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Persisted Management of Opiods and Chronic Pain

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Abstract

The term opioid refers to all compounds that bind to opiate receptors. Conventionally, the term opiate can be used to describe those opioids that are alkaloids, derived from the opium poppy; these include morphine and codeine.

Keywords: Opiates; Receptors; Enforcement; Lesion; Nociceptive pain

Introduction

Opioids include semi-synthetic opiates, drugs that are synthesized from naturally occurring opiates, as well as synthetic opioids such as methadone, fentanyl, and propoxyphene. The term narcotic is a legal designation and should not be used in the clinical setting; it refers to opioids and a few other drugs that are grouped with the opioids by law enforcement. In the United States, numerous opioids have been commercialized for oral, transdermal and intravenous administration [1]. Oral and transdermal formulations are usually administered for pain in the ambulatory setting. These include combination products, such as those containing hydrocodone and acetaminophen or ibuprofen, tramadol and acetaminophen, oxycodone and acetaminophen or aspirin, and those containing codeine and acetaminophen or aspirin [2]. The single entity formulations on the market include those containing morphine, and methadone. Opioids act by binding to specific proteins, called opioid receptors. Receptors are widely distributed. Those involved in pain modulation are situated in both the central nervous system and the peripheral nervous system. These receptors also bind endogenous opioid peptides, which are involved in pain modulation and numerous other functions in the body. Among these functions are those mediated by deep structures of the brain, which are involved in the modulation of reinforcement and reward mechanisms, mood and stress [3]. Opioid receptors are also found on cells from the immune system. In studies with rats, activation of these receptors with morphine is associated with varied effects, including sensitization of afferent nerves to noxious stimuli. When an opioid given for pain binds to receptors, analgesia may be accompanied by any of a diverse array of side effects related to the activation of receptors involved in other functions. These may include effects mediated by peripheral or by peripheral and central mechanisms, such as reduced peristalsis and itch, or primary central nervous system effects, such as miosis, somnolence, mental clouding, and respiratory depression. Central mechanisms also lead to changes associated with hyperalgesia and decreased responsiveness to opioids and it has been speculated that opioid-induced hyperalgesia may be a clinically relevant phenomenon leading to increased pain in some situations [4].

Discussion

Activation of other central nervous system pathways by opioids also may produce mood effects, either dysphoria or euphoria. Presumably, binding to those receptors involved in reinforcement and reward also occurs whenever an opioid is taken. In most individuals, when opioids are taken to treat pain, there appears to be no overt effect from change in these systems. In some cases, however, powerful reinforcement occurs, expressed as efforts to repeat the administration and these reinforcing outcomes may be associated with craving and with positive mood effects such as euphorigenic or pleasurable effects. These outcomes, which are uncommon but potentially serious when they occur, can occur in the presence or absence of pain [5]. Although these effects could be associated with iatrogenic addiction, they appear to be rare in patients who do not have risk factors suggesting the existence of the biological substrate for opioid-induced craving. Although several types of opioid receptors exist, opioid drugs largely produce their analgesic and reinforcing effects via activation of the mu opioid receptor; thus, opioids used for pain are often described as, mu agonists. Mu drugs that have the ability to fully activate opioid receptors are referred to as opioid agonists or full mu agonists. Those opioids that occupy, but do not activate, receptors are referred to as opioid antagonists, they can reverse the effects of mu opioid agonists [6]. Those opioids that either have a low intrinsic. or are agonists at another receptor and antagonists at the mu receptor are called agonist-antagonist drugs. Those with a low intrinsic activity are called partial opioid agonists and are characterized by a ceiling on most agonist activity, such that increases in dose will increase the drug's physiological and subjective effects only to a certain level and further dose increases produce no additional effects [7]. These differences in mu receptor interactions are clearly related to the clinical use of opioid drugs and their abuse liability. Agonist-antagonist drugs are less attractive than pure mu agonists to individuals with addiction and no pain. Although other biochemical and molecular processes are presumably relevant to variation in these effects, relatively little is known about the interactions among these processes in humans. The clinical use of opioid drugs is influenced by a variety of other characteristics, including pharmacokinetics. With the notable exception of methadone and buprenorphine, most opioids have relatively short half-lives and this has necessitated the development of new delivery systems designed to provide prolonged effects and a longer dosing interval. Clinically-relevant physical dependence and tolerance may occur with short term or long-term use of an opioid compound, particularly a pure mu agonist. These phenomena, which vary greatly in the clinical setting, represent neuro-adaptation processes. The neurophysiology of

