

THE ISCHEMIC NEUROPATHY OF THE LOWER LIMBS – INVOLVEMENT OF THE MOTOR FIBRES

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Keywords: ischemic neuropathy, multiplex mononeuropathy

Abstract: Secondary affection of the peripheral nerves of lower limbs during the chronic obliterative arteriopathy stands for ischemic neuropathy. The aim of this article is to establish the involvement degree of the peripheral nervous system and the muscle fiber in the ischemic pathology, by comparing the functional / clinical aspects and also to set an electroneurographic and electromyographic investigation grid for patients with ischemic pathology. The study results showed that the decrease of the motor potential amplitude is much more common in comparison with the decrease of conduction velocity, suggesting a predominantly axonal impairment of the motor nerves. Both the motor potential amplitude as well as the motor velocity decrease differently on the studied nerves, fact that suggests the presence of multiplex mononeuropathy. A significant proportion of the studied nerves are normal, sustaining the predominantly inhomogeneous effect on the peripheral nerves in ischemic neuropathy. The most commonly encountered electromyographic aspect is the chronic neuropathic type, more rarely with the presence of active denervation signs.

Cuvinte cheie: neuropatie ischemică, mononeuropatie multiplex

Rezumat: Articolul are ca scop evaluarea gradului de interesare a sistemului nervos periferic și a fibrei musculare în patologia ischemică, comparând aspectele funcțional/clinic și își propune să stabilească o grilă de explorare electroneurografică și electromiografică la pacienții cu patologie ischemică a membrelor inferioare. Rezultatele studiului au pus în evidență faptul că scăderea amplitudinii potențialului motor este mult mai frecvent întâlnită comparativ cu scăderea vitezei de conducere, ceea ce sugerează o afectare predominant axonală a nervilor motori. Atât amplitudinea potențialului motor cât și viteza de conducere motorie scad în mod diferit pe nervii studiați, aspect care sugerează prezența mononeuropatiei multiplex. Un procent semnificativ din nervii studiați sunt normali, susținând afectarea predominant inomogenă a nervilor periferici în neuropatia ischemică. Aspectul electromiografic cel mai des întâlnit este traseul de tip neurogen cronic, mai rar cu prezența de semne de denervare activă.

INTRODUCTION

The ischemic neuropathy stands for the secondary damage of the peripheral nerves during the chronic obliterative arteriopathy. Although frequently encountered in the current clinical activity, the literature data regarding this form of neuropathy are scarce and scattered, which implies the need of thorough electrophysiological research in patients with chronic obliterative arteriopathy of the lower limbs.

The morphopathological aspect of the peripheral nerve, encountered in atherosclerotic peripheral vascular diseases, is quite difficult to set, the research data being extremely limited. Although the vascular architecture of the peripheral nerves provides a high degree of resistance, both the chronic as well as the acute ischemic diseases show a process of axonal degeneration of focal character, both of the myelinated as well as of the non-myelinated nervous fibers. Moreover, one could also encounter a demyelination process, secondary to axonal lesions or due to the damage caused to Schwann cells by ischemia. (3,5,13)

Clinically, the neuropathy associated with obliterative arteriopathy has been described through two forms: as a mononeuritis multiplex, where it is considered that the unequal and asymmetrical disposition of the nerves, the dispersion in time and space of the neurological symptoms and the electro-

physiological alterations are connected rather to global ischemic phenomena, than a systemic disorder; as a polyneuropathy with mainly distal damage, more or less symmetrical, especially encountered in high arterial diseases (distal aorta, common iliac arteries, common femoral arteries). (1,3,4,6,11,19,20)

