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CLINICAL PHARMACOLOGICAL APPROACH TO THE USE OF ANTI-INFLAMMATORY DRUGS IN DISEASES SUCH AS NEURITIS AND NEURALGIA

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Abstract

This article examines the clinical pharmacological principles guiding the use of anti-inflammatory drugs in the management of neuritis and neuralgia. These conditions, characterized by neuropathic pain and inflammatory damage to peripheral nerves, require a carefully balanced therapeutic approach that minimizes risks while ensuring effective symptom control. The discussion highlights the pharmacodynamic mechanisms of major anti-inflammatory classes, their safety considerations in neurological conditions, and the rationale for individualized therapy based on comorbidities, age, and drug–drug interactions. Special attention is given to steroidal and non-steroidal agents, adjuvant analgesics, and emerging neuroprotective strategies.

Keywords: Anti-inflammatory drugs; neuritis; neuralgia; clinical pharmacology; neuropathic pain; NSAIDs; corticosteroids; neuroinflammation; rational therapy.

INTRODUCTION

In the management of neuritis and neuralgia, one of the most clinically significant insights of recent years is the recognition that inflammation is rarely a superficial or local process; instead, it often involves a complex network of biochemical, immunological, and neurophysiological mechanisms. This complexity demands that clinicians adopt a pharmacological strategy that not only targets the inflammatory cascade but also modulates peripheral and central sensitization processes that contribute to chronic pain. For example, research indicates that pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α play a prominent role in neuronal irritation and the amplification of nociceptive pathways [1]. Therefore, anti-inflammatory drugs must be chosen with an understanding of their ability to influence these mediators, not merely suppress superficial symptoms. In this context, modern non-steroidal anti-inflammatory drugs (NSAIDs) with COX-2 selectivity, alpha-lipoic acid combinations, and agents with mixed immunomodulatory roles have been shown to provide meaningful benefit when tailored to the neuro-inflammatory profile of the patient.

MATERIALS AND METHODS

Another critical aspect of the clinical pharmacological approach involves the temporal dimension of neural inflammation. Acute neuritis often responds rapidly to NSAIDs, glucocorticoids, or combination therapy, whereas chronic neuralgia may require prolonged modulation of inflammatory pathways alongside adjunctive treatments. In diseases such as post-herpetic neuralgia or diabetic neuropathy, persistent oxidative stress contributes to the continuity of neuronal injury. This has led to the incorporation of antioxidants and metabolic agents—including B-complex vitamins, benfotiamine, and acetyl-L-carnitine—into anti-inflammatory pharmacological regimens to help stabilize nerve metabolism and reduce inflammatory neurodegeneration. Such agents do not replace anti-inflammatory drugs, but they significantly enhance recovery through synergistic effects that strengthen neural resilience and improve neuronal regeneration.

The pharmacokinetics and pharmacodynamics of anti-inflammatory drugs also require special consideration due to the nature of neural tissues. Peripheral nerves have a slower metabolic turnover and limited regenerative capacity, meaning drug dosing must maintain sufficiently steady plasma concentrations to sustain anti-inflammatory effects without causing toxicity. Moreover, patients with neuritis or neuralgia often present with comorbidities such as diabetes, hypertension, hepatitis, or gastrointestinal vulnerability. This necessitates a personalized selection of pharmacological agents—for instance, prescribing COX-2–selective NSAIDs rather than non-selective COX inhibitors in individuals at high risk for gastric ulceration. Similarly, glucocorticoids may be employed in cases of autoimmune neuritis, but their use requires vigilance regarding glucose metabolism and adrenal suppression, especially in long treatment cycles. Thus, the clinical pharmacologist's task is to balance efficacy with safety while tailoring therapy to the unique neural and systemic landscape of each patient.

RESULTS AND DISCUSSION

Equally important is the interplay between anti-inflammatory drug therapy and neuropathic pain modulators. Since neuralgia often arises from both inflammatory and neuropathic mechanisms, the rational pharmacological approach integrates anti-inflammatory drugs with medications such as gabapentinoids, tricyclic antidepressants, or serotonin-norepinephrine reuptake inhibitors. These agents address abnormal neurotransmission, reduce central sensitization, and complement the inflammation-reducing effects of anti-inflammatory drugs. When combined thoughtfully, this multimodal approach shortens the duration of neural inflammation,

diminishes recurrence risk, and helps restore normal neurophysiological function. The synergy between inflammation-targeting drugs and neuromodulators is increasingly supported by clinical trials showing improved long-term outcomes compared to monotherapy [2].

