Postoperative Pain Management After Total Knee Arthroplasty

An Essay

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5HT 5-hydroxytryptamine

CGRP ..... Clacitonin gene-related peptide

CPGS ..... Chronic pain grade scale

DHN..... Dorsal horn neuons

GABA..... Gamma aminobutyricacid

IT..... Intrathecal

MPQ..... Mcgill pain questionnaire

NE..... Nor-epinephrine

NMDA..... N-MethyL-D-aspartate receptor

NRS..... Numeric rating scale

PCA..... Patient controlled analgesia

SF-36BPS ..... Short form 36 bodily pain scale

SP..... Substance P

TKA..... Total knee arthroplasty

TRPA1 ..... Transient receptor potential subfamily A member 1

TRPM8..... Transient receptor potential subfamily M member 8

TRPV1 ..... Transient receptor potential cation subfamily V member 1

VAS ..... Visual analog scale

Abstract

The routine use of peripheral nerve blocks and wound infiltration with long-acting local anesthetics as an adjuvant to local, regional and general anesthetic techniques can improve postoperative pain management after a wide variety of surgical procedures.

Multimodal analgesia which is achieved by combining different analgesics that act by different mechanisms and at different sites in the nervous system, resulting in additive or synergistic analgesia with lowered adverse effects of sole administration of individual analgesics, is needed for acute postoperative pain management due to adverse effects of opioid analgesics, which can impede recovery; Yet, the literature on multimodal analgesia often shows variable degrees of success, even with studies utilizing the same adjuvant medication.

Keywords: Nor-epinephrine- Chronic pain grade scale - Dorsal horn neuons- Gamma aminobutyricacid- Clacitonin gene-related peptide

INTRODUCTION

Pain is a vital function of the nervous system in providing the body with a warning of potential or actual injury. It is the most common symptom of any illness and is defined as unpleasant sensory and emotional experience associated with either actual or potential tissue damage. Pain is classified according to the arbitrary interval of time from onset into acute and chronic pain**,** pain that resolves quickly is called acute (i.e postoperative pain) and pain that lasts a long time is called chronic ***(Bonica and International Association for the Study of Pain, 2015).***

Despite the increasing interest in postoperative pain management and development of pain control modalities, more than half of the patients who undergo surgery experience inappropriate level of postoperative pain. In particular, pain after orthopedic surgery has been considered especially difficult to manage. Approximately half of total knee arthroplasty (TKA) patients present with extreme pain immediately after surgery ***(Parvizi et al., 2009).***

The classic pain pathway has been described as a neuronal signal that begins in the periphery from an injury or a noxious event and is transmitted via nociceptive receptors through the spinal cord, then upward to the brain. Nociceptive sensory neurons transduce physical noxious energy into an electrical signal (i.e action potentials), which then transmits the

location and intensity of the noxious stimulus via the spinal cord to the brain ***(Costigan et al., 2009).***

After a surgical incision, inflammatory mediators released by damaged tissue trigger an inflammatory cascade. This inflammatory response reduces the threshold and increases the responsiveness of nociceptors (sensory receptors on C fibres and Adelta fibres) to subsequent input in the damaged tissue, a phenomenon known as peripheral sensitization. The body's neurophysiological response to any insult, including surgery, may initially serve a protective function (i.e., pain limits further use) and promote healing. It is now known that a similar sensitization processes can also occur more centrally as a result of the ''afferent barrage'' induced by activation of nociceptors in response to surgery ***(Costigan et al., 2009).***

Postoperative Pain is a subjective experience and patient experiencing it is the only person who can provide an accurate description of it. A patient self report of pain is therefore regarded as the 'gold standard' for pain assessment to implement appropriate postoperative pain management strategies ***(Vickers, 2010).***

Postoperative Pain should be assessed regularly and systematically, at rest and during movement, by competent nurses along with other vital signs and it should be documented as the 'fifth vital sign'. Assessments should consider Intensity

of pain, Location, Duration and Description ***(Böyökyilmaz and Asti, 2010).***

A comprehensive pain management strategy combines the followings as recommended modalities for the management of postoperative pain after Total Knee Arthroplasty: Patient education, administration of preemptive analgesics, neuro axial analgesia (epidural analgesia), peripheral nerve block (femoral nerve block), periarticular injection, patient controlled analgesia and oral analgesics ***(ASA, 2012).***

