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Review Article

Choosing the Right Non-Steroidal Anti-Inflammatory Drug: A Critical Determinant for Analgesia in Osteoarthritis Management

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Abstract

Osteoarthritis (OA) management has evolved from focusing solely on symptomatic relief to a more patient-centered approach that addresses the underlying pathophysiology of the disease. This review examines the role of non-steroidal anti-inflammatory drugs (NSAIDs) in OA, with a particular focus on their effects on the synovial membrane. In OA, the synovium undergoes pathological changes due to inflammation, and diclofenac is known to concentrate in synovial fluid at higher levels than plasma. This accumulation is crucial, as the synovial fluid is the primary site for pain and inflammation relief, suggesting that higher absorption at this site results in longer-lasting pharmacological effects. Choosing the appropriate NSAID for OA requires balancing effective pain relief, improved patient outcomes, and enhanced quality of life while minimizing gastrointestinal, cardiac, and renal side effects.

Key words: Cyclooxygenase inhibitors, Inflammation, Non-steroidal anti-inflammatory drugs, Osteoarthritis

INTRODUCTION

Osteoarthritis (OA) is a degenerative joint disease characterized by cartilage breakdown, osteophyte formation, subchondral bone sclerosis, alterations in the joint capsule, and synovial membrane inflammation. OA is the most common type of arthritis globally and a major cause of disability in older adults.[1,2] According to The Global Burden of Disease Study 2019 report, the number of OA cases in India has significantly increased from approximately 23.46 million in 1990 to 62.35 million in 2019.[3] In OA, inflammation of the synovium leads to synovitis, which can be detected through imaging, arthroscopy, or histological methods. As a synovial membrane, housing metabolically active synoviocytes, supports joint health by nourishing chondrocytes through synovial fluid and clearing degradation products; thus, targeting synovitis treatment can be a game changer in the treatment and management of OA.[2]

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OA: Role of Synovitis

OA is associated with cartilage breakdown and synovial inflammation, which causes symptoms such as joint swelling, pain, and synovitis. Non-invasive imaging techniques such as magnetic resonance imaging and ultrasonography complement biopsy-based histological analysis for detecting synovitis. Inflammation of the synovial membrane triggers the release of pro-inflammatory mediators, which disrupts cartilage repair and accelerates degradation, resulting in a vicious cycle. Figure 1 illustrates the mechanism underlying synovial involvement in OA pathophysiology. This interplay highlights the potential of synovium-targeted therapies to relieve symptoms and slow OA progression.[2]

OA Management Guidelines

National institute for health and clinical excellence guidelines (NICE)

NICE guidelines recommend combining pharmacological treatments for OA with non-pharmacological approaches

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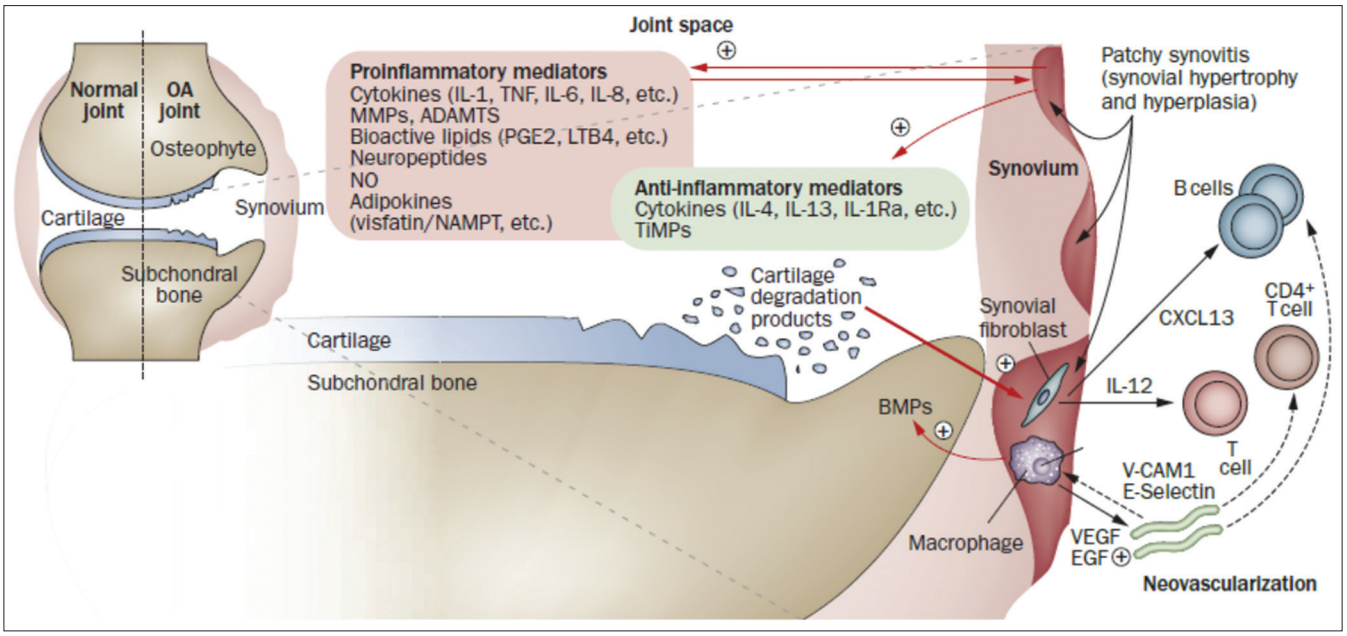

