Introduction

Pain is defined as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage”; however, it is largely subjective and varies from patient to patient. It is reported that more than 80% of people experience back pain at some point in their lives. In South Africa, back pain is the sixth most common complaint in the primary healthcare setting. It is therefore not surprising that back pain is the sixth leading cause contributing to the global disease burden, when one considers disease burden with regard to disability-adjusted life years. However, back pain is as a symptom, caused by various factors, and not a disease itself, which explains the limited and ineffective prevention strategies compiled to date.

Prevalence of back pain

Low back pain is a major public health problem affecting men and women from high-income, middle-income and low-income countries. The Global Burden of Disease Study identified back pain as the leading cause of disability globally. Certain studies found that back pain is most prevalent in ages 30 to 69 years and stated that women are more often affected than men. However, in Africa, the gender pattern may be reversed in lower-income countries where back pain is most reported amongst men. It is speculated that this may be due to the African culture, where men perform intensive physical labour and where there may be under-reporting of back pain by women due to possible gender inequalities. Maher, Underwood and Buchbinder concluded that high-income countries have more reported cases of back pain compared to middle- and low-income countries. Contrarily, there is an inverse correlation between the prevalence of back pain and patients’ educational status. There is also a logical connection with occupational factors, where the prevalence of back pain is increased in positions with high physical demands.

Cost and burden of back pain

Back pain is reported as the major reason for occupational absenteeism, decreased work productivity and early retirement globally, which results in considerable economic burden to individuals and the wider public. To put the issue into perspective: back pain is the chronic condition responsible for more people forced out of the workforce than any other.”
of the workplace than cardiovascular disease, diabetes, cancer and respiratory conditions combined. Direct and indirect cost implications for back pain, including healthcare expenditure and disability insurance, contribute to the strain on global healthcare costs. In the United States, lower back pain results in 34 million appointments with family physicians and primary care internists annually, leading to a total of 50 billion dollars in direct costs. The prediction for the coming decades is that disability and economic impact related to back pain will increase especially in low-income and middle-income countries (e.g. Asia, Africa and the Middle East), as health and social systems are not well established and are too ill-equipped to handle this burden along with the growing prevalence of infectious diseases.

**Pathophysiology of back pain**

Anatomically, back pain is described as pain localised from below the shoulder blades up to the gluteal folds and can involve muscles, ligaments, facet joints, spinal nerve roots, vertebral periosteum, intervertebral discs and blood vessels. Pain localised around the lumbosacral vertebra and iliac crests (from the twelfth rib to the coccyx) is known as lower back pain and is the most common form of back pain. There are five lumbar vertebrae located in the lower back, separated by intervertebral discs and the foramina, which accommodate the spinal nerves, radicular blood vessels and the sinuvertebral nerves. The spine has weight-bearing and shock-absorbing functions and is responsible for the protection of the spinal cord and nerve roots. The ligaments, facet joints and muscles surrounding the spine control balance, flexibility and stability. Back pain can arise from mechanical disorders of the spine (e.g. disc protrusion or spinal stenosis), non-mechanical diseases of the spine (e.g. cancer or infection) or referred pain from urogenital or gastrointestinal diseases. The ligaments of the intervertebral disc complex, facet joints and paravertebral musculature are supplied with nociceptive fibres, i.e. pain receptors. It is important to distinguish between pain in the spine itself and muscular pain, i.e. myofascial pain, as treatment strategies need to focus on the presumed source of the pain.

Acute pain acts as a physiological warning by the body of the presence of disease or a harmful condition. Further to this, acute lower back pain generally arises from increased tension in the muscles surrounding the spine with resulting tearing, either of the muscle fibres themselves or of the tendons between the muscles and bone. Back pain that originates in the musculoskeletal system can be described in three categories:

- **Muscle spasm**: Muscle spasm is considered a protective measure in an untrained or poorly conditioned muscle, which becomes inflamed due to persistent overuse. The surrounding muscles contract to prevent further movement in the inflamed area, which worsens the pain. Studies concluded that muscle spasm can also be caused by ischaemia in a specific muscle rather than muscle tension.

