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Adverse Effects of Pain Medication Used in Sport Medicine – A Narrative Review

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Abstract

Background: Acute and chronic pain is a very common problem amongst people involved in sports, whether professionally or recreationally. Most of them require the use of medication in order to alleviate the pain. Extensive use of pain medication is burdened with an increased risk of adverse effects.

Aim: The aim of this narrative review is to summarise the adverse effects of the most popular pain management medication currently in use in sport medicine.

Material and methods: A comprehensive literature review was conducted via the advanced research tool in PubMed in order to find eligible and relevant articles regarding pain medication and their adverse effects.

Results: Extensive intake of NSAIDs in high doses increases the risk of gastrointestinal bleeding. The greatest concern regarding the use of paracetamol is its hepatotoxicity. Antidepressants are characterised by a wide variety of adverse effects, such as hypertension, dry mouth, constipation, and an increased risk of falls. The most common adverse effects of gabapentinoids are somnolence, fatigue, and dizziness.

Conclusions: Most injuries experienced by athletes can be managed with NSAIDs, acetaminophen, and weak oral opioids prescribed for a short period of time, whereas chronic pain treatment should start with TCAs, SNRIs, or gabapentinoids. Prolonged intake of pain medication increases the risk of the occurrence of serious adverse effects. Physicians ought to monitor their patients and act accordingly should any adverse effects occur.

Keywords: analgesia, adverse effects, sport medicine, acute pain, chronic pain, pharmacotherapy

AI statement: Artificial intelligence tools have not been used to produce or alter the scientific content of the paper.

1. Introduction

The most accurate, up-to-date definition of pain has recently been revised in the International Association for the Study of Pain's (IASP) narrative review [1]. Pain is defined as an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage. According to the International Classification of Diseases 11th Revision (ICD-11) [2], pain can be defined as chronic if it persists or recurs for longer than 3 months.

Pain with a duration of less than 3 months is defined as acute. [3] Pain can also be classified based on its pathophysiological provenance. Firstly, nociceptive pain arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors. [4] Secondly, neuropathic pain is caused by a lesion or disease of the somatosensory nervous system. [4] Finally, nociplastic pain arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain. [4]

People who engage in strenuous physical exercise, whether professionally or recreationally, are at an increased risk of experiencing pain in the long term. The scale of this problem has been evaluated in numerous articles. For general pain, studies in collegiate and high school athletes report current pain prevalence rates of 26% to 53%. [5][6] In elite athletes, the lifetime prevalence of back pain is reported at 88.5%, with a 12-month prevalence of 81.1% and point prevalence of 49%. [7] In retired Olympians, 40% report current joint pain, with lumbar spine pain present in 19.3%. [8] Chronic pain in athletes is most commonly reported in the lumbar spine, shoulder, hip, knee, and ankle. [9][10]. The presence of musculoskeletal pain is associated with factors such as previous pain episodes, high training volume, and years of exposure to sport. [7][11][12]

Given the high prevalence of chronic pain amongst athletes, it comes as no surprise that alleviating pain requires extensive pain medication intake in this population. In collegiate athletes in the United States, 46% of female athletes and 38% of male athletes who reported pain are currently taking non-steroidal anti-inflammatory drugs (NSAIDs) for pain management. [9] Up to 69% of professional football players report using medication to reduce pain, with more than half using NSAIDs prior to matches. [13]

The scale of pain medication use amongst athletes is alarming, albeit understandable, given the fact that pharmacotherapy is a crucial element of pain therapy, providing relatively quick symptom relief in people experiencing pain. Unfortunately, extensive use of pain medication is burdened with an increased risk of adverse effects. Our goal is to provide a review of adverse effects of the most popular pain management medication currently in use in sport medicine.

2. Research methods

A comprehensive literature review was conducted via the advanced research tool in PubMed. Table 1 represents the queries used to find the appropriate articles. In order to identify additional eligible studies, reference lists from relevant reviews, guidelines, and trials were reviewed.

The authors reviewed articles referring to the definition of chronic and acute pain, mechanism of action, and adverse effects of pain medication, including nonsteroidal anti-inflammatory drugs, opioids, antidepressants, and gabapentinoids.