The principle of multimodal therapy is to use interventions that target several different steps of the pain pathway allowing agents to act synergistically while requiring lower total doses of each drug. This promotes more effective pain control with fewer associated side effects ***(Nuelle and Mann, 2007).***

AIM OF THE STUDY

The aim of this study is to provide a concise overview of the different postoperative pain management regimens after Total Knee Arthroplasty surgery aiming to minimize patient discomfort, facilitate early mobilization and functional recovery.

PHYSIOLOGY OF PAIN AFTER TKA

The most widely accepted definition of pain, was that coined in 1979 by the International Association for the Study of Pain, stating that it is an unpleasant sensory and emotional experience arising from actual or potential tissue damage. Though, later debated and many more arraying definitions were posed, yet fortunately there is a general agreement, that pain bears physical, psychosocial & psychological distress, to the unfortunate victim ***(Merskey and Bogduk, 2003).***

Pain is classified depending on:

1- Which region of the body is involved (superficial, deep, somatic or visceral).

2- Which system dysfunction is causing the pain (headache, back pain, sciatica).

3- Which etiological factor is precipitated (trauma, ischemia, cancer, neuralgia).

4- Which neurochemical modulation is it provoked (increase bradykinin, prostaglandins, Gamma aminobutyricacid (GABA) or decrease endorphins, Nor-epinephrine (NE), 5-hydroxytryptamine (5HT).

5- Which character pattern (aching, throbbing and burning) and intensity is presenting (sharp or dull aching).

6- Onset (rapid or insidious) and length of duration does it prevail (days, weeks, months).

(Raj, 2007)

Summing all these variables into a single classification seems impossible but simply pain can be divided into two main types ***(Basbaum et al., 2008):***

Acute pain

It is of sudden onset, transitory, lasting for hours to days (less than 30 days but can turn to sub-acute if it lasts up to 6 months) and resolves quickly once the underlying cause is cured (when the noxious stimulus is removed or the underlying damage or pathology has healed spontaneously or by treatment). It is of a clear cause, most often nociceptive (i.e. resulting from injury or inflammation of somatic or visceral tissue) ***(Lavand'homme, 2011)***.

Pain in this case, is considered as a protective response to injury, hence it is beneficial to the patient because it indexes that there is something wrong and motivates the person to get help and without this sensation the individual will ignore his illness resulting in complications and even death. Accordingly, the therapeutic objective is focused to treat the underlying cause ***(Lavand'homme, 2011).***

Chronic pain

It is of insidious onset, long standing, usually exceeding more than six months duration, yet can last for years or forever. It can either start as acute pain and continues beyond the normal time expected for resolution or recurs for various other

reasons or can even arise in absence of any detectable stimuli, damage or disease. It may be nociceptive or neuropathic (i.e. initiated or maintained by a primary lesion or dysfunction along the nervous system) ***(Chen et al., 2013)***.

Pain in this case, is considered as a maladaptive response to injury; hence it is not therapeutically beneficial to the patient. Accordingly, the therapeutic objective emphasis upon reducing the pain intensity to give relief, limit disability and improve function ***(Gerbershagen, 2011).***

Pain perception:

In order that a noxious stimulus transforms into a painful perception, a group of highly specialized structures are set into a sequential cascade of functional changes ***(Zhu et al., 2010):***

a. Recognition of the stimulus:

When its intensity reaches certain threshold by a specialized sensing structure NOCI CEPTORS. These are bare sensory peripheral nerve endings of primary afferent neurons that are networking throughout all organs and tissues (except brain).

They possess specific biophysical and molecular properties for differential coding of noxious sub modalities depending on the differential expression of selective channels that confer sensitivity to heat as Transient receptor potential subfamily A member 1 (TRPV1), cold as Transient receptor potential subfamily M member 8 (TRPM8), acidic milieu (as ASICs), and a host of chemical irritants as Transient receptor