- **Myofascial pain**: Myofascial pain results from overuse of or damage to muscle fibres that are then in a shortened or contracted state. Myofascial pain is characterised by localised, palpable trigger points, which cause radiating, aching-type pain upon palpation.

- **Peripheral fatigue**: Peripheral fatigue is the result of mononeuronal firing or muscle glycogen depletion that leads to impairment of the neuromusculoskeletal function in the paravertebral muscles.

Acute back pain can result from ligament sprains as well, where the ligament is extended beyond its normal range. Acute pain that remains untreated or is treated ineffectively can result in the development of chronic pain. Acute low back pain becomes chronic when there is no relief for a period of more than three months. Chronic low back pain can be the result of serious disease or damage but is mostly caused by deterioration of the bony structures and ligaments in the spine, which is considered a normal process with advanced age. The facet joints undergo degenerative or inflammatory changes, which result in the enlargement of these joints and therefore facet-mediated arthritic pain. A chronic pain experience is not only physiological, but involves somatic, psychological and social factors, leading to pain, distress and disability in the individual and affects social, recreational and work life.

**Causes of back pain**

The vast majority of people who consult a medical professional for back pain in primary care settings are diagnosed with non-specific back pain, as there is no apparent specific nociceptive cause. Golob and Wipf stated that in 97% of patients who presented with back pain at primary care settings can be accurately diagnosed with a known pathological cause, which are generally serious conditions, such as malignancy, infection and vertebral fracture, and require targeted treatment. Outlined briefly below (Figure 1) is some of the most common pathological causes of back pain:

- **Infections**
- **Lumbar spinal stenosis**
- **Radicular pain/Radiculopathy**
- **Vertebral fractures**
- **Inflammatory disorders**
- **Spinal cord compression**
- **Malignancy**

**Figure 1: Causes of back pain**
Radicular pain/Radiculopathy – There is nerve-root involvement, most commonly at L5 and S1. Patients typically present with leg pain which is worse than their back pain and which worsens further during coughing, sneezing, straining and straight leg raise tests. The most common cause is disc herniation or degenerative changes with local inflammation.

Lumbar spinal stenosis – Patients experience back pain, which spreads into one or both legs, while walking or during extended periods of standing. Relief is experienced during rest or lumbar flexion (leaning forward). The most common causes are degenerative changes due to facet osteoarthritis, ligamentum flavum hypertrophy and bulging discs, where the foramina are narrowed.

Vertebral fractures – These fractures are mainly caused by osteoporosis. The probability of developing vertebral fractures increases with female gender, an age of 70 years or above, severe trauma and the chronic use of glucocorticoids.

Inflammatory disorders (e.g. axial spondyloarthritis) – Axial spondyloarthritis is a chronic inflammatory disease that most commonly develops in people aged 20 to 40 years. It is characterised by morning stiffness in the lower back, which improves with exercise but not with rest. These patients are commonly erroneously diagnosed with non-specific low back pain.

Malignancy – Evidently, back pain will be a major symptom of myeloma, but metastatic cancer also commonly targets bone. The most common cancers that spread to bone are adenocarcinomas, e.g. breast, lung, prostate, thyroid and gastrointestinal cancers. The most common symptom of metastasis to the spine is sudden severe pain, but also neurological symptoms due to spinal cord compression or spinal instability.

Infections – Spondylodiscitis, vertebral osteomyelitis, epidural abscess and even bacterial infections such as tuberculosis and brucellosis, may all result in back pain. A study that was done in Uganda highlighted the burden of infectious diseases in low-income countries, when back pain resulting from infections such as tuberculosis and brucellosis was found. Immunosuppressed patients and intravenous drug users have an increased risk of developing spinal infections. Fever and malaise that occur initially are not specific and can lead to serious consequences if left untreated.