Query number	Query	Description
#1	"pain"[Text Word] OR "acute pain"[Text Word] OR "chronic pain"[Text Word]	The query was used to find articles regarding pain, acute pain, and chronic pain.
#2	"definition"[Text Word]	The query was used to find articles definitions for later use.
#3	"prevalence"[Text Word]	The query was used to find articles regarding prevalence for later use.
#4	"athletes"[Text Word] OR "sport"[Text Word]	The query was used to find articles regarding athletes.
#5	"adverse effects"[Text Word] OR "side effects"[Text Word] OR "adverse reaction"[Text Word]	The query was used to find articles regarding adverse effects.
#7	"pain management"[Text Word] OR "analgesia"[Text Word] OR "pain therapy"[Text Word]	The query was used to find articles regarding pain management, analgesia, and therapy of pain.
#8	"mechanism of action"[Text Word] OR "pharmacodynamics"[Text Word]	The query was used to find articles regarding mechanisms of action and pharmacodynamics.
#9	"nsaids"[Text Word] OR "nonsteroidal anti-inflammatory drugs"[Text Word] OR "ibuprofen"[Text Word] OR "diclofenac"[Text Word] OR "naproxen"[Text Word] OR "acetylsalicylic acid"[Text Word] OR "aspirin"[Text Word]	The query was used to find articles regarding nonsteroidal anti-inflammatory drugs.
#10	"acetaminophen"[Text Word] OR "paracetamol"[Text Word]	The query was used to find articles regarding acetaminophen.
#11	"opioids"[Text Word] OR "fentanyl"[Text Word] OR "tramadol"[Text Word] OR "morphine"[Text Word] OR "buprenorphine"[Text Word]	The query was used to find articles regarding opioids.
#12	"tricyclic antidepressants"[Text Word] OR "TCA"[Text Word] OR "TCAs"[Text Word]	The query was used to find articles regarding tricyclic antidepressants.

#13	"selective norepinephrine reuptake inhibitor"[Text Word] OR "selective norepinephrine reuptake inhibitors"[Text Word] OR "SNRI"[Text Word] OR "SNRIs"[Text Word]	The query was used to find articles regarding selective norepinephrine reuptake inhibitors.
#14	"selective serotonin reuptake inhibitor"[Text Word] OR "selective serotonin reuptake inhibitors"[Text Word] OR "SSRI"[Text Word] OR "SSRIs"[Text Word]	The query was used to find articles regarding selective serotonin reuptake inhibitors.
#15	"gabapentinoids"[Text Word] OR "gabapentin"[Text Word] OR "pregabalin"[Text Word]	The query was used to find articles regarding gabapentinoids.
#16	#1 AND #2	The query was used to find articles containing the definitions of acute and chronic pain.
#17	#1 AND #3 AND #4	The query was used to find articles regarding the prevalence of acute and chronic pain in athletes.
#18	#1 AND #3 AND #4 AND #7	The query was used to find articles regarding the use of pain medication among athletes.
#19	#7 AND #8 AND #9	The query was used to find articles regarding the mechanism of action of non-steroidal anti-inflammatory drugs.
#20	#5 AND #7 AND #9	The query was used to find articles regarding the adverse effects of non-steroidal anti-inflammatory drugs.
#21	#7 AND #8 AND #10	The query was used to find articles regarding the mechanism of action of acetaminophen.
#22	#5 AND #7 AND #10	The query was used to find articles regarding the adverse effects of acetaminophen.
#23	#7 AND #8 AND #11	The query was used to find articles regarding the

		mechanism of action of opioids.
#24	#5 AND #7 AND #11	The query was used to find articles regarding the adverse effects of opioids.
#25	#7 AND #8 AND #12	The query was used to find articles regarding the mechanism of action of tricyclic antidepressants.
#26	#5 AND #7 AND #12	The query was used to find articles regarding the adverse effects of tricyclic antidepressants.
#27	#7 AND #8 AND #13	The query was used to find articles regarding the mechanism of action of selective norepinephrine reuptake inhibitors.
#28	#5 AND #7 AND #13	The query was used to find articles regarding the adverse effects of selective norepinephrine reuptake inhibitors.
#29	#7 AND #8 AND #14	The query was used to find articles regarding the mechanism of action of selective serotonin reuptake inhibitors.
#30	#5 AND #7 AND #14	The query was used to find articles regarding the adverse effects of selective serotonin reuptake inhibitors.
#31	#7 AND #8 AND #14	The query was used to find articles regarding the mechanism of action of gabapentinoids.
#32	#5 AND #7 AND #14	The query was used to find articles regarding the adverse effects of gabapentinoids.
Table 1: Search strategy and keywords used for reviewing relevant literature.		

