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Letter to Editor

Clinico-Pathological Aspects of Spinal Nerve Root Entrapment

Naglaa Hussein*

Department of Physical Medicine and Rehabilitation, Albert Einstein College of Medicine, NY and Alexandria University, Alexandria, Egypt

DESCRIPTION

The patterns of the clinical presentation of spinal nerve root entrapment are subject of wide variability [1-3]. Although the sensory presentation patterns dominate different patterns of clinical presentation, other patterns namely; motor or sensorimotor patterns are also encountered [1,4].

This variability in the pattern of clinical presentation is thought to be reflection of the pathogenetic mechanisms involved in spinal nerve root entrapment.

Literature review revealed that the correlation between the patterns of clinical presentations and the pathophysiological mechanisms of spinal nerve root entrapment were not adequately considered. Many questions in this context could be raised for instance how far the degree and/ or the duration of nerve root compression and the resulting pathological changes affect the clinical expression of the disease.

As the sensory presentation is the most frequently encountered pattern of clinical presentations among cases of spinal nerve root entrapment [1-4]. Clinico-pathological analysis of sensory presentations is related to the higher vulnerability of sensory neurons of dorsal root ganglia to ischemia and compression [5-15] as compared to motor root. Furthermore, the regenerative potential of sensory neurons is less than that of the motor neurons [16-18].

The quality of the sensory manifestations i.e. numbness, paresthesia and pain could not be absolutely correlated to the degree of nerve root compression [5,15].

As numbness a major symptom in nerve root compression, was proved *in vivo* study to be involved with ischemia and not mechanical nerve fiber deformation [5,19].

The severity and the degree of spinal nerve root compression directly influence the course of the clinical manifestations of the condition. With low compression for example, 30-50 mm hg the consequent pathological changes of the nerve root will be reversible. If the compression is released after single trauma [5]. Hence its corresponding clinical manifestations.

Whereas, with sustained high pressure or repeated pressure, there will be different kinds of nerve lesions either segmental demyelination or in cases of severe trauma axonal degeneration [5]. This will be reflected on the extent of severity and potential for recovery of the clinical manifestations [5].

The occasional presence of different clinical manifestations in the same patient could be correlated to the presence of different kinds of pathology in individual nerve fibers contained in the spinal nerve root. In this regard, the presence of muscle weakness or sensory deficit that represent loss of nerve function could be associated with state of hyperexcitability of nerve tissue that gives rise to positive symptoms that is; pain, paresthesia and possibly muscle fasciculation from the respective nerves. This condition means that nerve fibers, although have decreased conduction velocity at the site of injury, still being hypersensitive to further mechanical stimulus at the injured segment [5].

The mode of spinal nerve root compression also affects the clinical presentation of the disease 5 and the direct application of this fact is seen when the lumbar nerve roots in the cauda equina are compressed in association with spinal stenosis, the pressure is distributed in circumferential manner around the roots at a slow rate [5].

This situation is compared with the mechanics when a dorso-lateral disc herniation induced displacement of nerve root with unilateral compression and increased intraneural tension. The different clinical symptoms induced by nerve root compression in association with spinal stenosis and herniated nucleus pulposus respectively may in part be a consequence of the different kinds of nerve tissue deformation induced in these two conditions [5].

REFERENCES

1. Ellenberg MR, Honet JC, Treanor WJ. Cervical radiculopathy. Arch Phy Med Rehabil. 1994;75(3):342-52.
2. Cailliet R. Low back pain syndrome. 4th Ed. Philadelphia: F.A Davis company. 1991:205-249.
3. Cailliet R. Neck and arm pain. 3rd Ed. Philadelphia: F. A. Davis company. 1991:124-164.

Correspondence to: Naglaa Hussein, Department of Physical Medicine and Rehabilitation, Albert Einstein College of Medicine, NY and Alexandria University, Alexandria, Egypt, Tel: 3474792686; E-mail: nagla_kashif@hotmail.com

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4. Young WB. The clinical diagnosis of lumbar radiculopathy. Semin ultrasound CT MR. 1993;14(6):385-388.
5. Rdevik B, Brow MD, Lundborg G. Patho-anatomy and pathophysiology of nerve root compression. Spine. 1984;9(1):7-15.
6. Lundborg G, Nordborg C, Rydevik B, Olsson Y. The effect of ischemia on the permeability of the presentation to protein tracers in rabbit tibial nerve. Acta Neuro Scand. 1973;49:287-294.
7. Rydevik B, Lundborg G. Permeability of intrneural microvessel and perineurium following acute, graded experimental nerve compression. Scan J Plast Reconstr Surg. 1977;11:179-189.
8. Hahnenberger RW. Effects of pressure on fast axoplasmic flow. An in vitro study in the vagus nerve of rabbits. Acta Physiol Scan. 1978;104(3):229-308.
9. Ochoa J. Histopathology of common mononeuropathies, nerve repair and regeneration. Edited by DL Jewett, HR Mc Caroll Jr. St Lowis, CV Mosby, 1980;36-52.
10. Rydevik B, Lundborg G, Bagge U. Effects of graded compression on intraneural blood flow-an in vivo study on rabbit tibial nerve. J Hand Surg. 1988;6:3-12.
11. Lundborg G, Rydevik B. Effects of stretching the tibial nerve of the rabbit. A preliminary study on the intraneural microcirculation and the barrier function of the perineurium. J Bone Joint Surg. 1973;55(22):390-401.
12. Yoshizawa H, Kobayashi S, Kubota K. Effects of compression on intraneural blood flow in dogs. Spine .1989;14(11):1220-1225.
13. Jacobs JM, Macfarlane RM, Cavanagh JB. Vascular leakage in the dorsal root ganglia of the rat studied with horse-raddish peroxidase. J Neurol Sci. 1976;29:95-107.
14. Sunderland S. Avulsion of nerve roots In: Vinken PJ, Bruyn GW, editor. Handbook of clinical neurology. Injuries of the spine and spinal cord, Part I, Chap 16. New York, American Elsevier, 1975;393-435.
15. Sunderland S. Nerves and nerves injuries. Second edition Edinburgh, London and New York, Churchill Livingstone, 1978;66-73.
16. Meier C, Sollmann H. Regeneration of cauda equina fibers after transisection and end-to end suture. J Neurol. 1977;215(2):81-90.
17. Nathaniel EJH, Pease DC. Regenerative changes in rat dorsal roots during Wallerian degeneration. J Ultrastr Res. 1963;9(5):533-549.
18. Rosomoff HL, David I, Renee S R. Chronic cervical pain: Radiculopathy or brachialgia. Non- interventional treatment. Spine. 1992;17(105):5362-5366.
19. Lunborg G, Gelberman RH, Minteer-Convery R, Lee YF, Hargens AR. Median nerve compression in the carpal tunnel: The functional response to experimentally induced controlled pressure. J Hand Surg. 1982:7(3):252-259.