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Letter to Editor

Pain Mechanisms in Spinal Nerve Root Entrapment

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DESCRIPTION

Sensory pattern of presentations is the most frequently encountered pattern among all different patterns of spinal nerve root entrapment. The sensory pattern of presentation usually composes of numbness, pain and paresthesia.

Physiological studies demonstrated that spinal nerve root compression per se may not be the cause of pain [1-3].

Certain hypothesis has been made to explain the presence of pain in case of spinal nerve root entrapment [1,2]. As the compression of nerve root is painful only briefly due to distortion and stretching of the nerve membrane with initial signal of depolarization and the generation of action potential. This will be blocked by accommodation, a fall in current density and so sustained pressure brings electrical silence and absence of sensation [2].

If the nerve root however is a site of chronic irritation by a herniated disc or spinal stenosis, pain will be evoked with minor stimulus. In addition, this irritated nerve root creates a state of hyperalgesia with production of certain chemical algesic substances called Neuropetides like prostaglandins, leukotrins, and associated products [2]. These substances are essential to the mechanism of inflammation and pain sensation [1,2].

Once hyperalgesia has been established tissue breakdown substance such as bradykinins, histamine, and other neuropeptides are released which in the presence of state of hyperalgesia, combine to produce pain. The clinical evidence of this stage is the improvement of pain after administration of non-steroidal anti-inflammatory drugs that block the production of prostaglandin [2].

Furthermore, it is useful to consider that injury to the adjacent axons creates a neurophysiologic condition called an Ephapse. The ephapse will allow for depolarization to trigger an action potential for the propagation of pain [2].

The notion that compression per se is not the cause of pain may be difficult to accept especially by surgeon, who obtained good operative results with relief of pain after removal of herniated disc. However, an explanation was postulated as the removal of a short lived herniated disc is accompanied by removal of the offending agent perpetuating the hyperalgesia, hence relief of pain . In more chronic situation however, removal of the disc alone may not relieve the pain. In this case, the peripheral tissue injury which is now severe and sustained perpetuate a continuing nidus for the production of hyperalgesia and algesic substances which interact to generate pain. Moreover, the pain memory that remains active long after an injury may play a role [2].

Another mechanism that has been described in the literature to explain the pain in spinal nerve root entrapment is what is called Chemical Radiculitis [1,2,4,5]. This takes place due to alteration in the biochemistry of the intervertebral disc by both injury and aging with degradation of the collagen/proteoglycan matrix producing glycosylation products which act as foreign body proteins, resulting in an inflammatory reaction. This inflammation was confirmed in experimental animals [6] and in man [7].

Autoimmune mechanisms also have been proposed to be involved in the inflammatory tissue reaction [8].

DISCUSSION AND CONCLUSION

In conclusion, compression of the spinal nerve root by herniated disc is not the sole factor in pain generation. Understanding the different mechanisms of origin of pain in cases of spinal nerve root compression whether cervical or lumbar and correlating it with the duration of disease helps in planning and individualize the management for each condition for optimal pain relief and recovery.

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