in running athletes a prospective, randomized, placebo controlled trial. Am J Sports Med; 31:268-275.
- Thomson CE, Crawford F, Murray GD, 2005, The effectiveness of extra corporeal shock wave therapy for plantar heel pain: a systematic review and meta-analysis". BMC Musculoskelet Disord 6: 19. doi:10.1186/1471-2474-6-19. PMC 1097736. PMID 15847689.

Science, Movement and Health, Vol. XIII, ISSUE 2 supplement, 2013 September 2013, 13 (2), 602-609

# CONTROVERSIES IN A CASE OF NEUROBORELIOSIS VERSUS AMYOTROPHIC LATERAL SCLEROSIS AND METHODS OF RECOVERY

# DOCU AXELERAD ANY<sup>1</sup>, DANIEL DOCU AXELERAD<sup>2</sup>

# Abstract

*Purpose*.Because of difficulties in making the diagnosis of neuroborreliosis the physician must correlate clinical with laboratory data to confirm the diagnosis.Early on, personality changes, psychiatric symptoms, or cognitive manifestations may be the first, and occasionally the only, symptoms that the patient or family is aware of. Amyotrophic lateral sclerosis (ALS) is the most common degenerative disease of the motor neuron system. The cause of ALS is unknown, although 5-10% of cases are familial. The diagnosis of ALS is primarily clinical. Electro diagnostic testing contributes to the diagnostic accuracy

*Material and methods:* we exam a 63 year patient hospitalized in Neurology Department of Clinical Hospital of Constanta, between 10-20.12.2012.

*Discussion.* Our patient has an history of exposure to B. Burgdorferi one year before the apparition of symptoms. Family describes personality changes and mild cognitive manifestations. We must say that in past history he has an ethanolic abuse. Next symptoms were muscle pain and trouble of gait. It was suspected to have borreliosis and lab results show a little increase of IGM antiborrelia. After one year of antibiotic treatment the gait is worse and appeared trouble of speech and patient was admitted in our department. On clinical examination we found sign of upper and

<sup>1</sup>Ovidius University, Constanta, Romania, General Medicine Faculty, ROMANIA

<sup>2</sup>Ovidius University, Constanta, Romania, Physical Education And Sport Faculty, ROMANIA Email: docuaxy@yahoo.com





lower motor neuron symptoms. We perform serum immunology and LCR for borrelia was norm, MRI cerebral and cervical scan was normal, EMG show fasciculation and fibrillation potentials. We begin a neuromotor and physiologic rehabilitation.

*Conclusions:* it is important if the symptoms are not clear and the results of immunology is not complete to not begin treatment for borreliosis and the interdisciplinary consult is necessary to complete the diagnosis. After antibiotic treatment we observe that clinical sign worst.

Key words: neuroboreliosis, amyotrophic lateral sclerosis, differential diagnosis

# Introduction

Early on, personality changes, psychiatric symptoms, or cognitive manifestations may be the first, and occasionally the only, symptoms that the patient or family is aware of.

Amyotrophic lateral sclerosis (ALS) is the most common degenerative disease of the motor neuron system.

The cause of ALS is unknown, although 5-10% of cases are familial.

The diagnosis of ALS is primarily clinical. Electro diagnostic testing contributes to the diagnostic accuracy.

# Material and methods

We exam a 63 year patient hospitalized in Neurology Department of Clinical Hospital of Constanta, between 10-20.12.2012 .We initiate a rehabilitation program for preventing the spasticity.

# **Results and discussion**

Table 1. The nerves

Our patient has an history of exposure to B. Burgdorferi one year before the apparition of symptoms. Family describes personality changes and mild cognitive manifestations. We must say that in past history he has an ethanolic abuse. Next symptoms were muscle pain and trouble of gait. It was suspected to have borreliosis and lab results show a little increase of IGM antiborrelia. After one year of antibiotic treatment the gait is worse and appeared trouble of speech and patient was admitted in our department. On clinical examination we found sign of upper and lower motor neuron symptoms.

We perform a cervical and thoracal MRI with disk hernia C6-7, Angi-CT of carotid vessel shows carotidian bulbar calcification bilateral, cerebral MRI normal.