Spinal cord compression (cauda equine syndrome) – Cord compression is most commonly caused by the herniation of an intervertebral disc, which mainly occurs during metastases. Other symptoms are motor weakness and bowel and/or bladder dysfunction.

Risk factors

Table I provides a checklist of risk factors that the healthcare worker can use to assess the likelihood of the development of back pain.

<table>
<thead>
<tr>
<th>Risk factor</th>
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<tr>
<td>Physically demanding jobs (manual handling, bending, twisting and whole-body vibration)</td>
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<td>Physical comorbidities, i.e. asthma, headache, diabetes</td>
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<td>Depression</td>
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<td>Smoking</td>
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<td>Obesity</td>
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<td>Lack of exercise</td>
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<td>Awkward postures</td>
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<td>Feeling tired</td>
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<td>Distractions during an activity</td>
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<td>Older age</td>
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<td>Female gender</td>
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<td>In-active/desk-bound work</td>
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<td>Psychologically arresting work</td>
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<td>Low educational status</td>
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<td>Workers’ compensation insurance</td>
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<td>Job dissatisfaction</td>
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<td>Insomnia</td>
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<tr>
<td>Anxiety/Stress</td>
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<td>Handling of objects far from the body</td>
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<td>Handling people or animals and unstable loading</td>
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<tr>
<td>A slip, trip or fall</td>
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<tr>
<td>Engagement in moderate or vigorous physical activity</td>
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<td>Sexual activity</td>
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<td>Alcohol consumption</td>
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Differential diagnosis of back pain

The American College of Physicians and the American Pain Society recommend that low back pain be divided into three categories: non-specific low back pain, low back pain with potential radicular symptoms, or low back pain due to an identified spinal cause, e.g. metastasis or infection. Differential diagnosis is initiated with a thorough physical examination and extensive history taking to determine which patients need to be referred for imaging and further diagnostic evaluation. The majority of patients who...
present with acute back pain can be treated with first-line non-pharmacological or pharmacological therapy and do not require any further evaluation. A number of “red flags” have been identified to identify patients who require further diagnostic evaluation, e.g. night pain, fever, a history of malignancy, unintentional weight loss, prolonged use of corticosteroids, severe trauma, pain occurring for an extended period of longer than four weeks, and the presence of contusion or abrasion.

**History taking**

History taking involves enquiries about the position, duration and severity of the back pain; whether the patient has suffered from back pain before and a comparison from the patient between the prior and the current back pain; as well as any event that may have triggered the onset of the pain. Unintentional weight loss, night fever or pain, changes in bowel and/or bladder function, or neurological symptoms such as weakness, numbness and disequilibrium also need to be determined. Bladder or bowel dysfunction may indicate cauda equina syndrome, which is a serious medical emergency. A detailed history of any failed treatment for the back pain, malignancy, bacterial infections, use of injectable drugs, corticosteroid use and back procedures should be recorded. The probability of spinal infection is increased with prior or current use of intravenous drugs, recent bacterial infections or if the patient has a fever. Mechanical back pain is generally located in the spine and may radiate to the gluteus, but does not spread into the legs, as is the case with radicular pain. An informal psychological evaluation to determine the presence of depression may be helpful, as psychological disorders are strong predictors of outcomes in terms of prolonged pain and disability.

**Physical examination**

A primary diagnosis cannot be the expected result of the physical examination, but the physical examination will contribute to the identification of factors that may suggest further evaluation. The patient’s back and posture must be examined to evaluate range of motion and to identify abnormalities such as scoliosis. The spine must be palpated to identify sensitive vertebræ or soft tissue, as well as the abdomen to evaluate referred pain from visceral organs, and the palpitation of potential areas of malignancy can also be included. The consultant must perform a neurological examination to evaluate reflexes, strength, sensation and stride. Suspected radiculopathy can be identified with a straight leg-raise test.