Figure 1: Involvement of the synovium in OA pathophysiology[2]

such as exercise, using the lowest effective dose for the shortest duration [Figure 2]. The risks and benefits should be carefully evaluated, especially for elderly patients and those with comorbidities.[4] Non-steroidal anti-inflammatory drugs (NSAIDs) are preferred for managing knee OA and may also be considered for other affected joints. Individual patient factors, including age, pregnancy status, existing medications and comorbidities, must also be considered. To reduce the likelihood of gastrointestinal complications, co-prescription of gastroprotective agents, such as proton pump inhibitors, is strongly recommended.[5]

Paracetamol and weak opioids are not routine treatments for OA and should only be used for occasional, short-term pain when other options fail.[5] If paracetamol does not provide adequate pain relief, oral NSAIDs or cyclooxygenase-2 (COX-2) inhibitors may be used at the lowest effective dose and for the shortest duration.[4]

American academy of orthopedic surgeons (AAOS) guideline

The AAOS recommends both non-selective and COX-2 selective oral NSAIDs as effective treatments for managing knee OA as they have been shown to significantly reduce pain and improve functional capacity in patients.[7] It also recommends the use of oral NSAIDs for the management of symptomatic hip OA, provided there are no contraindications.[8]

Evidence from clinical studies, moderate to high-quality, demonstrates significant improvements in pain relief and functional outcomes with NSAID therapy compared to placebo. Clinical studies by Baerwald *et al.* (2010), Kivitz *et al.* (2001), Makarowski *et al.* (2002), Schnitzer *et al.* (2011), and Svensson *et al.* (2006) found that oral NSAIDs reduced pain substantially, demonstrating their efficacy in treating hip OA. Despite their efficacy, the selection of an appropriate NSAID, along with careful consideration of the dosage and duration of treatment, should be tailored to the individual patient's needs, taking into account their comorbidities and overall health status.[7]

Choosing the Right Treatment is Critical: Balancing Efficacy and Safety

Choosing the appropriate treatment for OA is essential for achieving the best outcomes. NSAIDs are highly effective in managing inflammatory nociceptive pain by significantly reducing prostaglandin production. As a first-line option, their success depends on careful consideration of individual patient factors, ensuring maximum benefit and targeted relief.[1]

Since its introduction in 1973, diclofenac has undergone significant advancements to enhance its efficacy, safety, and convenience. Extended-release diclofenac sodium improves safety and enables once-daily dosing for chronic pain. Diclofenac potassium formulations offer faster absorption and quicker pain relief. These innovations, combined with new mechanisms of action, enhance its anti-inflammatory effects. Diclofenac remains a cornerstone in modern pain management.[9]

Are all NSAIDs equal?

NSAIDs inhibit COX, with COX-1 aiding gastric cytoprotection and platelet aggregation and COX-2 driving inflammatory response, vasoprotection, and renal blood