The encountered electrophysiological aspect is that of a monomelic neuropathy, multilineuropathy or distal polyneuropathy with more or less symmetrical disposition. In the case of ischemic neuropathy, the electroneurographic findings are: the decrease of the CMAP (compound muscular action potential) amplitude, the moderate increase of distal latencies, the also moderate decrease of motor and sensorial conduction velocities (MCV and SCV) and the increase of the F wave latency. (2,9,10,12) From an electromyographic point of view, one notices anomalies in the sense of axonal loss (hence sharp positives and fibrillation potentials) in the intrinsic muscles of the leg and to a lesser extent in the anterior tibial, gastrocnemian and solar muscles, reduced recruitment (of neuropathic type) or lack of recruitment, as well as records of re-innervation (large motor unit potentials and / or polyphasic potentials), occurring several months after the lesion. (7,8,14,15)

PURPOSE OF THE STUDY

The objectives consisted of:

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Article received on 25.01.2010 and accepted for publication on 09.02.2010
ACTA MEDICA TRANSILVANICA September 2010; 2(3)266-269

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- assessing the degree of involvement of the peripheral nervous system and muscle fiber in ischemic pathology, comparing the functional/clinical aspects.
- establishing an electroneurographic (ENG) and a myographic (EMG) exploration grids in patients with ischemic pathology of the lower limbs.

MATERIAL AND METHOD

The study was performed on 47 patients with a diagnosis of chronic obliterative arteriopathy of the lower limbs, which addressed to the Clinical Recovery Hospital of Cluj-Napoca. A grid of biochemical tests (including ESR and determinations of glicemia, cholesterol, triglycerides, urea, hemoglobin, WBC and platelets) excluded those patients who had peripheral nervous system involvement with other etiology (non-ischemic). The diagnosis of obliterative arteriopathy was established clinically and through Doppler examinations of the lower limbs. The electrophysiological testing was performed with a Keypoint Portable type device, existent in the Laboratory of electrophysiology "Mircea Serban" of the Clinical Recovery Hospital. The electroneurographic exploration comprised: the stimulus intensity at the maximum amplitude of the action potential, CMAP latency and amplitude at both the proximal and distal stimulation, the motor conduction velocity on the common peroneal and tibial nerves, bilaterally. The electromyographic examination consisted of a detection study at rest and in muscle contraction in extensor digitorum brevis (innervated by the common peroneal nerve) and abductor hallucis (the tibial nerve area), bilaterally. The values obtained were compared with normal values for the parameters of existing record in the literature. Normal values for common peroneal nerve, stimulated distally on the ankle and proximally in popliteal fossa are considered more than 5 mV for potential amplitude and more than 42m/s for conduction velocity. For tibial nerve, stimulated in the same regions, normal values were more than 3 mV for potential amplitude and more then 41m/s for conduction velocity. (7,8,12,16,17) The statistical processing of data was performed using SPSS program.

RESULTS AND DISCUSSION

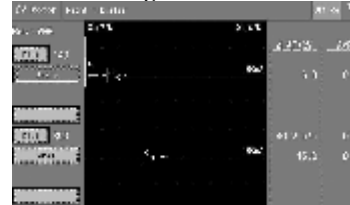
The lot of 47 patients with chronic obliterative arteriopathy confirmed through arterial Doppler examination, without other associated pathologies that could cause neuropathy, consisted of 21,3% women and 78,7% men, in concordance with the existent epidemiological data, which show a higher incidence of this pathology in males. The average age was 63,6 years.

In neuropathy associated with chronic obliterative arteriopathy, electroneurographic aspects encountered are: the decrease of CMAP amplitude, the moderate increase of distal latencies, as well as the moderate decrease of the MCV. Motor conduction velocities are not significantly affected in some groups of patients (probably in early stages of the arterial disease), being predominantly axonal affected, or they may be slightly lower in other described groups of patients, the most likely explanation being the preferential loss of fast conduction, myelinated axons, supported also by some histological studies (5,6,17,21,22).

