

Endang Mutiawati

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Neuropathic pain is an expression of nerve damage that has impaired excitability, in the form of increased excitability in the injured nerve and also healthy nerves around it. The pathophysiology of neuropathic pain is very complex and not yet fully known. Many theories have been proposed but have not been able to be fully explained. The presence of damaged nerve fibers in the nerve carrying pain will cause abnormal activity of afferent fibers in the form of ectopic discharge. Excessive ectopic discharge will become an ectopic generator which is the forerunner of the emergence of neuropathic pain, and eventually causes pain continuously, resulting in central and peripheral sensitization. Treatment of neuropathic pain has not provided satisfactory results. This is because the cause of the pain cannot be removed from the patient's body, such as diabetes mellitus, trigeminal neuralgia, cervical syndrome, carpal tunnel syndrome, lumbar stenosis, nucleus pulposus hernia and carcinoma. Besides that, the basis for this lack of success is because management is still not on target, so far the guidelines that are available only include drugs that inhibit nerve impulses in axons (Carbamazepine, phenitoin); axon tip (Gabapentin, pregabalin); broken up (amitriptyline, SSRI); as well as perceptions in the brain (opioids). While nerve regeneration drugs (neurotropics) have never been included as standard drugs in the management of neuropathic pain, even though by definition it is very clear that neuropathic pain arises from nerve damage. In conclusion, it should be reviewed what drugs should be the main drug in handling neuropathic pain. This is consistent with the concept of the definition of neuropathic pain where neuropathic pain due to primary nerve damage is one type of neuropathy whatever the cause.

References

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