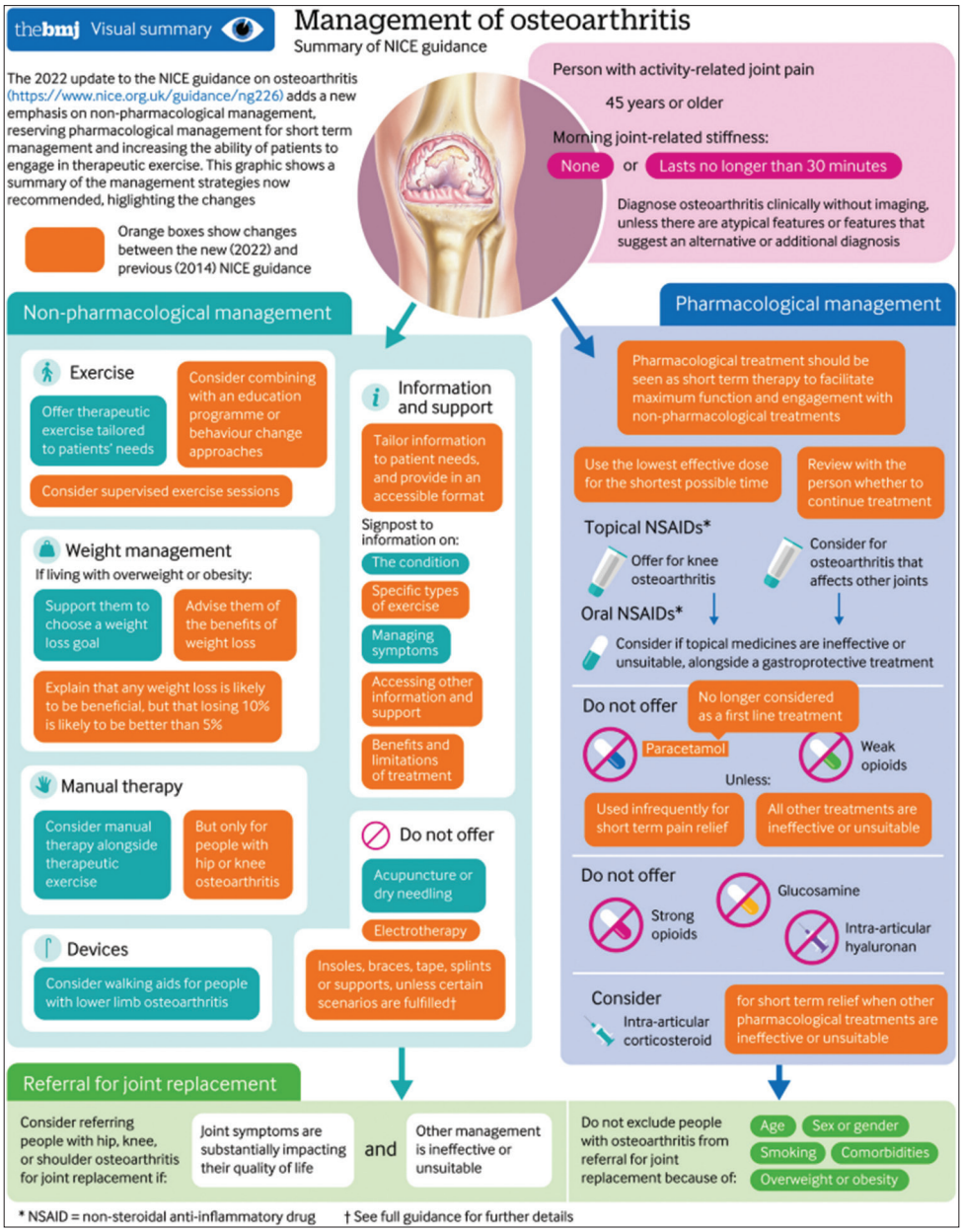

Figure 2: NICE guidelines on osteoarthritis management[6]

flow. They are classified as non-selective, COX-2 selective, or partially selective (diclofenac).[10] Not all NSAIDs exhibit the same ability to penetrate synovial fluid, which is a critical determinant of their therapeutic efficacy in conditions such as OA.[1] Diclofenac demonstrates superior synovial penetration, achieving concentrations 25% higher than aceclofenac[11,12] and 32% higher than etoricoxib,[12,13] with synovial fluid levels reaching approximately 81% of plasma levels compared to 57% for aceclofenac and 49% for etoricoxib.[11-13]

A study by Pandit *et al.* reported that diclofenac has a higher synovial fluid/plasma penetration ratio (81%) compared to aceclofenac (57%), with 24% greater synovial absorption. This superior penetration is associated with better efficacy in OA treatment, as higher synovial absorption extends the duration of its pharmacological effects.[14]

In an interesting meta-analysis by Da Costa *et al.*, involving 74 randomized trials with 58,556 patients, evaluated the efficacy of NSAIDs and paracetamol at specific daily doses (e.g., diclofenac 150 mg/day, etoricoxib 30–90 mg/day, rofecoxib 25–50 mg/day). The analysis identified diclofenac 150 mg/day as the most effective NSAID for improving both pain and function, with no role for single-agent paracetamol in OA treatment, regardless of dose.[15] In a study by Revel *et al.*, diclofenac showed superior pain relief and functional improvement compared to ibuprofen,

piroxicam, nimesulide, ketoprofen, salicylate, eltenac, etoricoxib and indomethacin, highlighting its effectiveness. These findings emphasize that while NSAIDs share similar mechanisms of action, their clinical efficacy can vary.[16]

Paracetamol and opioids lose preference in OA management

As per evidence from clinical studies, paracetamol in OA management is less effective for inflammatory pain due to its weak inhibition of COX-1 and negligible inhibition of COX-2, resulting in minimal impact on peripheral sensitization. Furthermore, in inflamed tissues, paracetamol may be inactivated by free radicals, nullifying any COX-2-related activity. Its analgesic effects are attributed to central mechanisms, primarily through activation of the endogenous cannabinoid system. When compared to NSAIDs, paracetamol demonstrates lower efficacy in treating inflammatory pain and has less central analgesic potency than opioids. Due to these limitations, NICE guidelines no longer recommend paracetamol and weak opioids as first-line treatments and discourage the routine use of strong opioids for pain management.[1,7,8]

Recent studies also suggest that in managing chronic pain, diclofenac has been shown to improve functional outcomes more effectively than opioids such as oxycodone and morphine, despite opioids achieving slightly greater pain score reductions. However, long-term efficacy data for opioid analgesics remain limited, contributing to their declining preference for OA treatment.[10]

Diclofenac: Best NSAID for Favourable Efficacy and Tolerability in OA

Diclofenac at a dosage of 150 mg/day has been shown to be the most effective NSAID for managing OA, providing superior improvements in pain relief and functional outcomes. Comparative studies indicate that diclofenac outperforms the maximum recommended doses of other commonly used NSAIDs, including ibuprofen, naproxen, and celecoxib. Its effectiveness makes it a preferred choice for patients requiring potent symptom management, highlighting its potential advantage over other NSAIDs in optimizing OA treatment.[15] Diclofenac sodium is commonly used in the long-term management of rheumatoid arthritis (RA), OA, and ankylosing spondylitis, where sustained symptom relief is essential. Maintaining patient compliance can be challenging due to the prolonged treatment duration that is generally required.[17]