CMAP amplitude reflects the number of excitable nerve fibers in a semi-quantitative manner (as the number of fibers that respond to a maximal stimulus). When CMAP amplitude decreases, it specifically and sensibly suggests an axonal damage. (Fig. 1.) In the case of neuropathy associated with chronic obliterative arteriopathy, it is lower in case of pain at rest, than in that of intermittent claudication, there being a relationship between the degree of clinical impairment (pain

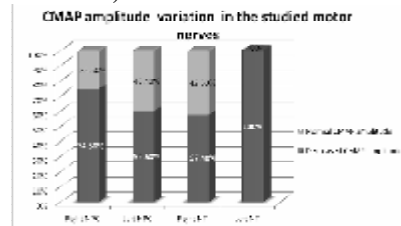
intensity and character) and CMAP amplitude. (2,9,12). The reduction of CMAP amplitude in the symptomatic leg is the most prominent electrophysiological abnormality. The CMAP amplitude is a less sensitive parameter in the cases with moderate symptoms, due to its wide variation, even in normal subjects (16,18).

Figure no. 1. Patient M.I., female, 62 years old, diagnosed with ischemic neuropathy, presenting decreased MCV and CMAP amplitude in the right tibial nerve



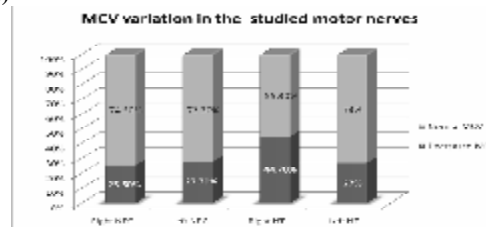
In our studied group of patients we found that there is an increased, irregular frequency of the CMAP decrease with percentages ranging between 59.6% and 100%, both in distal as well as proximal stimulation of the common peroneal and tibial nerves, bilaterally. This certifies axonal damage of the studied nerves and it is consistent with the previous data from literature. (Fig. 2.)

Figure no. 2. Changes in CMAP amplitude in the studied motor nerves (CMAP-compound muscle action potential, Right NPC – right common peroneal nerve, Left NPC – left common peroneal nerve, Right NT – right tibial nerve, Left NT – left tibial nerve)



On the other hand, the decrease of motor conduction velocity in the studied motor nerves occurs more rarely (ranging between 17% and 44.7%), suggesting that the process of demyelination is seen less frequently. (Fig. 3.)

Figure no. 3. MCV variation in the studied motor nerves (VCM - motor conduction velocity, Right NPC – right common peroneal nerve, Left NPC – left common peroneal nerve, Right NT – right tibial nerve, Left NT – left tibial nerve)

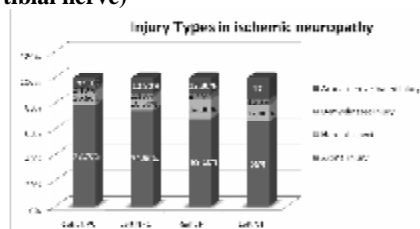


In concordance with the CMAP amplitude decrease and/or the MCV decrease, correlated with the increase of distal latency, one classified the types of lesions encountered in the studied lot into 4 categories: axonal lesions (the most frequent, between 66% and 78,7%), axonal-demyelinating lesions (between 8,5% and 17%) and demyelinating lesions (between 2,1% and 4,3%). One notices the presence of a normal itinerary, with a variable frequency according to the studied nerve (between 10,6% and 14,9%). (Fig. 4.) The axonal lesion was

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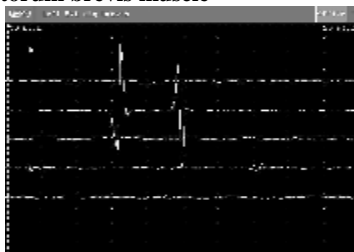
defined based on the Ohr criteria, which consist in a severe decrease in amplitude of normal form and duration CMAP, the minimum increase of distal latencies (but by no more than 50% of the normal average values) and normal conduction velocities or decreased ones, but no more than 40% in the absence of conduction blocks or temporal dispersion. The demyelinating lesions were defined based on the decrease in motor conduction velocity, the increase of distal latency, and normal or slightly low CMAP amplitude.