**Imaging**

Plain-film radiography, magnetic resonance imaging (MRI) and computed tomography (CT) are reserved for patients with a high possibility of infection, fracture or cauda equina, where it is necessary to determine the exact pathological reason for back pain to facilitate surgery or other interventions. Early and unjustifiable imaging do not result in improved outcomes as non-specific back pain generally improves within four to six weeks with or without treatment, but rather leads to unnecessary radiation exposure and increased patient anxiety, work absence, healthcare costs and utilisation of invasive procedures. Guidelines from the American College of Physicians, the American Pain Society, the National Institute for Health and Care Excellence in the United Kingdom and the “Choosing Wisely” campaign of the American Board of Internal Medicine suggest that imaging should not be performed routinely in patients with back pain, but should be reserved for patients with a suspected serious underlying condition.

**Management of back pain**

There is a plethora of either non-pharmacological or pharmacological treatment options in the management and treatment of back pain, especially lower back pain.

As illustrated in Figure 2, there are three key non-specific areas, which must be considered for the effective management of back pain.

![Figure 2: Key areas to consider in the management of back pain](image)

In the past few decades, emphasis on self-management, physical and psychological therapy has taken precedence over pharmacological treatment and surgery. Non-pharmacological methods are the first line of treatment and if unsuccessful, pharmacological treatments are used.

**Non-pharmacological management**

Exercise, complementary and alternative therapies (such as spinal manipulation, acupuncture, massage and mind-body interventions), psychological therapies (such as cognitive behavioural and operant therapy), physical techniques (traction, ultrasound, transcutaneous electrical nerve stimulation, low-level laser therapy, superficial heat or cold and back supports) are some of the non-invasive, non-pharmacological options available in the management of back pain.
**Exercise**

Exercise is a widely used treatment for back pain. A combination of general physical fitness, aerobic exercise, muscle strengthening and flexibility and stretching exercises are some of the interventions suggested in the treatment of back pain. Other exercises include traditional mind-body exercises e.g. Tai Chi and yoga which emphasise precise controlled movement and bodily awareness. Individual exercises seem to be more beneficial than group exercises in the treatment of back pain.

**Spinal manipulation**

This method provides modest short- and long-term relief of back pain, improves psychological well-being and increases functioning. High velocity thrust techniques such as mobilisation have been shown to be effective as reported by Maher et al.

**Acupuncture**

Acupuncture is a traditional Chinese-based therapy. To improve painful states, small solid needles are inserted into specific points in the body. An overview of systematic reviews done by Liu et al, showed that using acupuncture in isolation or as adjunct therapy provided short-term improvements in function and pain for chronic lower back pain.

**Pharmacological treatment**

In the treatment of lower back pain, the choice of a suitable drug should be based on the benefits, risks and costs.

**Non-opioid analgesics**

The following non-opioid related medicines are available for managing pain: paracetamol, and the nonsteroidal anti-inflammatory drugs (NSAIDs), for example naproxen, ibuprofen and mefenamic acid. They adequately treat mild pain and moderate-to-severe pain in combination with other medicines, particularly opioids, to provide more effective relief and reduce adverse effects.

**Paracetamol**

Paracetamol is one of the drugs of choice in pain management, due to its excellent safety profile and lack of any significant side-effects. It acts as a prodrug, with an active cannabinoid metabolite. In the brain and spinal cord, paracetamol follows deacetylation to its primary amine (p-aminophenol) which is conjugated with arachidonic acid to form N-arachidonoylphenolamide, a compound known as an endogenous cannabinoid. The involved enzyme is fatty acid amidase hydratase. N-arachidonoylphenolamide is an agonist at the Transient Receptor Potential Cation Channel, Subfamily V, Member 1 (TRPV1) receptors and an inhibitor of cellular anandamide uptake, which leads to increased levels of endogenous cannabinoids, inhibiting cyclooxygenases in the brain at concentrations that are probably not attainable with analgesic dosages of paracetamol. It is of interest to note that a cannabinoid-1 receptor antagonist, given at a dosage level that completely prevents the analgesic activity of a selective cannabinoid receptor agonist, completely inhibits the analgesic activity of paracetamol as well. This fact allows us to explain the mechanism of action of paracetamol in more detail. Despite this finding, however, the definite proof that the analgesic and antipyretic effects of paracetamol are dependent on COX-inhibition is still unclear. Hence, it works effectively when combined with codeine for more effective control of moderate-to-severe pain and discomfort.