Diclofenac's antiproliferative effect in synovial cells: Reduction in inflammation

Diclofenac undergoes significant first-pass metabolism, with approximately 60% of the active drug reaching systemic circulation.[9] Once ingested, diclofenac primarily exerts its anti-proliferative effects by activating peroxisome proliferator-activated receptor gamma (PPARγ) in synovial cells. PPARγ, a nuclear receptor, is essential for regulating cellular differentiation, metabolism, and inflammation. Upon activation by diclofenac, PPARγ inhibits the proliferation of synovial fibroblasts and promotes apoptosis, leading to a reduction in the number of inflammatory cells within the joint, thereby alleviating inflammation.[18]

Like other acidic NSAIDs, it binds strongly to plasma proteins, primarily albumin. Reduced protein binding in inflamed tissues with an acidic environment increases the free fraction of diclofenac, facilitating its intracellular action. Diclofenac also accumulates in synovial fluid, where its concentration surpasses plasma levels and remains detectable for extended periods of up to 11 h with a 50 mg enteric-coated tablet and 25 h with a 100 mg slow-release tablet, prolonging its therapeutic effects.[9]

The key actions of diclofenac in synovial cells include:[18]

Inhibition of cell proliferation: Diclofenac decreases cell viability in synovial fibroblasts, leading to reduced inflammation and joint damage.

Induction of apoptosis: The drug promotes programmed cell death in overactive synovial cells, which are often implicated in the pathogenesis of OA.

Reduction of inflammatory mediators: By activating PPARγ, diclofenac also inhibits pathways involving nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) and activator protein 1 (AP-1), further decreasing the expression of pro-inflammatory cytokines.

Diclofenac dosing: Role in reducing COX-1 side effects

Diclofenac is commonly dosed twice daily despite its short half-life of approximately 1–2 h, resulting in notable fluctuations in plasma concentrations. During peak levels, diclofenac inhibits both COX-1 and COX-2 enzymes, temporarily acting in a non-selective manner. However, as its plasma concentration decreases, diclofenac shifts toward greater COX-2 selectivity, reducing the potential for COX-1-related side effects while still providing effective anti-inflammatory action. This dynamic dosing approach helps to balance therapeutic efficacy with safety, minimizing adverse effects associated with COX-1 inhibition.[19]

Review of gastric, renal, and cardiac safety

According to the AAOS, selective COX-2 oral NSAIDs were initially developed to reduce gastrointestinal side effects compared to non-selective oral NSAIDs. However,

evidence from meta-analyses indicates that these selective agents have not demonstrated a significant reduction in gastrointestinal adverse events. This highlights the importance of evaluating both efficacy and safety when considering NSAIDs for OA management.[7] Wax matrix technology enhances diclofenac's formulation by minimizing stomach release, providing 24-h sustained pain relief, delivering precise doses, targeting intestinal absorption, and improving gastrointestinal tolerability.[20]

The NICE guidelines recommend considering an oral NSAID for individuals with OA if topical NSAIDs are ineffective or unsuitable. When selecting an oral NSAID, it is essential to prioritize options with a safer gastrointestinal profile to reduce potential adverse effects. Selective COX-2 inhibitors are associated with a lower risk of gastrointestinal complications compared to non-selective COX inhibitors, though this advantage has been somewhat overstated due to study designs that often involve daily maximum doses of ibuprofen, naproxen or diclofenac rather than the lowest effective dose taken as needed.[6] A study by Stillar *et al.* suggests that the addition of a PPI to NSAIDs such as (diclofenac, naproxen, and ibuprofen) reduces the relative risk of developing symptomatic ulcers to 37%.[19]

Hypertension and other cardiovascular risk factors are common in OA, affecting around 40% of individuals. The NHANES III survey found that 21% of 35-year-old adults have both OA and hypertension.[21] The MEDAL program, which involved 34,701 patients, compared the cardiovascular safety of etoricoxib (60 or 90 mg) and diclofenac (150 mg) over 18 months. Etoricoxib 90 mg showed higher rates of heart failure, edema-related treatment discontinuations, and hypertension-related discontinuations compared to diclofenac. These findings suggest that diclofenac is a safer option, particularly for hypertensive OA patients, with no cardiovascular safety advantage.[19]

For chronic kidney disease patients, diclofenac is a safe option when used at low doses for short durations, especially for those with an estimated glomerular filtration rate of >30 mL/min/1.73 m^2. It offers rapid pain relief, potent COX-2 inhibition, and a lower risk of renal side effects compared to other NSAIDs.[14]

Diclofenac Sustained Release: Extends Synovial Concentration and Efficacy for 25 h

The sustained-release formulation of diclofenac ensures prolonged therapeutic drug concentrations in synovial fluid, surpassing plasma levels and persisting for up to 25 h after plasma concentrations have significantly declined.[9] This extended-release mechanism maintains consistent drug delivery, enhancing efficacy while minimizing fluctuations in drug levels. Sustaining synovial concentration over a longer duration improves both treatment effectiveness and convenience, allowing for once-daily dosing and better patient compliance.[17]

