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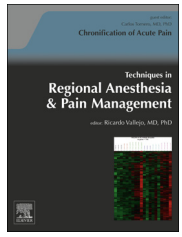

Preoperative risks factors in postoperative pain (or persistent postoperative pain)

Jean-Pierre Estebe, MD, PhD

Department of Anesthesiology, Intensive Care, and Pain, Medicine of University Hospital of Rennes, Rennes, France

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ABSTRACT

The management of postoperative pain should not only focus on the surgery procedure. Taking into account of many parameters that can change the course of the perioperative pain; an early preoperative anesthesia management should allow to improve various protocols. Some factors can be greatly improved during the preoperative period; others parameters can be modified by the protocol during or after the surgery.

Introduction

A significant number of patients report ongoing pain after surgery. Since the first description from Kehlet et al,[1] several studies have reported the prevalence of chronic pain (CP) after surgery at 3–24 months approximately 20% or more.[2,3] A number of observational studies have investigated the risk factors associated with the presence of CP or persistent (or prolonged) postoperative pain (PPP).[4-7] These phenomenons exert an enormous effect on the quality of life with most economic burden.

Preoperative risk factors

In modern anesthesia and analgesia, it is critically important for recognizing patients at risk of PPP and identifying potentially modifiable risk factors. As pain is a multifaceted and highly personal experience, various factors were now well-known as predictors of PPP intensity or analgesic consumption. Systematic review had recently reported variables that were commonly associated with a greater risk of PPP.[8] Theses factors could modify per and postoperative analgesic protocol for the same surgery. However, there is a lack of comprehensive framework for epidemiological studies to improve the knowledge about etiology and prognosis of PPP.

Identification of the predictive factors for PPP: The nonmodifiable factors

Gender

There were conflicting findings regarding correlation between gender and PPP.[9] This is partially because of the variations of hormonal status (distinct phases of menstrual cycle vs pre and postmenopausal women or pregnancy, or hormonal treatment).[10] Female patients were found to have more PPP. Usually, women report clinical pain to be more intense than men, with greater frequency of painful episodes presenting at more body sites.[11] Conversely, placebo analgesia effect is more often observed in male than in female subjects,[12,13] may be because of gender role (or stereotypes as gender schema theory that learned masculinity encourages stoicism). The effect of childhood or adult molestation (sexual abuse) could not be neglected.

Age

Although it is still discussed, it was commonly found to have negative correlation with both PPP and analgesic

E-mail address: jean-pierre.estebe@chu-rennes.fr

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consumption.[8,14] This negative correlation suggested that the younger the pain, the more PPP or analgesic requirement for the same surgical procedure.[8]

Comorbidities

Different comorbidities could harder the analgesic treatment.[2] If it remains discussed, patients with high resting blood pressure seems to have lower PPP.[15,16] Cardiovascular response to pain was not extensively studied. However, pulse oximeter, a noninvasive technique, used to assess oxygen saturation, could be used to monitor the sympathetic response to noxious stimuli (perfusion index calculated from pulse oximeter waveforms) that could be particularly interesting with patient with decreased awareness or impaired cognitive function.[17]

Obesity

The prevalence of CP is comparable with the prevalence of obesity. Recent and large studies showed the increase risk of CP prevalence with the increase of body mass index.[18] The increasing prevalence of pain is not only for rheumatologic pain (low back pain and arthritis) but also for neuropathic pain, fibromyalgia, and headaches. There is of course some interaction between diabetes, CP, and obesity. Over-weight could interfere particularly for orthopedic surgery like knee or spine surgery.[2] Diabetic patients had a high risk of pre, per, and postoperative neuropathic pain.[19]

On the other hand, malnutrition or cachexia as observed in cancer is also a clinical presentation for neuropathic pain. The risk of neuropathic pain could be particularly increased when adjuvant chemotherapy was introduced before surgery.