Paracetamol is available orally, in several tablet and liquid formulations. The dosage, however, should be guided by the age and general condition of the patient.

Paracetamol was once recommended as first-line due to its low cost. However, the ineffectiveness of paracetamol in acute lower back pain, hepatotoxicity and the risk of asymptomatic elevation of aminotransferase levels at dosages of 4 g/d even in healthy adults poses a potential for harm.

**Nonsteroidal anti-inflammatory drugs**

NSAIDs competitively inhibit the cyclooxygenase (COX) enzyme, the enzymes facilitate the bioconversion of arachidonic acid to inflammatory prostaglandins. This results in the blockade of prostaglandin synthesis and subsequently dampened inflammatory responses. COX-1 and 2 are isozymes that only vary genetically. NSAIDs have three pharmacologically preferred attributes, i.e. analgesic, anti-inflammatory and anti-pyretic activity. They generally have similar analgesic properties but selection is based on their receptor selectivity. COX-1 receptor activation produces gastric effects that mediate hypersecretion of gastric acid, thinning of the lumen and propagates the development of gastric ulcers. These medicines have various formulations.

Aspirin (S0) and paracetamol (with its scheduling status being dependent upon pack size and route of administration) are more freely available than the oral formulations of the NSAIDs. For the most part, the topical formulations of the NSAIDs are available as over-the-counter (OTC) consumer products as well. Oral NSAIDs have steadily been becoming more readily available as OTC products, where they have traditionally been S3 prescription drugs. In the OTC setting, the oral NSAIDs are typically only available in the lower dosage ranges for the product in question, and in limited supply, for use in the acute setting. Patients that seem to require more frequent or chronic treatment with a suitable NSAID, should be encouraged to seek medical advice and obtain a prescription from their doctor.

It is important to note that NSAIDs have ceiling analgesic effects but the COX-2 mediated anti-inflammatory effects are dose-dependant. COX-2 is not detected in most normal tissues, but its expression is rapidly induced by stimuli such as proinflammatory cytokines (IL-1b, TNFo), lipopolysaccharides, mitogens and oncogenes (phorbol esters), fibroblast growth factor, epidermal growth factor, luteinising hormone, LH) and fluid-electrolyte haemostasis, resulting in increased synthesis of PGs in inflamed and neoplastic tissues.
NSAIDs such as aspirin, ibuprofen, diclofenac, ketorolac and mefenamic acid, have analgesic and anti-inflammatory properties, which are useful in the management of pain.\(^{28}\)

Non-selective NSAIDs (ibuprofen) and COX-2 inhibitors such as celecoxib have an increased risk for myocardial infarction.\(^{23}\) Physicians must take cognisance of these risk factors and when a NSAID or a COX-2 inhibitor is prescribed, the lowest dose for the shortest duration should be considered.\(^{16}\) However, in patients over 45 years old who have a high risk of gastro-intestinal toxicity, a cost-effective proton pump inhibitor should be co-prescribed.\(^{29}\) Aspirin and naproxen are the safest NSAIDs in high-risk cardiovascular patients especially due to the anti-platelet properties of aspirin.\(^{30}\)

**Opioid analgesics**

Opioid analgesics will provide analgesia for moderate to severe pain, for the vast majority of patients and with a wide margin of safety. This group of analgesics includes the following examples: codeine, morphine, oxycodone, methadone, fentanyl and pethidine. Opioids can be divided into weak and strong opioids. Weak opioids are used alone or in combination with other analgesics, in management of moderate pain. Strong opioids are usually reserved for severe pain.\(^{1}\)