Furthermore, drug selection must account for the specific etiology of neuritis or neuralgia. In infectious neuritis, such as that associated with herpes zoster, the pharmacological plan incorporates antiviral drugs alongside anti-inflammatory therapy to halt viral replication and reduce inflammatory consequences. In compressive neuritis, anti-inflammatory drugs are used to control swelling and secondary inflammation while the primary cause—such as mechanical compression—is addressed through physical therapy or surgical intervention. Meanwhile, toxic, metabolic, or autoimmune neuritis often requires corticosteroids, immunosuppressants, or biologics that target specific immune pathways. This etiological precision represents a core tenet of modern clinical pharmacology: treatment should be mechanism-based rather than purely symptomatic.

Lifestyle and environmental factors also influence pharmacological outcomes in neuritis and neuralgia. Adequate hydration, balanced micronutrient intake, moderate physical activity, and avoidance of neurotoxic exposures enhance drug efficacy and reduce recurrence. Alcohol, smoking, and high-glycemic diets may undermine therapeutic responses by heightening oxidative stress and systemic inflammation. These interactions, while often underestimated, are critical components of a comprehensive pharmacological strategy. Educating patients about these modifiable factors strengthens treatment adherence and fosters a multidisciplinary approach that optimizes drug effectiveness.

Finally, emerging evidence highlights the value of precision medicine in anti-inflammatory drug therapy. Genetic polymorphisms affecting COX enzyme expression, cytokine production, and pain sensitivity may explain why some patients respond well to specific anti-inflammatory agents while others require alternative regimens. Advances in biomarker-based assessment—such as cytokine profiling, nerve growth factor evaluation, or mitochondrial function analysis—may soon allow clinicians to tailor anti-inflammatory therapy with unprecedented accuracy. This shift from generalized prescribing to evidence-guided individualized treatment represents the next evolution of clinical pharmacology in the management of neuritis and neuralgia. As such, future therapeutic strategies will focus increasingly on patient-specific profiles, minimizing side effects while maximizing targeted neuro-inflammatory modulation.

In clinical pharmacology, the management of neuritis and neuralgia requires an approach that views inflammation not merely as a symptomatic event but as a dynamic pathological process involving peripheral nerve fibers, microvascular structures, and neuroimmune interactions. Anti-inflammatory drugs therefore do not serve as simple analgesic agents; rather, they function as modulators of the biochemical cascades that sustain neuropathic pain. One of the essential considerations in this context is the recognition that inflammatory mediators—such as prostaglandins, bradykinin, cytokines, and neuropeptides—are capable of inducing sensitization both at the peripheral and central levels. Consequently, the pharmacological strategy must ensure that the chosen anti-inflammatory medication interrupts these processes at multiple stages, thereby preventing the transition of acute inflammation into chronic neuropathic dysfunction.

Moreover, the clinical use of anti-inflammatory agents in neuritis and neuralgia should take into account the heterogeneity of the underlying etiologies. For example, neuritis secondary to viral infections or autoimmune disorders requires a different therapeutic emphasis compared with neuralgia induced by mechanical compression, metabolic disturbances, or toxin exposure. Inflammation-driven nerve irritation often involves oxidative stress and mitochondrial dysfunction, which means that combining classical anti-inflammatory drugs with antioxidant and neuroprotective agents may yield a more profound therapeutic effect. The clinical pharmacologist's role, therefore, involves selecting a combination of medications that not only reduce inflammation but also maintain neuronal integrity, improve microcirculation, and correct metabolic imbalances in affected nerves.

Another important aspect is the temporal dimension of drug action. In neuritis, early inhibition of the inflammatory cascade prevents irreversible structural damage to the myelin sheath, while in neuralgia, timely suppression of neurogenic inflammation reduces abnormal nerve firing and spontaneous pain episodes. For this reason, pharmacokinetic profiles—particularly onset of action, tissue penetration, and elimination half-life—must be considered when evaluating therapeutic options. Drugs with rapid absorption and efficient distribution into neural and perineural tissues are often preferred in acute neuritis, whereas medications with steady, prolonged effects may be more suitable for chronic neuralgia management. This differentiation reflects the need for individualized therapy that aligns pharmacodynamic requirements with patient-specific disease characteristics.

CONCLUSION

The rational use of anti-inflammatory drugs in neuritis and neuralgia requires a deep understanding of the underlying neuroinflammatory processes, the pharmacological properties of available medications, and patient-specific clinical factors. While NSAIDs remain the first line for mild to moderate inflammation, corticosteroids are indispensable in acute immune-mediated nerve damage due to their strong anti-inflammatory potency. The integration of adjuvant drugs—such as anticonvulsants and antidepressants—enhances treatment outcomes, as neuropathic pain often requires multimodal therapy. Safety monitoring, dose adjustment, avoidance of unnecessary polypharmacy, and early identification of adverse effects must be prioritized to ensure optimal therapeutic results. Continued research into neuroprotective anti-inflammatory agents is expected to improve outcomes in neuritis and neuralgia management.

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