3. Results

3.1 Oral nonsteroidal anti-inflammatory drugs (Oral NSAIDs)

The mechanism of action of NSAIDs consists of inhibiting the cyclooxygenase-1 (COX-1) and the cyclooxygenase-2 (COX-2) isoenzymes. COX mediates the production of prostaglandins and thromboxane A₂ from arachidonic acid, which play a crucial role in triggering the inflammatory cascade. COX-1 is expressed constitutively and is involved in maintenance of the gastric mucosal barrier, platelet aggregation, and renal blood flow. COX-2 on the other hand, is inducible and mostly mediates pain and inflammation. Non-selective NSAIDs such as diclofenac, ibuprofen, or naproxen reversibly inhibit both COX-1 and COX-2, whereas selective COX-2 inhibitors such as celecoxib or meloxicam inhibit COX-2 preferentially, thus decreasing the risk of gastrointestinal bleeding. [14] In contrary to other NSAIDs, acetylsalicylic acid (aspirin) blocks COX-1 irreversibly. [15]

The efficacy of oral NSAIDs in rapid pain management has been meticulously researched. Numerous randomized controlled trials report reduced pain within 2 hours of treatment. Moreover, oral NSAIDs are considered to be among the most effective treatments in reducing pain at 1 to 7 days. [16]

The most common adverse effects of oral NSAIDs eventuate directly from their mechanism of action. Non-selective COX inhibitors are associated with dyspepsia (10-20%) and complicated ulcers (1-4%), which may lead to gastrointestinal bleeding. These adverse effects usually increase in a dose-dependent manner. [17][18] In terms of COX-2 inhibitors, one particular adverse effect of celecoxib raises safety concerns in a certain group of patients. Patients with prior myocardial infarction are at an increased cardiovascular risk when exposed to high doses of celecoxib. [19]

Interestingly, oral NSAIDs administration has been proven to affect the body's response to resistance training. A study conducted by Lilja et al. [20] evaluated the influence of ibuprofen and acetylsalicylic acid on muscle mass and strength. The results showed that muscle volume and adaptive response to resistance training were attenuated in patients taking maximal over-the-counter doses of ibuprofen.

3.2 Topical NSAIDs

The quality of evidence in terms of the effectiveness of topical NSAIDs in the form of gel or cream is equivocal. On the one hand, many studies emphasise their effect on physical function improvement and pain relief. [16] On the other hand, there are studies which cast doubt on that matter. The benefit of diclofenac gel or solution and ketoprofen gel barely reached statistical

significance when evaluated. [21] Interestingly, one study showed that the placebo carrier gel was better than the carrier gel with ketoprofen. [22]

3.3 Acetaminophen (paracetamol)

The analgesic mechanisms of acetaminophen (paracetamol) have not been fully understood. More recent research [23] indicates that acetaminophen's metabolite acts on receptors located in the midbrain and medulla, which act as co-localised mediators of pain modulation.

The efficacy of acetaminophen is reflected by its widespread use. It is considered to be amongst the most effective treatment options in reducing pain at 1 to 7 days. [16] Paracetamol alone or in addition to NSAIDs provides good outcomes in adult patients with musculoskeletal injuries in the acute setting. [24] Moreover, paracetamol intake has been proven useful in decreasing neuromuscular fatigue in football players. [25]

In terms of acetaminophen's adverse effects, its hepatotoxicity remains the greatest concern. It has been the leading cause of acute liver failure in the United States since 1998. [26] Paracetamol overdose has been associated with an increased risk of chronic liver failure. [27]