Figure no. 4. Lesion types in the lot of studied patients (Right NPC- right common peroneal nerve, Left NPC – left common peroneal nerve, Right NT – right tibial nerve, Left NT – left tibial nerve)



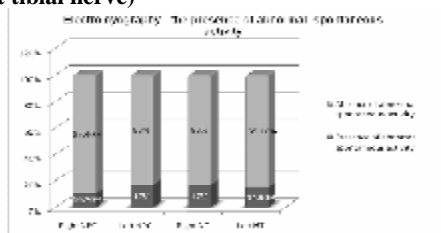
From an electromyographic (needle EMG) point of view, one notices severe anomalies in the muscles, within the lines of positive sharp waves (PSW) axonal loss and fibrillation potentials in the intrinsic leg muscles and, on a smaller scale, in the anterior tibial, gastrocnemian and solar muscles. If the ischemic lesion is severe, there can also be a decreased recruitment (of neuropathic type) or no recruitment at all. Evidence of the reinnervation can be seen months or years after the nerve was damaged, including motor unit action (MUAP) and/or polyphasic potentials.

Figure no. 5. Patient M. N, male, 56 years old, with chronic axonal polyneuropathy, presenting fasciculations in the left extensor digitorum brevis muscle



On the studied lot, we noticed during the electromyography (detection studies in extensor digitorum brevis muscle and the abductor halucis muscle), the presence of pathological activity at rest in a small number of patients (between 10, 7% and 17%).

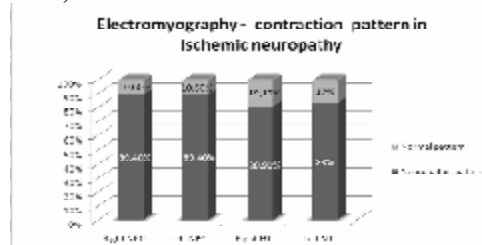
Figure no. 6. Electromyographic aspects: the presence of pathological activity at rest in the studied muscles (Right NPC – right common peroneal nerve, Left NPC – left common peroneal nerve, Right NT – right tibial nerve, Left NT – left tibial nerve)



In contradiction, a chronic neuropathic type itinerary stood out, having a much higher frequency in comparison with

the presence of anomalies at rest (between 80,9% and 89,4%). We noticed a reduced recruitment (of neuropathic type), as well as proofs of reinnervation, including large motor unit action potentials (MUAP) and/or polyphasic potentials.

Figure no. 7. Electromyographic aspects: itinerary types during contraction, in the studied muscles (Right NPC – right common peroneal nerve, Left NPC – left common peroneal nerve, Right NT – right tibial nerve, Left NT – left tibial nerve)



CONCLUZII

The patients diagnosed with chronic obliterative arteriopathy of the lower limbs present altogether damage of the peripheral nerves, which clinically manifests itself in two ways: as a polyneuropathy, with predominantly distal damage, more or less symmetrical or as a mononeuritis multiplex, with unequal and asymmetrical disposition of the affected nerves, which is more common encountered.

The electrophysiological picture consists mainly of the same aspects: monomelic neuropathy, multineuropathy or distal polyneuropathy with more or less symmetrical distribution. Electroneurographically, in the case of ischemic neuropathy one notices the CMAP (compound muscle action potential) amplitude decrease, distal latencies moderate increase, the also moderate decrease of motor and sensorial conduction velocities and the increase of the F wave latency. The damage on the motor nervous fibers, in the case of ischemic neuropathy, manifests itself through a decrease of CMAP amplitude, more frequently encountered than a decrease of the MCV. Both the motor potential amplitude as well as the decrease in motor velocity decrease differently on the studied nerves studied, which suggests the presence of multiplex mononeuropathies. The axonal lesions are much more frequently encountered than the demyelinating or axonal-demyelinating ones. A significant proportion of the studied nerves are normal, sustaining the predominantly inhomogeneous effect on the peripheral nerves in ischemic neuropathy. The most commonly encountered electromyographic aspect is the chronic neurogenic type itinerary, more rarely with the presence of active denervation signs.