A study by Altman *et al.* suggests that with the administration of 100 mg slow-release diclofenac sodium tablet can be detected in synovial fluid for up to 25 h. Studies have shown that diclofenac significantly reduces synovial fluid levels of prostaglandin E2 (PGE2) and interleukin-6, which are key inflammatory mediators.[9] Clinical studies confirm that diclofenac sodium enteric-coated tablets are effective in managing inflammation and pain associated with RA, OA, ankylosing spondylitis, and acute gout with substantial pain relief and functional improvement, as comparable to NSAIDs such as indomethacin, ibuprofen, and naproxen.[9]

Modified-release diclofenac formulations are designed to minimize gastric damage, thereby reducing adverse events and enhancing dosing convenience. The controlled release is achieved through a multilayer matrix structure comprising alternating hydroxypropyl methylcellulose layers and the active drug. The outer layer swells without eroding, acting as a barrier, while the inner layer gradually dissolves the drug over 8–10 h. This formulation reduces gastric exposure, reducing the risk of gastroduodenal irritation by using a pH-sensitive coating that prevents dissolution in the stomach, releasing the drug in the small intestine. Diclofenac reaches its maximum concentration within 0.5–1.5 h, with better patient compliance (≥90%) seen in OA patients receiving 75 mg twice daily. Gastrointestinal side effects, such as abdominal pain and indigestion, are common but consistent across all NSAIDs.[9]

CONCLUSION

In conclusion, targeting synovial inflammation/synovitis is a critical strategy for improving OA treatment and management outcomes. The NICE guidelines have revised their stance, no longer recommending paracetamol as a first-line treatment due to its limited effectiveness and avoiding strong opioids due to their potential risks. Since the discovery of diclofenac, it has undergone significant evolution, with the development of new formulations aimed at enhancing efficacy, safety, and patient convenience. Extended-release formulations enable once-daily dosing for chronic pain management, but diclofenac potassium has been shown to provide faster pain relief and improved functional outcomes. These advancements, coupled with its enhanced anti-inflammatory effects and improved dissolution and absorption kinetics of diclofenac potassium, ensure diclofenac's ongoing relevance in modern OA therapy by offering faster and more effective

pain relief. Diclofenac is preferred over aceclofenac and etoricoxib due to its superior adverse event profile.

EXPERT OPINION

1. Dr. Amit Kumar Agarwal
Indraprastha Apollo Hospital, New Delhi

In my clinical practice, I prefer using a standard NSAID or COX-2 inhibitor (excluding etoricoxib 60 mg) for OA management according to the guideline recommendations as paracetamol and weak opioids are not routinely effective. I frequently prescribe diclofenac for both acute and chronic pain due to its high efficacy, particularly in treating synovitis. The slow-release formulation offers sustained effects, reducing inflammatory markers. Diclofenac has a better gastroprotective profile and is more effective than generic NSAIDs. I also avoid prescribing oral narcotics due to their adverse effects.

2. Dr. Ashwani Kumar
Divya Prastha Hospital, New Delhi

In my clinical practice, I usually recommend a standard NSAID or COX-2 inhibitor (excluding etoricoxib 60 mg) for OA management, often prescribing diclofenac for both acute and chronic pain. Diclofenac effectively targets synovitis, reducing pain and inflammation. The slow-release formulation offers prolonged therapeutic effects with a high synovial fluid-to-plasma concentration ratio, resulting in improved outcomes. I prefer diclofenac over etoricoxib as etoricoxib is found to be a significant factor in raising blood pressure in patients with hypertension. I also avoid oral narcotics due to adverse effects and minimal pain relief.

3. Dr. Amit Kumar Mishra
Sharda Hospital, Greater Noida

I manage OA by using either a standard NSAID or COX-2 inhibitor (excluding etoricoxib 60 mg) as the first-line treatment. I avoid paracetamol, weak opioids, and strong opioids due to limited effectiveness and risk of adverse effects. Diclofenac is the preferred choice for acute and chronic pain, as it effectively reduces pain and improves physical function. Synovitis, a key factor in disease progression, can be managed with diclofenac due to its high synovial fluid-to-plasma ratio and prolonged therapeutic effects. I avoid oral narcotics due to their minimal pain relief and increased side effects.

4. Dr. Sanjay Kumar Gupta
SRMSIMS, Bareilly

In my opinion, I recommend either a standard NSAID or COX-2 inhibitor (excluding etoricoxib 60 mg) as the first-line treatment for OA, avoiding paracetamol and weak opioids. Diclofenac is my preferred choice due to its effectiveness in reducing pain and improving function, particularly in managing synovitis. The slow-release formulation offers prolonged effects and better gastroprotection. I believe diclofenac is more effective than generic NSAIDs. I also avoid etoricoxib in patients with hypertension as it demonstrates a higher risk of increasing blood pressure. Oral narcotics are not recommended due to their minimal pain relief and increased adverse effects.

