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Dual Acting Anti Inflammatory Drugs Dealing with Anti Inflammatory Treatment

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INTRODUCTION

Prostaglandins are widely used in gynecology and ophthalmology as active physiological agents forming a new class of drugs for the treatment of cardiovascular disease and some bronchial asthma. The development of sterile inflammation is an example of an intracellular process in which the prostaglandins produced can and in fact trigger vasodilation, increased vascular permeability, pain and fever. These effects of prostaglandins and leukotrienes characterize states of classical inflammation, including sterile conditions. The use of Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) can achieve therapeutic effects by inhibiting prostaglandin secretion. Prostaglandins play a special role in treating glaucoma. Prostaglandin analogues are potent drugs that reduce intraocular pressure by 20%-40% through a unique mechanism of action. Prostaglandin analogues are considered first-line therapy because they have a balanced safety profile. However, patients with a history of inflammatory diseases such as uveitis, herpes, keratitis, and those planning cataract removal should exercise caution when using prostaglandin analogues.

DESCRIPTION

Rheumatic diseases are the leading cause of disability in the Western world, and non-steroidal anti-inflammatory drugs remain the most commonly used treatment. However, NSAIDs cause some serious side effects, the most important of which are stomach damage, stomach ulcers, and kidney damage. Attempts to develop non-steroidal anti-inflammatory drugs without these drawbacks, especially gastrointestinal toxicity, have followed several strategies. Therefore, non-steroidal anti-inflammatory drugs have been associated with gastroprotective agents to counter the adverse effects of inhibition of prostaglandin synthesis. In any case, a few comes about propose a conceivable inclusion of NO within the pathogenesis of joint pain and consequent tissue annihilation. A most promising approach appeared to be the arrangement of novel NSAIDs, focused on at the inducible isoform of prostaglandin synthase (COX-2); they show up to be destitute of gastrointestinal harmfulness, in that they save mucosal prostaglandin union. In any case, a number of later considers have raised genuine questions almost the two central precepts that back this approach, to be specific that the prostaglandins that intercede aggravation and torment are produced solely through COX-2 which the prostaglandins that are vital in gastrointestinal and renal work are delivered exclusively by means of COX-1. The items produced by the 5-lipoxygenase pathway (leukotrienes) are especially critical in aggravation; without a doubt, leukotrienes increment microvascular penetrability and are powerful chemotactic specialists. Besides, restraint of 5-lipoxygenase in a roundabout way diminishes the expression of TNF-alpha (a cytokine that plays a key part in aggravation). These information and contemplations explain the endeavors to get drugs able to repress both 5-lipoxygenase and cyclooxygenases, the so-called double acting anti-inflammatory drugs. Such compounds hold the movement of classical NSAIDs, whereas dodging their fundamental disadvantages, in that diminished generation of gastroprotective prostaglandins is related with a concurrent diminished generation of the gastro-damaging and bronchoconstrictive leukotrienes.

CONCLUSION

In addition, much obliged to their component of activity, double acting anti-inflammatory drugs might not just ease indications of rheumatic infections, but might moreover fulfil, at slightest in portion, the criteria of a more authoritative treatment. In truth, leukotrienes are proinflammatory, increment microvascular penetrability, and are strong chemoattractants, drawing in eosinophils, neutrophils, and monocytes into the synovial layer.

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