3.4 Opioids

The analgesic effect of opioids is achieved via binding to μ , δ , or κ opioid receptors, which are located in the central and peripheral nervous system. This mechanism leads to inhibition of nociceptive neurotransmission and modulation of pain perception. [28]

The effectiveness of opioids notwithstanding, their burden of increased risk for harms cannot be underestimated. [29] The most frequent adverse effect associated with long-term opioid therapy is constipation, occurring in up to 95% of patients receiving opioids for chronic pain. In those cases, prophylactic treatment is essential to minimise this effect. [30] If constipation occurs in patients taking opioids orally, the treatment can be modified by administering opioids such as fentanyl via transdermal patches. [31]

Another adverse effect attributed to opioid therapy is nausea and vomiting, occurring in approximately 15% to 30% of patients. [32] Cognitive impairment or somnolence may occur at the start of opioid therapy or during dose escalation. These effects are usually temporary and can be reduced by the use of a low starting dose and progressive up-titration. [33]

An uncommon, albeit life-threatening complication of opioid use is respiratory depression. Opioids activate μ receptors located on key neurons within the brainstem respiratory network, which leads to hyperpolarization of respiratory neurons, thus suppressing neuronal excitability and disrupting the generation of the inspiratory rhythm. [34] This particular adverse effect is due to the fact that tolerance to opioids develops rapidly. [35]

The risk of developing addiction to opioid medications in patients who are being treated for chronic non-cancer pain is highly variable, depending on patient selection, diagnostic criteria, and study methodology. The most recent meta-analysis [36] found that the pooled prevalence of opioid dependence and opioid use disorder in chronic non-cancer patients treated with opioids is 9.3% (95% CI = 5.7–14.8%; $I^2 = 99.9\%$). Physical dependence is defined by the occurrence of a withdrawal syndrome following abrupt dose reduction of the opioid or the administration of an opioid antagonist. [32] Withdrawal syndrome may manifest itself as a plethora of symptoms, including anxiety, abdominal cramps, or chills. Although it should be noted that the signs of dependence are not always indicative of addiction. [37] The wide variety of adverse effects results in a substantial discontinuation rate of 22.9% for oral opioids and 12.1% for transdermal opioids. [38]

3.5 Antidepressants

3.5.1 Tricyclic Antidepressants (TCAs)

The group of tricyclic antidepressants has a long history of use in the treatment of pain not related to cancer. TCAs such as amitriptyline or nortriptyline inhibit the reuptake of both serotonin and norepinephrine by blocking their respective transporters. However, they antagonise cholinergic and histaminic receptors, which is the primary cause for the anticholinergic, antihistaminic, and cardiovascular side effects of TCAs. [39] Side-effect profiles of TCAs include arrhythmias, hypertension, and postural hypotension, the last of which may increase the risk of falls and is especially true for amitriptyline. [40] Drugs that are less likely to increase the risk of falls are nortriptyline, imipramine, and desipramine. [26] Despite this fact, currently available evidence supports use of TCAs, especially in neuropathic pain. [41]

3.5.2 Selective Norepinephrine Reuptake Inhibitors (SNRIs)

Selective norepinephrine reuptake inhibitors have found use in pain treatment as well. SNRIs inhibit both the serotonin transporter and the norepinephrine transporter, blocking reuptake of serotonin and norepinephrine into presynaptic neurons. Most commonly used SNRIs are duloxetine, venlafaxine, and milnacipran. Unlike TCAs, SNRIs do not affect muscarinic, histaminic, or alpha-adrenergic receptors. [42] However, noradrenergic stimulation induces such adverse symptoms as sweating, dry mouth, constipation, and high blood pressure. [43]

3.5.3 Selective Serotonin Reuptake Inhibitors (SSRIs)

The effectiveness of selective serotonin reuptake inhibitors in pain management is rather limited. SSRIs act by selectively inhibiting the serotonin transporter, therefore blocking the reuptake of serotonin from the synaptic cleft into the presynaptic neuron. This increases extracellular serotonin levels and enhances serotonergic neurotransmission in the central nervous system.