5. Dr. Pradeep Tripathi
Ratnadeep Hospital, Kanpur

In my view, I prefer using a standard NSAID or COX-2 inhibitor (excluding etoricoxib 60 mg) as the first-line treatment. I avoid paracetamol, weak opioids, and strong opioids due to their limited effectiveness and side effects. I frequently prescribe diclofenac, as it significantly reduces pain, improves function, and is more effective than other NSAIDs. Synovitis plays a key role in disease progression. The slow-release formulation of diclofenac offers sustained relief and reduces inflammatory markers. It also provides better gastroprotection and is preferable over generic NSAIDs. I avoid etoricoxib in patients with hypertension because it poses higher risks for increasing blood pressure and I also avoid oral narcotics due to their minimal pain relief and adverse effects.

6. Dr. Ankur Singhal
Ramkrishna Care Hospital/Dr. Singhal Joint Clinic, Raipur

In my practice, I avoid paracetamol, weak opioids, and strong opioids due to limited effectiveness and side effects. I frequently prescribe diclofenac, which is 3–1,000 times more effective than other NSAIDs, which effectively reduces pain and improves physical function. Synovitis, a key factor in disease progression, is well-managed with diclofenac's slow-release formulation. It offers sustained therapeutic effects, better gastroprotection, and is preferable over generic NSAIDs. I avoid etoricoxib in patients with hypertension and do not recommend oral narcotics due to their increased adverse effects.

7. Dr. Pankaj Gupta
Skin and Bones Clinic, New Delhi

In my opinion, I frequently prescribe diclofenac for both acute and chronic OA pain due to its superior efficacy. It effectively reduces pain, improves physical function, and is 3–1,000 times more potent than other NSAIDs. Synovitis plays a key role in OA progression, contributing to increased pain and impaired function. With its high synovial fluid-to-plasma concentration ratio and slow-

release formulation, diclofenac offers sustained therapeutic effects for managing synovitis. The SR formulation allows once-daily dosing and provides comparable efficacy to controlled-release opioids, making it ideal for patients with chronic pain and relevant comorbidities.

8. Dr. Mukesh Tripathi
Savitri Devi Wellness Center, Satna

In my clinical practice, the first choice for OA management is either a standard NSAID or a COX-2 inhibitor, excluding etoricoxib 60 mg. Diclofenac is highly effective in reducing pain and improving physical function, particularly in managing synovitis, which contributes to increased pain and impaired function in patients with OA. Its high synovial fluid-to-plasma concentration ratio and enhanced gastroprotective profile make it a preferred option. Compared to etoricoxib, diclofenac has a lower risk of raising blood pressure in patients with hypertension. Non-selective NSAIDs carry a similar risk for gastrointestinal adverse events compared to selective COX-2 inhibitors, but opioids increase adverse effects without providing consistent pain relief.

9. Dr. Nitin Rathi
Shree Goverdhan Hospital, Betul

In my view, I recommend either a standard NSAID or a COX-2 inhibitor (excluding etoricoxib 60 mg) as the first-line treatment according to the guideline recommendations. I avoid paracetamol, weak opioids, and strong opioids due to their limited effectiveness and adverse effects. Diclofenac stands out in treating both acute and chronic pain, effectively reducing pain and improving physical function. It targets synovitis, which contributes to pain and disease progression. The sustained-release formulation provides prolonged therapeutic effects, enhances gastroprotection, and allows once-daily dosing, offering efficacy comparable to controlled-release opioids. I preferred diclofenac over etoricoxib in patients with hypertension to minimize blood pressure concerns.

10. Dr. Saswata Datta
The Arogyo, Burdwan

I don't commonly prescribe diclofenac for acute and chronic OA pain, but it is highly effective at relieving pain and boosting physical function. It is 3–1,000 times more effective than other NSAIDs. Targeting synovitis with slow-release diclofenac is beneficial. The slow-release formulation provides prolonged presence (~25 h), leading to sustained therapeutic effects. I agree that diclofenac SR is superior to generic NSAIDs. I avoid etoricoxib in patients with hypertension to lower blood pressure risks.

11. Dr. Pramod Shaligram Ingale
Manorama Clinic, Ranaghat

I typically choose either a standard NSAID or a COX-2 inhibitor, excluding etoricoxib 60 mg. Diclofenac is my preferred option for both acute and chronic pain due to its ability to effectively reduce pain, improve physical function, and is 3–1,000 times higher efficacious than other NSAIDs. Synovitis, a key factor in OA progression, can be managed with diclofenac's slow-release formulation, offering sustained effects. It also provides enhanced gastroprotection and comparable efficacy to controlled-release opioids. Unlike etoricoxib, diclofenac poses a lesser risk for elevated blood pressure in patients with hypertension.

12. Dr. Gagandeep Singh Sachdeva
Sohana Hospital, Mohali

In my practice, I avoid routinely prescribing paracetamol or weak opioids. Diclofenac is my go-to choice for both acute and chronic pain, as it is 3–1,000 times more effective than other NSAIDs. Synovitis plays a key role in OA progression, and diclofenac's slow-release formulation provides sustained therapeutic effects with once-daily dosing. I do not believe generic NSAIDs are less effective than diclofenac SR for long-term management. While patient comorbidities are important, I find diclofenac is less likely to raise blood pressure when compared to etoricoxib.