We perform serum immunology and LCR for borrelia was norm. Borrelia Ig G, Ig M in LCR and serum. Albumine in LCR 356 mg/dl, albumine in serum 40.4g/l, QAlb 8.8, Ig G in LCR 30MG/L, Ig G IN SERUM 8.22G/L, QIgG 3.6, IgM in LCR 0.19mg/l, IgM in serum 0.5 g/l, QIg M( all results show absence of intratecal sintesis of Ig G and Ig M).

	Inserti	Spontaneous				Motor unit potential				
EMG	on activit y	Fibrill at.	PSW	Fascic ul.	Other discharg es	Amp	Dur	Poly	Recruitme nt pattern	
Tibialis anterior R		-	++		-	+	+	-	sarac	
Vastus lateralis R		-	-	-	-	+	+	_	sarac	
Tibialis anterior L		+	-	-	-	+	+	-	sarac	
Gastroc caput med L		-	+++	-	-	+	+	-	sarac	
Vastus lateralis L		-	-	-	-	+	+	+	sarac	
Abd pollicis brev R		-	-	-	-	+	+	-	sarac	
Abd dig min (man) R		-	-	-	-	+	+	-	sarac	
Biceps R		+	+	-	-	+	+	-	Sarac	





MNCV	Site/Segment	Latency	Amplitud e	Duratio n	Area	Distan ce	NCV
		ms	mV	ms	mVms	mm	m/s
Medianus R	Wrist-Abp	6.6	1.7	13.2	9.3		
	Elbow-Wrist	11.5	1.2	12.4	7.8	195	39.9
Ulnaris R	Wrist-ADM	3.6	2.9	9.1	7.9		
	Down Elbow-Wrist	7.6	2.4	9.2	8.5	205	51.5
Peroneus R	maleolla lat-EDB	4.4	2.1	7.2	8.4		
	fibulla-maleolla lat	10.6	2.1	9.4	9.4	280	45.2
Peroneus L	maleolla lat-EDB	4.3	2.4	9.8	8.2		
	fibulla-maleolla lat	10.5	2.2	9.7	8.4	270	44.0

SNCV	Site/Segment	Latency	Amplitud e	Duratio n	Area	Distan ce	NCV
		ms	uV	ms	uVms	mm	m/s
Medianus R	Wrist-index finger	3.4	22.7	3.1	24.2	150	43.9
Ulnaris R	Wrist-V finger	2.2	13.9	1.7	7.86	115	51.6
Suralis R	Leg-Malleolus Lat	2.4	6.31	1.7	104.4	100	41.5
Suralis L	Leg-Malleolus Lat	2.9	3.39	1.9	54.0	120	41.4

MUD	n°	Durate	Amplitu de	Area	Phases	Turns	Rise time
NIOF		ms	uV	uVms			us
Tibialis anterior R	1	13.9	617.1	1600.0	3	3	2100.0
	2	11.1	454.2	1000.0	2	2	3100.0
	3	14.5	953.4	1500.0	6	8	1000.0
	4	11.4	410.7	1000.0	2	2	2200.0
	5	15.4	1900.0	3900.0	2	3	1300.0
Mean values		13.3	867.1	1800.0	(0% poly.)		
Tibialis anterior L	1	10.5	477.3	921.3	1	3	4300.0
	2	13.3	1800.0	2800.0	3	8	1400.0
	3	10.4	995.4	2000.0	4	6	1500.0
Mean values		11.4	1090.9	1907.1	(0% poly.)		
Gastroc caput med L	1	11.5	1200.0	2200.0	3	3	1500.0
	2	8.2	313.2	627.9	2	2	2000.0
	3	9.8	523.9	967.3	2	2	2000.0
Mean values		9.83	679.0	1265.1	(0% poly.)		
Vastus lateralis L	1	12.4	1000.0	2100.0	3	3	1300.0
Mean values		12.4	1000.0	2100.0	(0% poly.)		
Vastus lateralis L	1	13.1	1000.0	2100.0	3	3	1300.0
	2	14.7	3800.0	4400.0	8	15	568