[44] The most widely used SSRIs include fluoxetine, escitalopram, and sertraline. SSRIs are considered to be less effective in the treatment of pain, allegedly due to their lack of action on noradrenaline reuptake. [45] Moreover, the latest research highlights the metabolic changes induced by SSRIs, such as weight gain or dyslipidemia. [46]

3.6 Gabapentinoids

Gabapentinoids exert their analgesic effect in pain management by binding to the $\alpha 2\delta$ -1 subunit of voltage-gated calcium channels on presynaptic neurons in the central nervous system, particularly in the spinal dorsal horn. This binding reduces calcium influx into nerve terminals, leading to decreased release of excitatory neurotransmitters such as glutamate and norepinephrine, thereby dampening neuronal excitability and synaptic transmission in pain pathways. [47]

Most commonly reported adverse effects of gabapentoid use are fatigue, dizziness, somnolence, and weight gain. [40] For instance, gabapentin at doses of 1200 mg daily or more may be responsible for the occurrence of drowsiness, somnolence, or sedation in up to 14% of its users. The exact same portion of patients may experience ataxia or gait disturbance. [48] Pregabalin is considered to have a generally similar adverse effect profile to gabapentin, with the exception of euphoria, which is mostly associated with pregabalin. [49] This particular side effect creates a recreational misuse potential, which has been widely discussed in medical literature. [50]

4. Discussion

Pharmacotherapy is an essential part of pain management. Contemporary pharmacology offers a vast array of options for the therapy of pain, although each of the available options comes with specific shortcomings.

Numerous studies have assessed the efficacy of different combinations of pharmaceuticals and their adverse effects in types of pain most commonly encountered in sport medicine. In terms of short pain relief within 2 hours of treatment, topical NSAIDs, oral NSAIDs, acetaminophen alone, and acetaminophen with diclofenac are considered to be generally effective. Interestingly, transbuccal fentanyl may also appear as a considerable solution, inasmuch as when compared to placebo, it has turned out to be even more effective than the aforementioned non-opioid medications. [16]

In spite of that, oral NSAIDs are responsible for the occurrence of adverse effects within the gastrointestinal system, especially when administered frequently in high doses. Nonetheless, they are still less harmful than opioids alone or opioids combined with acetaminophen. Moreover, placebo-controlled studies have provided high-quality evidence that opioids such as tramadol and fentanyl alone or together with acetaminophen are frequently responsible for

neurologic adverse effects. [16] Thus, physicians ought to proceed with utmost caution in administering opioid medication. For most injuries, wherein pain cannot be alleviated by NSAIDs or acetaminophen alone, a three day prescription of opioids should be sufficient. [51] In terms of chronic, especially neuropathic pain treatment, most guidelines suggest using TCAs, SNRIs, and pregabalin or gabapentin as first-line drugs. Despite their numerous adverse effects, they are still considered to be fairly tolerable and safe. The second line of drugs includes tramadol, capsaicin, and lidocaine patches. Finally, third-line drugs consist of strong opioids. [41]

5. Conclusions

Pharmacotherapy is a crucial tool in pain medication. Most injuries experienced by athletes can be managed with oral NSAIDs, topical NSAIDs, acetaminophen, and oral or transbuccal opioids prescribed for a short period of time. Prolonged intake of the aforementioned medication, especially in high doses, may lead to the development of serious adverse effects. Treatment of chronic pain should be conducted in accordance with recent guidelines, which include drugs such as TCAs, SNRIs, and gabapentinoids, primarily used for other indications. Physicians ought to monitor their patients and act accordingly should any adverse effects occur.

Supplementary Materials

Not applicable.

Author Contributions

Mateusz Kubicki – conceptualisation, methodology, formal analysis, writing – review and editing, supervision

Patryk Hebda – investigation, resources, formal analysis

Roman Cemaga – investigation, formal analysis, project administration

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Adam Wolski – formal analysis, resources

Maria Król – investigation, resources, writing

Paweł Pustuła – resources, writing, rough preparation

Ewa Szplit – writing, investigation

Mieszko Czapliński – writing, investigation

Wiktoria Michnowska – rough preparation, visualisation

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Conflicts of Interest

The authors declares no conflict of interest in relation to this study.

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