Talanta 85 (2011) 8–27

Contents lists available at ScienceDirect

Talanta

journal homepage: www.elsevier.com/locate/talanta

Review

Review of the applications of different analytical techniques for coxibs research

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ARTICLE INFO

Article history:
Received 13 October 2010
Received in revised form 15 April 2011
Accepted 19 April 2011
Available online 28 April 2011

Keywords:
NSAIDs
COX-2 inhibitors
Analytical methods
Pharmaceuticals
Biological samples
Validation

ABSTRACT

An extensive survey of the literature published in analytical and pharmaceutical chemistry journals has been conducted and analytical methods which were developed and used for the determination of some of the COX-2 inhibitors, a subclass of non-steroidal anti-inflammatory drugs (NSAIDs) in bulk drugs, formulations, and biological fluids have been reviewed. This review covers the time period from 1999 to present, during which over 140 analytical procedures including chromatographic, spectrometric, electrophoretic and voltammetric techniques were reported. Presented applications concern analysis of coxibs from pharmaceutical formulations and biological samples.

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Abbreviations: ACN, acetonitrile; AI, active ingredient; APCI, atmospheric pressure chemical ionization; BCG, bromocresol green; BCP, bromocresol purple; CD, cyclodextrin; CE, capillary electrophoresis; CMS, chlorophenylmethylsulfone; COX, cyclooxygenase; CZE, capillary zone electrophoresis; ^{1}D, first derivative spectrophotometry; DAD, diode array detector; EtOH, ethanol; FDA, Food and Drug Administration; HMDE, hanging mercury drop electrode; HPLC, high-performance liquid chromatography; HPTLC, high-performance thin layer chromatography; I.S., internal standard; ICP, inductively coupled plasma; IR, infrared; λ, wavelength; LC, liquid chromatography; LOD, detection limit; LOQ, quantitation limit; M, concentration [mol L^{-1}]; MEKC, micellar electrokinetic capillary chromatography; MeOH, methanol; MS, mass spectrometry; NMR, nuclear magnetic resonance; NSAIDs, non-steroidal anti-inflammatory drugs; OSA, octane sulfonic acid; PDA, photo diode array; PG, prostaglandin; PPAC, 4-n-pentyl-phenylacetic acid; RP-HPLC, reversed-phase high-performance liquid chromatography; RRLC, rapid resolution liquid chromatography; RSD, relative standard deviation; SDS, sodium dodecyl sulfate; TBA, tetrabutylammonium; TBAHS, tetrabutylammonium hydrogen sulfate; TCAA, trichloroacetic acid; TEA, triethylamine; TFA, trifluoroacetic acid; THF, tetrahydrofuran; TLC, thin layer chromatography; UPLC, ultra performance liquid chromatography; UV, ultraviolet.

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0039-9140/$ – see front matter © 2011 Elsevier B.V. All rights reserved.
doi:10.1016/j.talanta.2011.04.052

1. Introduction

Rheumatoid arthritis is an autoimmune disease that causes inflammation in the lining of the joints, which results in pain, stiffness, swelling, joint damage and loss of function in the joints. Osteoarthritis is the result of wear and tear of the material that cushions joints, usually in weight-bearing joints. Osteoarthritis is often accompanied by some inflammation but not to the degree seen in rheumatoid arthritis. Presently millions of people live with the pain of arthritis, which may be mild or debilitating. There are many medications available to relieve arthritis pain and slow the progression of the disease.

Non-steroidal anti-inflammatory drugs (NSAIDs) are readily available and effective and thus are extensively used by patients. The growing demand for these agents stimulates a search for new, even more effective drugs, but also calls for higher levels of quality control of these therapeutic substances and preparations, so that they are in the highest possible degree free from any impurities that may come from the production process, as well as from decomposition products of active or auxiliary substances. Therefore, it seems appropriate to develop new analytical methods regarding their analysis.

The progress of analytical chemistry in the scope of instrumentalisation of the methods of chemical analysis is reflected in the use thereof in pharmacopeia monographs as well as in the standards adopted by manufacturers. A constant place is occupied by chromatographic (especially liquid chromatography (LC)) and spectrophotometric methods. Electromigrational and voltamperometric methods are also used for the determination of NSAIDs.

2. NSAIDs—COX-1 and COX-2 inhibitors

Form a historical viewpoint, the first NSAID with therapeutic benefits was aspirin, which has now been used for more than 100 years. In the 1970s, a scientific breakthrough occurred with the elucidation of the molecular mechanism of aspirin and other NSAIDs. Vane, Samuelson and Bergstrom succeeded in showing that these anti-inflammatory substances block the biosynthesis of prostaglandins (PGs) which contribute to a variety of physiological and pathophysiological functions. PGs are produced by most cells and are also present in tissues, which explains their broad spectrum of biological responses. PGs mediate a number of characteristic features of the body's response to tissue injury or inflammation. The outstanding effects of the PGs include their cytoprotective properties in the gastrointestinal tract and control of renal functions in the kidney. PGE_2 is the most important PG which mediates the typical symptoms of inflammation: rubor, calor, tumor, dolor and function laesa. Dilatation of small blood vessels initiates the development of redness and heat; the increase in vascular permeability causes the characteristic swelling of tissues. Moreover, PGs sensitize peripheral nerve endings and nociceptors to transmit pain signals to the brain and the spinal cord.