Ovidius University Annals, Series Physical Education and Sport / SCIENCE, MOVEMENT AND HEALTH

Vol. XIII, ISSUE 2 supplement, 2013, Romania The journal is indexed in: Ebsco, SPORTDiscus, INDEX COPERNICUS JOURNAL MASTER LIST, DOAJ DIRECTORY OF OPEN ACCES JOURNALS, Caby, Gale Cengace Learning, Cabell's Directories



MUP	n°	Durate	Amplitu de	Area	Phases	Turns	Rise time
		ms	uV	uVms			us
	3	9.3	630.0	940.7	2	2	1400.0
Mean values		12.4	1810.0	2480.2	(-1431655765% poly.)		
Abd dig min (man) R	1	9.5	2100.0	2600.0	3	3	1600.0
	2	19.1	3300.0	7700.0	4	6	1500.0
Mean values		14.3	2700.0	5150.0	(0% poly.)		
Biceps R	1	16.5	2200.0	5300.0	3	3	2300.0
	2	18.2	206.6	738.7	2	2	3600.0
Mean values		17.4	1203.3	3019.4	(0% poly.)		

Figure 1. Results

















#### VCM:

Nerves median, ulnar dr, peronier bilateral - with amplitude CMAP decrease, VCM normal.

# VCS:

Nerves median, ulnar dr, sural bilat – with amplitude SNAP and VCS normal.

#### EMG with needle:

At the level of muscle examined we observe pathological spontaneous activity (PSW ++, little fibrillation).

Recutare pattern poor. PUM with duration and amplitude increased.

We begin a neuromotor and physiologic rehabilitation. Rehabilitation programme objectives: induce of voluntar motor activity; prevent wrong movement; prevent muscle retractures and joints diformities, decrease spasticity. Rehabilitation programme: we used physical programme for reduce pain, spasticity ALMEIDA (2012), ASHWORTH (2012), BALDINGER (2012) and also kinetic method for each objective. In each month we followed the evolution using specific scale assessment.

#### Conclusions

It is important if the symptoms are not clear and the results of immunology are not complete to not begin treatment for borreliosis and the interdisciplinary consult is necessary to complete the diagnosis. After antibiotic treatment we observe that clinical sign worst.

### References

- Almeida, JP., Silvestre, R., Pinto, AC., De Carvalho, M., 2012, Exercise And Amyotrophic Lateral Sclerosis.Neurol Sci. 2012 Feb;33(1):9-15
- Ashworth, NL., Satkunam, LE., Deforge, D., 2012, Treatment For Spasticity In Amyotrophic Lateral Sclerosis/Motor Neuron Disease. Cochrane Database Syst Rev. 2012 Feb 15;2:CD004156
- Baldinger, R., Katzberg, HD., Weber, M., 2012, Treatment For Cramps In Amyotrophic Lateral Sclerosis/Motor Neuron Disease. Cochrane Database Syst Rev. 2012 Apr 18;4:CD004157.
- Blackhall, LJ. (2012), Amyotrophic Lateral Sclerosis And Palliative Care: Where We Are, And The Road Ahead. Muscle Nerve. 2012 Mar;45(3):311-8





- Ludolph, AC., Brettschneider, J., Weishaupt, JH., 2012, Amyotrophic Lateral Sclerosis. Curr Opin Neurol. 2012 Oct;25(5):530-5.
- Turner, MR., Barnwell, J., Al-Chalabi, A., Eisen, A., 2012, Young-Onset Amyotrophic Lateral Sclerosis: Historical And Other Observations.Brain, 2012 Sep;135(Pt 9):2883-91.
- Mandell, H., Steere, AC., Reinhardt, BN., Yoshinari, N., Munsat, TL., Brod, SA., Clapshaw PA.,

1989, Lack Of Antibodies To Borrelia Burgdorferi In Patients With Amyotrophic Lateral Sclerosis, N Engl J Med., 1989 Jan 26; 320(4):255-6.

Waisbren, BA., Cashman, N., Schell, RF., Johnson, R., 1987., Borrelia Burgdorferi Antibodies And Amyotrophic Lateral Sclerosis, Lancet, 1987 Aug 8;2(8554):332-3.