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REVIEW ON: ANTI INFLAMMATORY DRUGS

Yashoda N. Tidake[1*], Darshana H. Wani[1], Tushar M. Phalke[1], Priyanka D.Pote[1], Mayuri S. Borbane[1],Rushikesh V. Khorde[1],Shilpa S.Biradar[1], Dr.U.S.Desai[2], Dr. K.S.Shaikh[2]

Student[1], Professor[2]

Department of Pharmaceutics, PES's Modern College of Pharmacy, Nigdi, Pune-411044

Abstract

Inflammation is a biological response of the immune system that can be triggered by a variety of factors, including pathogens, damaged cells and toxic compounds. These factors may induce acute and/or chronic inflammatory responses in the heart, pancreas, liver, kidney, lung, brain, intestinal tract and reproductive system, potentially leading to tissue damage or disease. Inflammatory responses but, more importantly in clinical terms, in the healing process as well. This includes molecular, cellular, animal and clinical studies related to the study of inflammatory conditions and responses, and all related aspects of pharmacology, such as anti-inflammatory drug development, trials and therapeutic developments, etc

NSAIDs are a class of medications used to treat pain, fever, and other inflammatory processes. This activity describes the indications, mechanism of action, administration, adverse effects, contraindications, monitoring, and important points for providers regarding NSAIDs.

Keywords-

Inflammation, Pyrexia, stimuli, response

INTRODUCTION:

Based on visual observation, the ancients characterised inflammation by five cardinal signs, namely redness (*rubor*), swelling (*tumour*), heat (*calor*; only applicable to the body' extremities), pain (*dolor*) and loss of function (*functio laesa*).

More recently, inflammation was described as "the succession of changes which occurs in a living tissue when it is injured provided that the injury is not of such a degree as to at once destroy its structure and vitality"

The classical description of inflammation accounts for the visual changes seen. Thus, the sensation of heat is caused by the increased movement of blood through dilated vessels into the environmentally cooled extremities, also resulting on the increased redness (due to the additional number of erythrocytes passing through the area). The swelling (oedema) is the result of increased passage of fluid from dilated and permeable blood vessels into the surrounding tissues, infiltration of cells into the damaged area, and in prolonged inflammatory responses deposition of connective tissue. Pain is due to the direct effects of mediators, either from initial damage or that resulting from the inflammatory response itself, and the stretching of sensory nerves due to oedema. The loss of function refers to either simple loss of mobility in a joint, due to the oedema and pain, or to the replacement of functional cells with scar tissue.

These processes involve the major cells of the immune system, including neutrophils, basophils, mast cells, T-cells, B-cells, etc.

HISTORY

The Roman polymath Celsus discovered the four indicators of inflammation that doctors still seek for today in the first century B.C.: redness, swelling, heat, and pain. Later, loss of function was added to the list by German doctor Rudolf Virchow, who undoubtedly had that shattered ankle in mind. Procedure that dates back to Greek and Egyptian antiquity. Aulus Celsus, a Roman author who lived between 30 BC and 45 AD, identified the primary four symptoms of inflammation as redness, warmth, swelling, and pain. Terms like edoema are still used to describe inflammation. We now understand that the first three symptoms most likely result from the microvasculature's reaction to inflammation. Galen, Marcus Aurelius's medical professional and surgeon

TYPES OF INFLAMMATION [1]

There are two types of inflammation:

- **Acute inflammation:** The response to sudden body damage, such as cutting your finger. To heal the cut, your body sends inflammatory cells to the injury. These cells start the healing process.

Acute inflammation may cause:

- **Flushed skin at the site of the injury.**
- **Pain or tenderness.**
- **Swelling.**
- **Heat**
- **Chronic inflammation:** Your body continues sending inflammatory cells even when there is no outside danger. For example, in rheumatoid arthritis inflammatory cells and substances attack joint tissues leading to an inflammation that comes and goes and can cause severe damage to joints with pain and deformities.

Chronic inflammation may causes:

- Abdominal pain.
- Chest pain.
- Fatigue. (example: systemic lupus)
- Fever. (example: tuberculosis)
- Joint pain or stiffness. (example: rheumatoid arthritis)
- Mouth sores. (example: HIV infection)
- Skin rash. (example: psoriasis)

MOST COMMON CAUSES OF INFLAMMATION [2]

The most common reasons for chronic inflammation include:

- **Autoimmune disorders**, such as lupus, where your body attacks healthy tissue.
- **Exposure to toxins,** like pollution or industrial chemicals.
- **Untreated acute inflammation**, such as from an infection or injury.

