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Toxicity Consequences of Chemically Contrived Anti-Inflammatory Drugs: NSAIDs

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Editorial

Anti-inflammatory drug are potentially used as pain-killer and reducing the tissue inflammation for faster wound healing. Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) block the inflammatory pathways by inhibiting the action of enzymatic activity and signaling molecule. They compounds have also some toxic effect on other tissue or organs, that results tissue or organ damage or failure.

Conventionally described as a physiological immune response, inflammation is a process which is solely elicited by multitude of stimuli like injuries in tissues and pathogenic infections. It is basically the body's shielding strategy to subdue the harmful infections encountered and bring back to normal function. It is a non-specific response which is broadly classified as acute and chronic based on their rate of onset and persistence over time [1,2]. Acute inflammation is characterized by its brisk onset resulting in a rapid decline subsequently and is short-lived. Fever occurrence and elevated generation of leucocytes are the most vital features of acute inflammation. On the other hand, chronic inflammation is marked by its prolonged existence resulting in more perilous consequences. Despite the beneficial aspects of acute inflammatory activities as a way of overcoming the damages caused to the body, long term persistence of inflammation might be harmful to the body. And thereby, several anti-inflammatory agents which can be naturally derived or synthetically manufactured are implemented on a large scale.

The working mechanisms of these agents are quite varied as some of them intrude in the inflammatory processes while others provide relief based on the corresponding symptoms. Corticosteroids are one of the most substantial anti-inflammatory agents whose mechanism of anti-inflammatory activity is attributed to apparent immuno-suppression. They are responsible for directing the anti-inflammatory process by lessening the circulating lymphocytes by apoptosis [3]. Evidences owing to several researches have shown that they are mostly found to be distributed in spleen and bone marrow.

Apart from corticosteroids as anti-inflammatory agents, non-steroidal drugs are also prominently recognized as potential inhibitors of inflammation. The chief non-steroidal anti-inflammatory drug to be widely acknowledged is aspirin [4,5]. It is markedly constituted by acetylsalicylate as the key component. Its defense mechanism is attributed to the hindering of cyclo-oxygenase inflammatory pathway by impeding the generation of thromboxanes and prostaglandins which are known as the mediators of inflammatory activities. COX-1 and COX-2 are the two enzymes classified in the enzymatic conversion of released arachidonic acid as a result of tissue damage to prostaglandins and thromboxanes attributing to the cyclo-oxygenase pathway [6]. Aspirin directs the selective obstruction of COX-2 enzymes which leads to the inhibition of inflammatory mediators' production. And hence non selective impediment of both the COX enzymes in turn results in multiple complications of normal physiological activities such as side effects inclusive of gastrointestinal problems, ulcer formation and hemorrhage [7]. Gastric problems are the major and most recognized side effects of aspirin and other related NSAIDs. This can be justified by the fact that COX-1 enzyme is considerably responsible for securing the mucosa of gastrointestinal tract.

Other conceding after-effects of NSAIDs also include minimizing the healing of tendon, cartilage and ligaments due to the retarded regeneration of muscles. These chemically contrived anti-inflammatory agents are also known to cause detrimental effects on the functional properties of kidneys and eventually leading to renal failure which is chiefly characterized by the suppression of production of prostaglandins [8]. Hemorrhage, perforation and ulceration in both large and small intestines have also been depicted as some conventional side-effects of NSAIDs [9]. Other than the customarily observed side-effects such as gastritis and membranal bleeding, there are numerous relative effects identified like edema, hypertension, hyperglycemia, elevated deposition

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of fats in stomach, back and chest, dermatitis and depression [10]. And therefore it may be inferred that ingestion of chemically manufactured anti-inflammatory drugs like aspirin as one of the most common NSAIDs have life-threatening after-effects despite providing favorable benefits in terms of inhibition of inflammation.

The significant impact of NSAIDs on other organs which is attributed as its toxicity properties led to the sole substitution of these chemically prepared anti-inflammatory agents to naturally fabricated ones. Inflammation impeded by agents that are acquired from naturally occurring products with anti-inflammatory properties is studied on a wide range by apprehending the multitude of side effects and toxic properties of these NSAIDs such as aspirin.

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