13. Dr. Atul Vishnoi
Krishna Orthopedic Center, Moradabad

I prefer using a standard NSAID or COX-2 inhibitor (excluding etoricoxib 60 mg) for managing OA, as paracetamol and weak opioids are generally ineffective. I commonly prescribe diclofenac for both acute and chronic pain, particularly for its effectiveness in treating synovitis. The slow-release formulation ensures prolonged therapeutic effects, helping to reduce inflammatory markers. Diclofenac offers better gastroprotection and is more effective than generic NSAIDs. I avoid oral narcotics due to their adverse effects and limited ability to offer pain relief.

14. Dr. Nihar Ranjan Padhi
Betul Fracture Hospital, Betul

In my clinical practice, I frequently prescribe diclofenac for both acute and chronic OA pain due to its effectiveness, especially in managing synovitis. Synovitis is a significant contributor to the onset and progression of OA. Diclofenac's efficacy is due to its high synovial fluid-to-plasma concentration ratio and the slow-release

formulation, providing sustained therapeutic effects for up to 25 h. It significantly reduces synovial fluid levels of PGE2 and inflammatory cytokines. While selecting an NSAID, considering patient comorbidities is essential to minimize adverse effects, including gastrointestinal risks.

15. Dr. Gaurav Nath Bhalla
Bhalla Medical Center, Kanpur

As per my practice, diclofenac plays a crucial role in managing OA by effectively reducing pain and enhancing physical function. It is 3–1,000 times more potent than other NSAIDs, especially when addressing synovitis, which exacerbates pain and functional limitations. The sustained-release formulation provides once-daily dosing, improved gastroprotection, and demonstrates comparable efficacy to controlled-release opioids. When diclofenac is used in combination with a proton pump inhibitor, it can reduce the risk of gastrointestinal side effects by about 40%. In contrast, etoricoxib may raise blood pressure in patients with hypertension. Non-selective NSAIDs are generally associated with more adverse effects and less consistent pain relief outcomes.

16. Dr. Argha Saha
Khoshbagan, Burdwan

In my practice, I prioritize the use of a standard NSAID or COX-2 inhibitor (excluding etoricoxib 60 mg) for managing OA, and avoid prescribing paracetamol or weak opioids. I frequently opt for diclofenac due to its strong effectiveness in alleviating pain and enhancing physical function, particularly in cases involving synovitis. Diclofenac's slow-release formulation ensures sustained effects and enhanced gastroprotection. It offers similar efficacy to controlled-release opioids but with fewer adverse effects. I agree that non-selective NSAIDs may carry a similar gastrointestinal risk compared to selective COX-2 inhibitors.

17. Dr. Soudip Sinha
Healthpoint, Rampurhat

In my clinical practice, I find that oral diclofenac's efficacy in managing synovitis is supported by its high synovial fluid-to-plasma concentration ratio, slow-release formulation, and significant reduction in inflammatory markers such as PGE2. The sustained-release formulation provides prolonged therapeutic effects, enhances gastroprotection, and offers once-daily dosing, making it convenient for patients with chronic pain. I avoid using oral narcotics for OA due to their notable increase in medication-related adverse effects and lack of consistent pain alleviation. Diclofenac offers a more effective, safer alternative for long-term management.

18. Dr. Shamik Chaudhuri
Bs Road near Natun Bazar, Coochbehar

I prioritize using a standard NSAID or COX-2 inhibitor (excluding etoricoxib 60 mg) according to the guideline recommendations for OA management, as it effectively reduces pain. Synovitis, a key factor in OA progression, can be managed with treatments that target its inflammatory components. I find diclofenac effective in treating chronic pain due to its high synovial fluid-to-plasma concentration ratio and once-daily dosing. Unlike etoricoxib, which can elevate blood pressure in patients with hypertension, diclofenac poses no such risk and offers consistent pain relief without significant side effects.

19. Dr. Himanshu Agrahari
Agrahari Orthopedic Center, Gorakhpur

In my clinical practice, I prefer using a standard NSAID or COX-2 inhibitor (excluding etoricoxib 60 mg) to manage OA, as it effectively reduces pain and improves physical function. Diclofenac stands out for its efficacy, being 3–1,000 times more effective than other NSAIDs. Targeting synovitis can improve outcomes as synovitis is closely associated with pain and functional impairment. The sustained-release formulation offers prolonged therapeutic effects with once-daily dosing, a better gastroprotective profile, and comparable efficacy to controlled-release opioids, without the elevated blood pressure risks seen with etoricoxib.

20. Dr. Soumya Raj Banerjee
Bardhaman Apollo Hospital, Burdwan

I frequently prescribe diclofenac for managing acute and chronic OA pain, as it effectively reduces pain and improves physical function. I believe targeting synovitis is the key to improved outcomes, as it significantly contributes to both pain and structural progression of OA. The sustained-release formulation of diclofenac offers benefits such as enhanced gastroprotection, once-daily dosing, and comparable efficacy to controlled-release opioids. I observed a few gastrointestinal side effects when diclofenac is used alongside a proton-pump inhibitor and prefer to avoid oral narcotics because of their adverse effects and inconsistent pain relief.