Some lifestyle factors also contribute to inflammation in the body. You may be more likely to develop chronic inflammation if you:

- Drink alcohol in excess.
- Have a high body mass index (BMI) that falls within the ranges for obesity, unless that is a result of being very muscular.
- Exercise at your maximum intensity too frequently, or you don't exercise enough.
- Experience chronic stress.
- Smoke.

MODE OF ACTION OF INFLAMMATION [3]

The immune system's reaction to adverse stimuli like pathogens, damaged cells, poisonous substances, or radiation is inflammation, which has the dual purpose of eliminating harmful stimuli and starting the healing process. Therefore, inflammation is a defence mechanism that is essential to health.

The coordinated activation of signalling pathways that control the amounts of inflammatory mediators in resident tissue cells and inflammatory cells drawn from the circulation constitutes the inflammatory response. Numerous chronic diseases, including as cancer, diabetes, rheumatoid arthritis, and intestinal and cardiovascular diseases, have an inflammation-based aetiology. Although the exact nature and location of the first stimulus determine the inflammatory response processes, they all have a similar mechanism that may be summed up as follows:

1) Negative stimuli are recognised by cell surface pattern receptors;

2) Inflammatory pathways are triggered;

3) Inflammatory markers are produced and

4) The recruitment of inflammatory cells.