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physical dependence and tolerance are closely related to each other and to the phenomenon of opioid-induced hyperalgesia [8]. The possibility that opioid administration, particularly at relatively high doses, may lead to increased pain has contributed to the controversy about opioid therapy for non-cancer pain, notwithstanding the limited evidence that this phenomenon occurs in clinical settings. Brief Overview of Chronic Pain Chronic pain has been described as pain that has persisted for at least 1 month following the usual healing time of an acute injury, pain that occurs in association with a non-healing lesion, or pain that recurs frequently over a period of months. In most clinical and research reports, chronic pain is typically defined as pain that has persisted for at least 3 months. The prevalence of chronic pain in the general population is believed to be quite high, although published reports have varied greatly. Cautious cross-national estimates of chronic pain range would represent millions of Americans. A national survey in the US, estimated that the prevalence among adults of moderate to severe non-cancer chronic pain was 9%. A large survey of general populations across several European countries reported the prevalence for chronic painful physical conditions. Chronic pain is a highly complex phenomenon, which may or may not be primarily driven by tissue injury. Conventionally, the most common forms of chronic pain are divided into those labelled nociceptive, or pain caused by on-going stimulation of pain receptors, and those labelled neuropathic, or pain presumed to be related to damage to or dysfunction of the peripheral or central nervous system. These categories of pain simplify a complex reality in which both acute and chronic pain are induced by multiple peripheral and central mechanisms, which continually interact with each other and with numerous pain modulating systems [9]. The perturbations that ultimately results in pain perception are caused by neurophysiological processes and other related systems. For example, recent evidence has begun to highlight the role of neuro-immune activation following a tissue injury as an important mechanism in the development of chronic pain. The role of cytokines and other inflammatory mediators is obvious in inflammatory nociceptive pains, such as some types of arthritis, but new data suggest an equally salient role in the development of chronic neuropathic pain associated with central sensitization of neural pathways following peripheral injury . All chronic pain is profoundly influenced by psychological processing and responses. Pain severity and pain-related functional impairment are often found to be associated with psychological and social factors, and patients with identical diseases associated with pain, such as degenerative disk disease, may vary greatly in their reports of pain severity and pain behaviours. There is an extensive literature documenting the importance of operant conditioning factors and cognitive behaviour factors in the maintenance of chronic pain behaviours. Chronic pain also is influenced by psychosocial and psychiatric disturbances, such as cultural influences, social support, comorbid mood disorder, and drug abuse. Classic studies of pain behaviour indicate that cultural differences in the beliefs and attitudes towards pain and the social. The contribution of psychological, social and psychiatric factors should not lead to the conclusion that a pain syndrome is primarily psychogenic. Pain related exclusively or primarily to psychological factors occurs, but is far less prevalent than pain associated with organic processes that are powerfully influenced by psychosocial mediators and psychiatric comorbidities [10]. The pattern of suffering or the pain-related disability that often occurs in concert with persistent pain commonly touches on all domains of function. Patients with chronic pain may demonstrate pain-related interference with ability to perform usual activities at home, work, or school; maladaptive or dysfunctional behaviours, social isolation, and poor sleep patterns; and frequent health care utilization. The recognition that acute pain can compromises health has led major medical associations and accreditation committees to designate pain severity as a fifth vital sign, along with blood pressure, temperature, heart rate, and respiration.

Conclusion

Further recognition of the increased interest in the assessment and management of pain is underscored by the U.S. Federal Law that declared the first decade of the 21st century as the Decade of Pain Control and Research

Acknowledgement

None

Conflict of Interest

None

References

1. Manchikanti L, Fellows B, Singh V (2002) Understanding psychological aspects of chronic pain in interventional pain management. Pain Physician US 5: 57-82.
2. Manchikanti L, Hirsch JA, Smith HS (2008) Evidence-based medicine, systematic reviews, and guidelines in interventional pain management: Part 2: Randomized controlled trials. Pain Physician US 11: 717-773.
3. Birnesser H, Oberbaum M, Klein P, Weiser M (2004)The Homeopathic Preparation Traumeel® S Compared With NSAIDs For Symptomatic Treatment Of Epicondylitis. J Musculoskelet Res EU 8: 119-128.
4. Ozgoli G, Goli M, Moattar F (2009) Comparison of effects of ginger, mefenamic acid, and ibuprofen on pain in women with primary dysmenorrhea. J Altern Complement Med US 15: 129-132.
5. Raeder J, Dahl V (2009) Clinical application of glucocorticoids, antineuropathics, and other analgesic adjuvants for acute pain management. CUP UK: 398-731.
6. Maroon JC, Bost JW, Borden MK, Lorenz KM, Ross NA, et al. (2006) Natural anti-inflammatory agents for pain relief in athletes. Neurosurg Focus US 21: 1-13.
7. Bliddal H, Rosetzsky A, Schlichting P, Weidner MS, Andersen LA, et al. (2000) A randomized, placebo-controlled, cross-over study of ginger extracts and ibuprofen in osteoarthritis. Osteoarthr Cartil EU 8: 9-12.
8. Zundert JV (2007) Clinical research in interventional pain management techniques: the clinician's point of view. Pain Practice US 7: 221-229.
9. Demir IE, Tieftrunk E, Maak M, Friess H, Ceyhan GO (2011) Pain mechanisms in chronic pancreatitis: of a master and his fire**.** Langenbecks Arch Surg EU 396: 151-160**.**
10. Imani F, Rahimzadeh P (2012) Interventional pain management according to evidence-based medicine. Anesth Pain Med UK 1: 235-236.