REFERENCES

- Andary MK, Barinder SM. Ischemic Monomelic Neuropathy. eMedicine, Sept, 2007
- Blum AS, Rutkove SB. The Clinical NeuroPhysiology Primer. Humana Press Inc., 2007
- Cambier J, Masson M, Dehen H. Neurologie. ed. Masson , Paris, 2000
- Candelise L, Hughes R, Liberati A, Uitdehaag B, Warlow C. Evidence-based Neurology:Management of Neurological Disorders. Blackwell Publishing, 2007
- Câmpeanu E, Șerban M, Dumitru E. Neurologie Clinică. vol II, Ed. Dacia, Cluj-Napoca, 1980
- Chroni E, Papapetropoulou V, Tsolakis J, et al. Chronic ischemic monomelic neuropathy from critical limb ischemia. Neurology. Jun 11 2002;58(11):1705

CLINICAL ASPECTS

7. DeLisa JA, Lee HJ, Baran EM, Lai K, Spielholz N, Mackenzie K. Manual of nerve conduction velocity and clinical neurophysiology. Third Edition, Raven Press, New York, 1994
8. Faye Chiou Tan. EMG Secrets. Hanley & Belfus, Philadelphia, 2004
9. Fournier E. Examen electromyographique et etude de la conduction nerveuse- semiologie electrophysiologique. Ed. Technique et documentation, 1998
10. Fournier E. Atlas d electromyography. Technique et documentation, 2000
11. Laghi Pasini F, Pastorelli M, Beermann U, De Candia S. Peripheral Neuropathy Associated with Ischemic Vascular Disease of the Lower Limbs. *Angiology*, Vol. 47, No. 6, 569-577, 1996
12. Leis AA, Trapani VC. Atlas of Electromyography. 1st Edition, Oxford University Press, 2000
13. Nukada H, Andre M van Rij, Packer S, McMorran D. Pathology of acute and chronic ischaemic neuropathy in atherosclerotic peripheral vascular disease. *Brain*, 1996, 119, 1449-1460
14. Pabón Meneses R, Gila L, Urriza J, Imirizaldu L, Arrechea M, Lacruz F. Pseudo-conduction block in nonsystemic vasculitic neuropath. *An Sist Sanit Navar*. 2009 May-Aug;32(2):279-87.
15. Pease WS, Lew HL, Johnson EW. Johnson's Practical Electromyography. Fourth Edition, Lippincott Williams and Wilkins, 2007
16. Preston DC, Shapiro BE. Electromyography and Neuromuscular Disorders, Clinical-Electrophysiologic Correlations. Second Edition, Elsevier, 2005
17. Ropper AH, Brown RH. Adams and Victor's Principles of Neurology. 8th ed., McGraw Hill, New-York, 2005
18. Rossi CM, Di Comite G. The clinical spectrum of the neurological involvement in vasculitides. *J Neurol Sci*. 2009, Oct 15;285(1-2):13-21.
19. Shy ME, Lewis RA. An approach to patients with peripheral neuropathy. *Continuum*, peripheral neuropathy, vol. 9, number6, dec. 2003
20. Ugalde V, Rosen BS. Ischaemic Peripheral Neuropathy. *Phys Med Rehabil Clin N Am*, 2001. May, 12
21. Weber F, Ziegler A. Axonal neuropathy in chronic peripheral arterial occlusive disease, *Muscle&nerve*, 2002, 26:471-476
22. Weinberg DH, Simovic D, Isner J, et al. Chronic ischemic monomelic neuropathy from critical limb ischemia. *Neurology*. Sep 25 2001;57(6):1008-12.