Tapentadol is a novel centrally acting synergistic analgesic with μ-opioid-agonist opioid and additional noradrenaline reuptake inhibition properties, which result in increased levels of extracellular norepinephrine. The benefits of combining both effects in a single molecule results in decreased potential of drug-drug interactions. By this action tapentadol may be useful particularly in neuropathic pain. This drug was approved by the American FDA November 2008 and its use in South Africa was approved in June 2018.\(^{31}\)

Adverse reactions experienced with tapentadol include those seen in patients taking opioids and sympathomimetic drugs, viz. anxiety, increased heart rate, respiratory depression, constipation, decreased appetite, drug withdrawal syndrome. Gradual dis-continuation of the drug is advised due to risk of withdrawal syndrome.\(^{31}\)

Opioids are derivatives of morphine and bind to opioid receptors.\(^{30}\) Their use is contentious and should be used judiciously in selected patients for a short duration and closely monitored due to the risks of addiction and respiratory depression.\(^{16,30}\) Tramadol, a weak opioid with some serotonin norepinephrine reuptake inhibitor (SNRI) activity provides short-term improvements in pain and function but long-term use data is lacking.\(^{19}\) Cautionary measures must be taken as serotonin syndrome has been shown to occur when combined with certain anti-depressants.\(^{30}\)

**Skeletal muscle relaxants**

Skeletal muscle relaxants are classified into two main categories namely, antispastic and antispasmodic medications. Antispastic medications (e.g. baclofen) act on the spinal cord or on the skeletal muscles themselves to improve muscle hypertonicity and involuntary spasms. Antispasmodic medications lessen muscle spasms through alterations in central nervous conduction. These agents are divided into benzodiazepines and nonbenzodiazepines.\(^{32,33}\)

A new skeletal muscle relaxant in South Africa is Myprocam®.\(^{33}\) The active ingredient is cyclobenzaprine, a nonbenzodiazepine antispasmic agent, which blocks nerve impulses recognised as pain. Cyclobenzaprine is structurally related to the tricyclic antidepressants, like amitriptyline and nortriptyline. It is categorised as a muscle relaxant with a mechanism of action not fully understood, but is thought to be an agonist of the

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**Table 3: The World Health Organization’s three-step analgesic ladder**

<table>
<thead>
<tr>
<th>Mild to moderate pain: Non-opioid analgesics</th>
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<tbody>
<tr>
<td>• Aspirin, paracetamol or ibuprofen</td>
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<tr>
<td>• With/without an adjuvant*</td>
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<table>
<thead>
<tr>
<th>Moderate pain: Weak opioid analgesics</th>
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<tbody>
<tr>
<td>• Codeine, tramadol or buprenorphine</td>
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<tr>
<td>• With/without a non-opioid, such as aspirin, paracetamol or ibuprofen</td>
</tr>
<tr>
<td>• With/without an adjuvant*</td>
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<table>
<thead>
<tr>
<th>Severe pain: Strong opioid analgesics</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Morphine, hydromorphone, oxycodone, buprenorphine or tapentadol</td>
</tr>
<tr>
<td>• With/without a non-opioid, such as aspirin, paracetamol or ibuprofen</td>
</tr>
<tr>
<td>• With/without an adjuvant*</td>
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</table>

*Examples of adjuvants include corticosteroids, antidepressants, hypnotics and anticonvulsants/antiepileptic agents
α2-receptor at the descending noradrenergic neurons within the supraspinal area of the brain stem. Some evidence also revealed serotonergic antagonism of the 5-HT2 receptors.33,34

Myprocam® is often combined with analgesics like ibuprofen or naproxen and is used in addition to rest and physical therapy for short-term relief of muscle spasm associated with acute, painful musculoskeletal conditions. The extended release cyclobenzaprine has shown to have superior efficacy to the placebo in the treatment of lower back pain and has lower frequency of adverse effects compared to other skeletal muscle relaxants.35 The recommended adult dose is a 15 mg capsule, taken once daily. Some patients may require up to 30 mg per day, administered as one Myprocam® 30 mg capsule, taken once daily, or as two Myprocam® 15 mg capsules, taken once daily.32,34

