10.5005/jp-journals-10046-0098

REVIEW ARTICLE

What is the Minimum Knowledge of Pain Medicine needed for Other Specialty?

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ABSTRACT

Trying to treat something, i.e, invisible brings forth challenges, obstacles, and belief systems that may prevent proper treatment. Chronic pain clearly falls into this category but the undertreatment of pain exists in all aspects of health care. One main reason for this misery is the existence of inadequate knowledge among clinicians regarding pain management. The basic knowledge about the pain pathway, modulation of pain, concept of sensitization, psychological aspect of pain, use of different pharmacological agents in various types of pain along with pathophysiology of chronic and persistent pain is very important in understanding different kinds of pain in a better way. But unfortunately, in our undergraduate and postgraduate curriculums, not much emphasis has been made on this important matter. Hopefully, busting these deficiencies among clinicians will ensure that no one lives with unnecessary pain or receives an incomplete pain treatment plan again.

Keywords: Chronic pain, Pain pathway, Sensitization.

How to cite this article: Sharma RS, Das G. What is the Minimum Knowledge of Pain Medicine needed for Other Specialty? J Recent Adv Pain 2018;4(1):32-35.

Source of support: Nil

Conflict of interest: None

INTRODUCTION

According to the International Association for the Study of Pain, pain is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage" and is derived from a 1964 definition by Merskey.[1] Since then multiple researches and publication have been advocated in the field of pain, but minimal attempts have been taken toward educating the clinicians regarding various aspects of pain.

The first thing is that clinicians must have adequate knowledge of pain pathway. In our undergraduate and postgraduate days, limited information regarding pathway is shared. We knew that after the stimulation of nociceptors, an action potential is generated at the site of nociceptor (transduction), which are then further carried by A-delta fibers and C fibers toward the higher center (transmission) and make us feel pain via stimulation of sensory cortex (perception). But there are multiple gray areas that must be noticed for proper understanding and management of pain. The primary afferent neurons have their cell body located in the lateral division of the dorsal root ganglion and their central processes enter the dorsal horn in the lateral division of the dorsal root. The first-order neurons synapse with the second-order neurons whose axon crosses the midline and fibers ascends in the contralateral spinothalamic tract. They synapse with the third-order neurons in the thalamus and their axons project into the somatosensory cortex. Complex brainstem circuitry provides both descending inhibitory and excitatory alterations of nociceptive responses at the level of the spinal cord. Cells are situated side by side in the rostral ventromedial medulla (RVM) that imposes either excitatory or inhibitory influence. The periaqueductal gray (PAG) and the anterior pretectal nuclei provide inhibitory input to the spinal cord through connections with descending pathways from the dorsolateral pons. From PAG descending inhibitory fibers (bulbospinal tract) start and some of the fibers project into different limbic nuclei. Descending noradrenergic input to the spinal cord from the dorsolateral pons has been shown to limit responses to acute noxious stimulation.[2] The PAG in the midbrain makes descending connections not only with the RVM[3] but also with the noradrenergic system of the dorsolateral pons.[4] The cholinergic neuronal system is another major reticular system of the midbrain and other portions of the brainstem that provides descending influence on nociception. The cholinergic mechanisms can reduce nociception, as well as potentiate opiate analgesia.[5,6] Several forebrain areas with descending projections have an influence on nociception, including the central nucleus of the amygdale and many parts of the limbic cortex (Fig. 1).[7,8]

Another thing to be noted in pain pathway is the concept of modulation, which is a neural process that acts specifically to reduce the activity in the pain transmission system and reduce the perception of pain in healthy individuals. Some of the modulation systems are:

- Endogenous pain modulation system via endogenous opioids like endorphins and encephalins.

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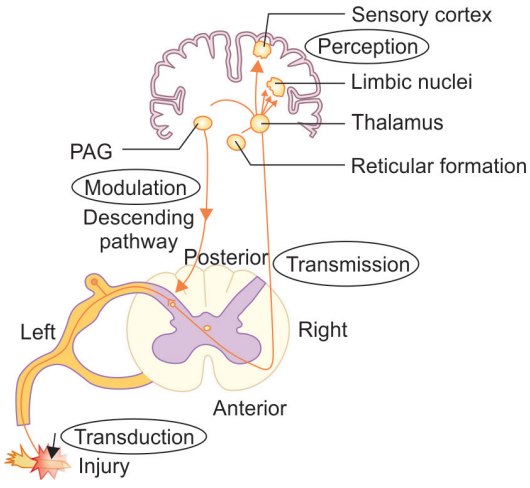

Fig. 1: Afferent pain pathway (Courtesy, basics of pain management by Gautam Das, reprinted with permission)