Genetic factors

There is a growing interest in the concept of genetic variability accounting for some of the variability in response to painful stimuli (ie, abnormality in the voltage-gated sodium channel Na(v)1.7).[20,21] Pharmacogenetics refers to the genetic differences between the individuals act. At each level pharmacokinetics and pharmacodynamics (PKC and PKD) of opioids could be modified. In the liver, the cytochrome P450 (particularly CYP2D6 and CYP3A4) plays an important role with 4 general phenotypes: extensive, intermediate, poor, and ultra-rapid metabolizer. Cathecol-o-Methyltransferase gene may contribute to the variability of opioid consumption. P-glycoprotein (P-gp), highly expressed in endothelial cells, is an efflux transporter which influences opioid transport particularly through the intestine (oral route) and the blood-brain barrier. The activity of P-gp is affected by genetic variability (ABCB1-MDR1 is a highly polymorphic gene). Other drug administrations also affect these enzymatic activities. The OPMR1 encodes the opioid receptor μ (MOR) with various variants. Particularly the A118 polymorphism (variant A/A, A/G, and G/G) could modulate the opioid analgesia. Compared with A/A homozygous or heterozygote, patients with homozygous G/G could obtain least pain relief. MOR interacts with each others (ie, δ-opioid receptor) to form heteromeric complexes that affect opioid signaling.

Previous surgery

Re-operation at the same site[22-24] or at the other side but at the same level[25] significantly increased the risk of PPP; not only because of difficulties during the surgery.[25,26]

Identification of the predictive factors for PPP: Modifiable factors

The experience of pain in humans is multidimensional phenomenon manifesting sensory-discriminative, cognitive-evaluation, and affective-motivational components. All these factors must be highlighted, in the aim to introduce a specific preoperative treatment. If in physiology (ie, patient free-pain), unconditioned response to stress and condition stress-induced analgesia are currently evaluated in controlled protocol; in physiopathology (ie, CP patient), various mechanisms can be attenuated or reversed in case of central sensitization.[27]

Preoperative pain

Preoperative pain can be divided into 3 subcategories: preoperative pain or analgesic experience, patient's perception regarding pain or analgesia, and pain threshold.

Preoperative pain: higher level of preoperative pain (as referred by surgery) has frequently been identified as a risk factor for postsurgical pain across a range of surgery types,[2,28] if the proportion of neuropathic pain before surgery is low.[29] There is a clear correlation with the increase risk of acute PPP and PPP.

Pain thresholds or pain tolerance: were correlated with PPP or analgesia.[8] Various tests could be used (heat pain test, cold pressor test, and suprathreshold pain stimulation). The neuropathic pain must be evaluated by the DNA scale.[30] Some simple questionnaires were proposed as preevaluation test.[31]

Other pain sites: greater number of pain sites had correlation with PPP.[2] On the one hand, in physiology, repetitive painful stimulation (or sensitization) in healthy subjects were reported to decrease the pain perception over time and cause habituation.[32-35] On the other hand, if some use the counter stimuli for pain relief (ie, proprioceptive feedback), results of habituation remain discussed in physiopathology situation. It was reported that social observational learning (meaning previous observational situation) could have a nocebo hyperalgesia effect.[35] However, as described earlier, in most of the cases, PPP are coexistent with other CPs (ie, lumbar back pain or shoulder pain, and so on).[32]

Preoperative analgesic drugs consumption is a marker of CP history.[36] The last decade, it was described the opioids-induced hyperalgesia phenomenon (or opioid tolerance, hyperalgesia induced by opioids). The acute opioid tolerance was first clearly described for remifentanil but it was now recently described for all the intraoperative opioids use.[37]

Chronic preoperative opioid-tolerant patients are also a major problem of the management of the PPP.[38,39] It is particularly true if patient had other addict behaviors, such as smoking, alcohol consumption, recreational drug use, and polysubstance abuse like benzodiazepine consumption,[40,41] or self-perceived risk of addiction.[42]

Psychosocial factors predict the trajectory of acute and CP or analgesic consumption.[43,44] Various tests are commonly