Side-effects include dizziness and drowsiness. Other anti-cholinergic effects such as dry mouth, blurred vision, constipation and urinary retention will be expected due to activity on cholinergic receptors. Cardiac arrhythmias like QTc prolongation is likely to arise and it should be used with caution in patients with a history of arrhythmias or who are using any medications prolonging the QTc interval. Myprocam® is contra-indicated in patients older than 65 years, or in patients with impaired liver function.33

Adequate evidence for the effectiveness of the prolonged use of Myprocam® is not available and therapy for longer periods of use is seldom warranted; the duration of use should therefore only be for short periods of not more than three weeks.33 Skeletal muscle relaxants are used as adjunctive treatment for lower back pain and not considered first line because of the adverse effects, such as sedation.30

A stepwise approach to pain management

The World Health Organization’s (WHO) ‘analgesic ladder’ (Figure 3) serves as the foundation for treatment and relief of pain together with rehabilitative and psychological modalities. This multidimensional approach offers the greatest potential for maximising analgesia and minimising adverse effects.36

Role of the pharmacist in pain management

Community pharmacists are key healthcare resources in chronic lower back pain management, yet their roles in patient care are often not fully utilised. Research suggests that failure to receive professional pharmaceutical advice can lead to inadequate chronic pain management, incorrect medication use and increased likelihood of experiencing adverse events from self-management. The provision of pharmacist-driven clinical interventions and professional health services from the community pharmacy can serve a simple, imperative and cost-effective contribution to improved health outcomes.37

The significant role of pharmacists in medication use is underlined given the probability that patients suffering from chronic pain are consuming multiple analgesic medications. Chronic pain management with multiple medications for synergistic effects may facilitate pain relief, however, this strategy highlights potential harm exposure. Since pharmacists are significant role players in profiling all possible medications contributing to polypharmacy, they can be actively involved in minimising dangerous exposure due to medication abuse and misuse.37

In addition to the pharmacist’s role in polypharmacy, pharmacists are also provided with a promising platform for interventions, since chronic pain patients tend to use non-prescription OTC drugs to self-manage and medicate pain. The availability of these OTC drugs from pharmacies often leads to purchasing of analgesic medicines without the necessary and valuable advice provided by pharmacists. Pharmacies are thus considered significant checkpoints whereby patient’s queries can be attended to, clinical interventions can be made if necessary, and the relevancy of non-prescription medications can be confirmed. This can be achieved by becoming aware of the factors that hinder patients from seeking appropriate advice and to develop a greater understanding of chronic pain management.37

Possible pharmacist-driven interventions will include: specialised education, medication review, consultation, self-management, appropriate referrals, provision of an action plan, and follow-up. Pharmacists can complete a paper-based medication assessment of each patient’s medical record. Patients can also be requested to keep a pain diary to guide interventions in follow-up consultations.37

A systematic review conducted by Bennett et al. (2011) suggested daily follow-up telephone consultations which include recommendations and advice with regard to dosage adjustments, drug information, and supportive counselling. Patients receiving these educational interventions experienced remarkable benefits in the reduction of adverse effects and pain intensity. The study further indicated that educational interventions by pharmacists had the most significant impact on patients suffering from chronic low back pain.37

Pharmacists providing these educational interventions to enhance patient understanding and knowledge by relaying behavioural instructions, information and advice associated with chronic pain management empower patients to manage their lower back pain more successfully.37

Conclusion

Back pain can be a debilitating condition that can affect productivity in the workplace. This commonly reported primary healthcare condition requires a thorough patient history to identify the cause and the risk factors for back pain. Risk factors such as depression, physically demanding jobs, insomnia and anxiety play a crucial part to the diagnosis and management of back pain. Non-pharmacological methods such as acupuncture, spinal manipulation and appropriate exercise are valuable first-line treatments and upon failure of these treatments, pharmacological management such as the use of NSAIDs, opioids and muscle relaxants should be used when warranted. The pharmacist can play a significant role in the effective management of these patients.
References