21. Dr. Nirmalya Deb
Park Clinic, Kolkata

In my clinical practice, I prescribe a standard NSAID or COX-2 inhibitor (excluding etoricoxib 60 mg) according to the guideline recommendations for OA management, avoiding paracetamol or weak opioids. Diclofenac stands

out due to its high efficacy in reducing pain and improving function, especially in synovitis, which contributes to OA progression. The sustained-release formulation offers advantages such as enhanced gastroprotection, once-daily dosing, and comparable efficacy to controlled-release opioids. I also believed that combining diclofenac with a proton-pump inhibitor reduces gastrointestinal side effects by about 40%, improving patient tolerance.

22. Dr. Premesh Kumar Sarkar
New CMR, Kolkata

In my view, I prefer using a standard NSAID or COX-2 inhibitor (excluding etoricoxib 60 mg) for managing OA, as per the guideline recommendations. Diclofenac is particularly effective in alleviating pain and improving physical function, especially in cases of synovitis, which accelerates OA progression. Its sustained-release formulation offers benefits such as enhanced gastroprotection, once-daily dosing and similar efficacy to controlled-release opioids. I also find that combining diclofenac with a proton-pump inhibitor reduces gastrointestinal side effects by approximately 40%, enhancing patient comfort and compliance.

23. Parkash Singh
Parkash Hospital, Amritsar

Diclofenac is particularly effective in reducing pain and improving physical function, especially in synovitis, which contributes to OA progression. The sustained-release formulation offers benefits such as enhanced gastroprotection, once-daily dosing, and comparable efficacy to controlled-release opioids. I also believe that combining diclofenac with a proton-pump inhibitor can reduce gastrointestinal side effects by about 40%, improving patient compliance. I avoid using oral narcotics for the management of OA due to significant adverse effects.

24. Dr. Shubhang Aggarwal
NHS Hospital, Jalandhar

In my clinical practice, I prefer prescribing standard NSAIDs or COX-2 inhibitors (excluding etoricoxib 60 mg) as per the current guideline recommendations for OA management, with diclofenac being my preferred choice due to its high efficacy in managing both acute and chronic pain. Synovitis is a key factor in OA progression, contributing to pain and impaired physical function. Diclofenac's slow-release formulation offers prolonged therapeutic effects, reduces inflammation and enhances gastroprotection. I recommend combining diclofenac with a proton-pump inhibitor to reduce gastrointestinal side effects by about 40%. I avoid oral narcotics due to their increased risk of adverse effects and limited pain relief.

25. Dr. Harvinder Singh Bakshi
Bakshi Hospital, Bhatinda

In my clinical practice, I rarely prescribe paracetamol or weak opioid analgesics, instead preferring diclofenac for its greater effectiveness in reducing pain, improving physical function, and treating synovitis. It has a controlled-release formulation that offers approximately 25 h of well-sustained therapeutic impact, an everyday administration, and the best gastroprotective profile in comparison to other generic NSAIDs. I find that combining diclofenac with a proton-pump inhibitor can reduce gastrointestinal side effects by approximately 40%, which enhances patient compliance. I avoid using oral narcotics for managing OA, as they are associated with significant medication-related adverse effects and provide limited pain relief when compared to other treatment options.

26. Dr. Vivek Agarwal
Agarwal Orthopedic Dental Diagnostic Center, Delhi

In my clinical practice, I regularly prescribe diclofenac due to its effectiveness in decreasing pain and enhancing functional ability while addressing synovitis, which leads to both pain and disease progression. It has a controlled-release formulation that gives approximately 25 h of constant plasma concentration, once-a-day administration and superior gastroprotective properties compared to generic NSAIDs. When it comes to patients with hypertension, I do not prescribe etoricoxib because this drug significantly increases blood pressure.

27. Dr. Sandeep Kumar
Budha Hospital, Bijnor

In my clinical practice, I often prescribe diclofenac for treating synovitis, the primary cause of pain and disease activity, as it effectively relieves pain and improves physical function. This formulation offers ≥25 h of therapeutic benefits with once-daily dosing and shows a superior gastroprotective profile compared to most generic NSAIDs. I do not recommend etoricoxib, as it carries a higher risk of increasing blood pressure in patients with hypertension compared to other drugs in the same class.

28. Dr. Jatinder Sachdeva
Sachdeva Orthopedic Center, Faridabad

I prefer using an NSAID or COX-2 inhibitor (excluding etoricoxib 60 mg) for managing OA, in accordance with guideline recommendations, as paracetamol and weak

opioids are generally ineffective. I consistently recommend diclofenac due to its strong efficacy in pain relief, improving physical function, and managing synovitis, which contributes to pain and the progression of OA. Its sustained-release formulation allows for once-daily dosing and offers enhanced gastroprotective benefits compared to other generic NSAIDs. I avoid prescribing etoricoxib to patients with hypertension because it can elevate blood pressure.

29. Dr. Abdus Salam
Avadh Ortho Center

I often prescribe diclofenac because it is highly effective in reducing pain, improving physical function, and lowering synovial fluid levels of PGE2 and inflammatory cytokines. Its slow-release formulation provides prolonged therapeutic effects (~25 h), once-daily dosing has a good gastroprotective profile, comparable to controlled-release opioids. It is known that etoricoxib significantly raises blood pressure when compared to diclofenac, especially in patients with hypertension. I find that combining diclofenac with a proton-pump inhibitor can reduce gastrointestinal side effects by around 40%, which significantly improves patient compliance.

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