- Segmental inhibition via release of inhibitory neurotransmitters like gamma-aminobutyric acid and glycine.
- Gate control theory, which states that activation of the larger diameter A-beta fibers leads to inhibition of pain signals transmitted via smaller diameter A-delta and C fibers. Transcutaneous electrical nerve stimulation, spinal cord stimulation, and peripheral nerve stimulation are based on this principle.
- Descending inhibitory pathways, which are activated by serotonin (5HT) and norepinephrine (NE). This pathway sends an inhibitory signal in the dorsal horn and reduces the intensity of signal. Antidepressant medications activate this system and increase the level of 5HT and NE, thus helping in reduction of the intensity of pain.[9-11]

So the pain pathway can be concluded as series of events taking place in the following order:

Transduction → Transmission → Perception →Modulation

Another important knowledge that clinicians must be aware of is the concept of sensitization, which is a phenomenon of inappropriate response to normal stimulus and it is the basis of neuropathic pain. It explains why a simple needle prick pain is amplified multiple times in some individuals. This is of two types:

1. Peripheral sensitization, which is "increased responsiveness and reduced threshold of nociceptive neurons in the periphery to the stimulation of their receptive fields."[12] Following factors play an important role in peripheral sensitization:
   - Upregulation of active nociceptors
   - Sensitization of the primary afferent terminals
   - Ectopic discharges
   - Phenotypic switching
   - Somatic-sympathetic coupling
2. Central sensitization, which is "increased responsiveness of nociceptive neurons in the central nervous system to their normal or sub threshold afferent input."[13] Following are the components of central sensitization:
   - Central reorganization
   - Disinhibition
   - Ectopic discharges
   - Wind-up phenomenon
   - Activation of wide dynamic range neurons

The results of sensitization are increase in intensity, area, and distribution of pain and decrease in the tolerability of pain along with unresponsiveness to conventional analgesics.

Moving to next topic about which clinicians must be aware of is psychological aspect of pain. It is a subjective feeling and every individual at some point of life would have suffered from it. The most commonly reported association of chronic pain is with depression and has bee extensively studied. Prevalence of depression in patients with chronic pain is reported to be between 18 and 85%.[14] Pain has other affective components, such as anxiety, sadness, unpleasant mood, disturbed mood, disturbed sleep, irritability, decreased concentration, and loss of appetite.[15] Effective screening tools have been devised to identify psychological conditions of which famous ones are patient health-related questionnaire scale,[16] hospital anxiety and depression scale,[17] the Gilliam Asperger's disorder scale. All of these scales determine psychological component of pain if present. So proper psychological assessment should be done on a routine basis using one of the above-mentioned tools prior to any management plan.

The last thing that every clinician must have knowledge about is determination of type of pain, whether it is nociceptive or neuropathic, as management of neuropathic pain is different from nociceptive pain. The World Health Organization ladder plays a vital role in management of nociceptive pain, but its role is limited in case of neuropathic pain where anticonvulsants and antidepressants have definite role. So proper assessment of types of pain is mandatory prior to its management as, e.g., treating a patient with diabetic neuropathy with nonsteroidal anti-inflammatory drugs may end into end-stage renal failure. Multiple scale for assessment of neuropathic pain has been devised, of which famous ones are Pain DETECT tool,[18] Leeds assessment of neuropathic symptoms and signs,[19] and Douleur Neuropathique en 4 Questions questionnaire (Table 1).[20]

CONCLUSION

Management of any pain syndrome requires a great depth of knowledge with respect to anatomy, physiology, pharmacology, and psychology. Through this article, we have tried to focus on basic knowledge of pain, which

Table 1: Comparison among various scales for assessment of neuropathic pain[21]

Items	*LANSS*†	*Pain DETECT*†	*DN4*	*NPQ*	*ID Pain*
Symptoms					
Pricking, tingling pin and needles	✓*	✓*	✓*	✓*	✓*
Electric shocks or shooting	✓*	✓*	✓*	✓*	✓*
Hot or burning	✓*	✓*	✓*	✓*	✓*
Numbness		✓*	✓*	✓*	✓*
Pain evoked by light touching	✓*	✓*		✓*	✓*
Painful cold or freezing pain			✓*	✓*	
Pain evoked by mild pressure		✓			
Pain evoked by heat or cold		✓			
Pain evoked by changes in weather				✓	
Pain limited to joints‡					✓
Itching			✓		
Temporal patterns		✓			
Body map – spatial distribution of pain		✓			
Autonomic changes	✓				
Signs					
Brushallodynia	✓*	✓*			
Raised soft touch threshold		✓			
Raised pin prick threshold	✓*	✓*			

*Highlight items shared by two or more tools; †Screening tools that involve clinical examination of neuropathic pain signs; ‡Used to identify non-neuropathic pain; Adapted from Bennett et al

every clinician must acquire in order to make individuals pain-free and giving them the quality of life they deserve.

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