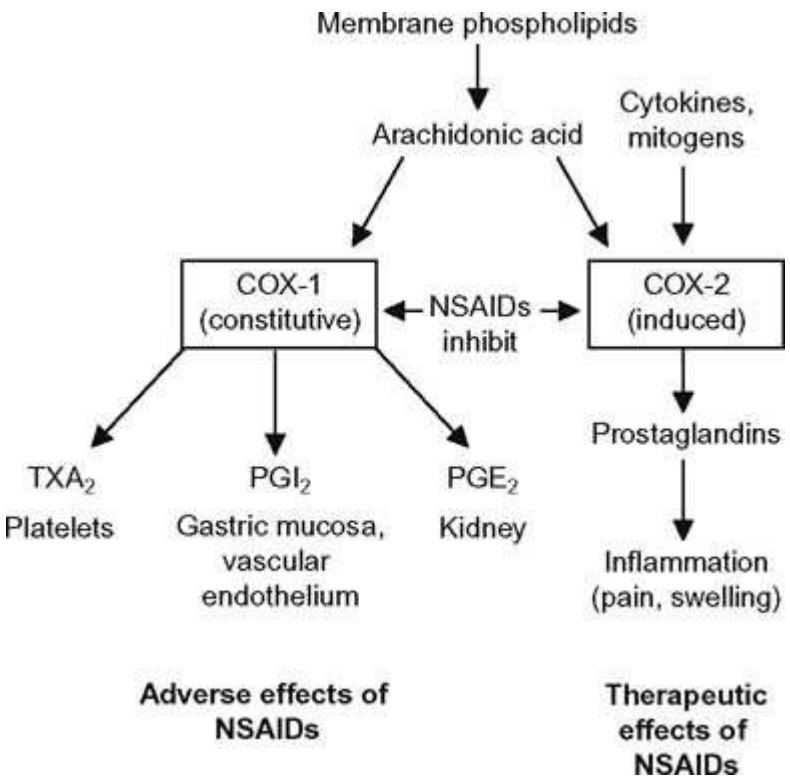


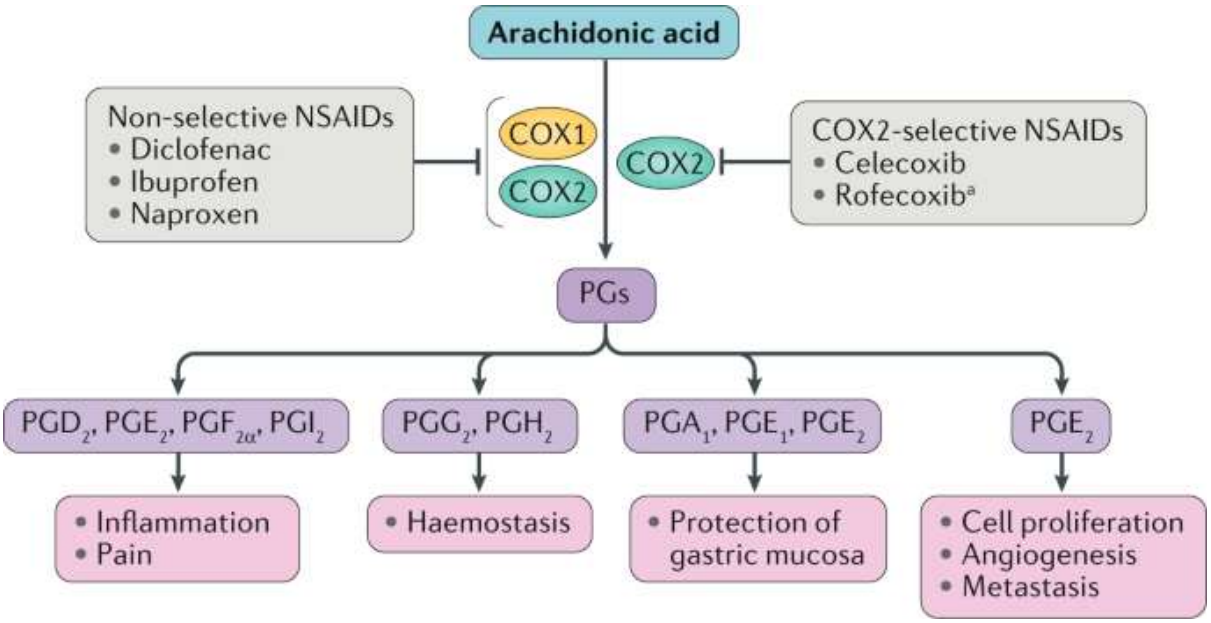
Fig: Inflammatory responses diagram

Nonsteroidal Anti-inflammatory Drugs (NSAIDs) [3]

Nonsteroidal anti-inflammatory drugs (NSAIDs) are a drug class FDA-approved for use as antipyretic, anti-inflammatory, and analgesic agents.These effects make NSAIDs useful for treating muscle pain, dysmenorrhea, arthritic conditions, pyrexia, gout, migraines, and used as opioid-sparing agents in certain acute trauma cases.

NSAIDs are typically divided into groups based on their chemical structure and selectivity: acetylated salicylates (aspirin), non-acetylated salicylates (diflunisal, salsalate), propionic acids (naproxen, ibuprofen, acetic acids (diclofenac, indomethacin), enolic acids (meloxicam, piroxicam) anthranilic acids (meclofenamate, mefenamic acid), naphthylalanine (nabumetone), and selective COX-2 inhibitors (celecoxib, etoricoxib).

Anti-inflammatory drugs reduce inflammation, minimizing its direct effect on pain-nerve stimulation and sensitivity, as well as decreasing the resulting inflammatory heat and swelling. In this way, NSAIDs help relieve pain.



Mechanism of Action [5]

Cyclooxygenase (COX) inhibition is the primary mechanism by which NSAIDs work. Arachidonic acid must be converted into cyclooxygenase in order to produce thromboxanes, prostaglandins, and prostacyclins. The absence of these eicosanoids is thought to be responsible for NSAIDs' therapeutic benefits. In particular, prostaglandins produce vasodilation, raise the temperature set-point in the hypothalamus, and contribute to anti-nociception. Thromboxanes are involved in platelet adhesion.

COX-1 and COX-2 are the two cyclooxygenase isoenzymes. In the body, COX-1 is constitutively produced and is involved in maintaining the lining of the gastrointestinal tract, renal function, and platelet aggregation. The body does not express COX-2 constitutively; rather, it expresses when there is an inflammatory reaction. The majority of NSAIDs inhibit both COX-1 and COX-2 and are nonselective. COX-2, however, selective NSAIDs, such as celecoxib, only target COX-2 and have a different set of side effects. Importantly, COX-2 selective NSAIDs should offer anti-inflammatory treatment without harming the gastric mucosa because COX-2 is primarily involved in inflammation and COX-1 is the primary mediator for maintaining gastric mucosal integrity.

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