Prostaglandins, formed by cyclooxygenases (COX) are important mediators for a number of physiological processes and pathophysiological conditions, including inflammation and pain. COX catalyzes the conversion of arachidonic acid (or other 20 carbon fatty acids) to PGG_2 and PGH_2, which are subsequently converted to a variety of eicosanoids that include PGE_2, PGD_2, $PGF_{2,a}$, PG_{T2} and thromboxane. The prostacyclins, prostaglandins, and thromboxanes act as important mediators of physiological and inflammatory responses. Two main isoforms of cyclooxygenase have been identified. The discovery of two COX, the constitutive form COX-1 and the inducible form COX-2, brought forth a new generation of NSAIDs, COX-2 inhibitors [1]. The COX-1/COX-2 model did not explain the properties of paracetamol (acetaminophen). Although its antipyretic and analgesic effects might be explained by inhibition of COX-2, it was not anti-inflammatory. In 2002, Dan Simmons' group reported the discovery of a new COX isoenzyme that was putatively the specific target of acetaminophen. They have named COX-3 [2].

Generally, the NSAIDs inhibit both COX-1 and COX-2. Most NSAIDs are mainly COX-1 selective (e.g., aspirin, ketoprofen, indomethacin, piroxicam). Others are considered slightly selective for COX-1 (e.g., ibuprofen, naproxen) and others may be considered slightly selective for COX-2 (e.g., nabumetone, meloxicam). The mechanism of action of coxibs (e.g., celecoxib) is primarily selective inhibition of COX-2. At therapeutic concentrations, the COX-1 isoenzyme is not inhibited thus gastrointestinal toxicity may be decreased. COX-1 inhibitors inactivate platelet cyclooxygenase irreversibly and at high dosages the COX-1 inhibition is generalized and more damage to the gastrointestinal tract results, so that COX-1 is mainly associated with homeostasis. On the contrary, inducible COX-2 would be the mayor isoenzyme responsible for the production of proinflammatory mediators, and for these reasons COX-2 inhibitors had no effect on platelet aggregation, and lower rates of gastrointestinal, pulmonary and renal side effects would be expected. The main advantage of selective COX-2 inhibitors is that they cause fewer gastrointestinal complications than conventional NSAIDs.

The term NSAID is an abbreviation of a class of drugs as non-steroidal anti-inflammatory drugs. This nomenclature was given to the class to differentiate it from steroids, the other major anti-inflammatory class of drugs. In addition to their anti-inflammatory effects, agents belonging to the NSAID class possess both analgesic and antipyretic activities. Hence, NSAIDs are sometimes referred to as non-narcotic analgesics or as aspirin-like drugs (aspirin was the first member of the class to be discovered). The class of NSAID drugs illustrates the close relationship between the chemical structure of drugs on one side and their biological effects and kinetic properties on the other side. Recently, some NSAIDs have emerged as part of a new class of cancer chemotherapeutic and chemopreventive agents. NSAIDs are agents that reduce inflammation by inhibiting the COX enzymes. However, in spite of their beneficial effects, NSAIDs have a tendency to interfere with the body's ability to protect the stomach lining as well as protect platelet function. Therefore, manifestations of toxicity may be unacceptable in many patients. This paved the way for the discovery and development of newer agents called COX-2-specific inhibitors (coxibs), which are like NSAIDs in that they inhibit the inflammatory conditions while they preserve homeostatic functions such as the integrity of the stomach lining or platelet control. The new class of agents has efficacy comparable to NSAIDs, but with a much improved safety profile such that their use in both the treatment of acute and chronic pain, with or without inflammatory conditions, has been widely accepted. In less than a two decades after the discovery of COX-2, clinical trials have demonstrated that treatment with highly selective COX-2 inhibitors (especially more selective coxibs) causes significantly fewer serious gastrointestinal adverse events than does treatment with classical NSAIDs [3].

Coxibs are appropriate second-line agents when a patient has specific factors that preclude NSAID use. The choice of appropriate NSAID or coxib should be based on patient risk factors, adverse effects and cost. Today, there is an abundant availability of NSAIDs and coxibs that patients may be initially treated with, and perhaps switched during the therapy. Therefore, development of an assay that has a generic application for quantitative determinations of a number of NSAIDs and coxibs has significant utility.

NSAIDs are widely prescribed drugs in clinical practice for the treatment of osteoarthritis, rheumatoid arthritis and other painful conditions. All available NSAIDs can inhibit both COX-1 and COX-2 enzymes at a given dose. NSAIDs side effects are mostly caused by COX-1 inhibition in the stomach, kidney, uterus and platelets

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