Sulindac Tablets USP, 150 mg and 200 mg

Only as directed.

**Pharmacodynamics**

Aspirin is a non-steroidal anti-inflammatory drug (NSAID) that exhibits anti-inflammatory, analgesic, and antipyretic effects in animals. The mechanism of action of aspirin is not completely understood but is related to its ability to inhibit prostaglandin synthesis.

**Pharmacokinetics**

Absorption

Sulindac Tablets are a rapidly disintegrating, enteric-coated, sodium starch glycolate. The tablet core contains a potato starch and magnesium stearate, which are absorbable substances. After oral administration, sulindac is readily absorbed from the gastrointestinal tract into the portal circulation and then to the systemic circulation. In general, the extent of absorption of sulindac is not appreciably influenced by the age, sex, or weight of patients. The absolute bioavailability of sulindac in adults is approximately 60%.

Distribution

Sulindac distribution is readily reversible to unbound sulindac. It is distributed into the tissues with high protein binding and is also found in the cerebrospinal fluid, choroid plexus, and bone. Sulindac is extensively metabolized in the liver and excreted mainly as inactive metabolites in the urine and to a lesser extent in the bile. The plasma half-life of sulindac is approximately 3 hours.

Metabolism

Sulindac undergoes a major biotransformation of its sulfuric acid moiety, catalyzed by the cytochrome P450 liver enzyme system. This results in the formation of sulfide, sulfoxide, and sulfone metabolites. These metabolites are known to be responsible for the anti-inflammatory and analgesic effects of sulindac.

Elimination

The elimination of sulindac is dependent on hepatic metabolism and renal excretion. The hepatic metabolism of sulindac is mediated by cytochrome P450 2C9 and 3A4 enzymes. The renal excretion of sulindac is primarily through the glomerular filtration process. The mean elimination half-life of sulindac is approximately 3 hours.

**Pharmacologic Actions**

Sulindac is an NSAID that has been demonstrated to be effective in the treatment of pain, fever, and inflammation in a variety of experimental and clinical settings. It is believed to inhibit the synthesis of prostaglandins, which are involved in the mediation of pain, fever, and inflammation.

**Clinical Pharmacology**

Sulindac is rapidly and completely absorbed from the gastrointestinal tract. The oral bioavailability of sulindac is approximately 60%. The peak plasma concentration of sulindac is achieved within 1-2 hours after a single dose. The plasma half-life of sulindac is approximately 3 hours. Sulindac undergoes extensive metabolism in the liver and is excreted primarily as inactive metabolites in the urine and to a lesser extent in the bile. The plasma half-life of sulindac is approximately 3 hours.

**Absorption**

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**Distribution**

Sulindac is widely distributed throughout the body. It is distributed into the tissues with high protein binding and is also found in the cerebrospinal fluid, choroid plexus, and bone. Sulindac is extensively metabolized in the liver and excreted mainly as inactive metabolites in the urine and to a lesser extent in the bile. The plasma half-life of sulindac is approximately 3 hours.

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Sulindac Tablets USP, 150 mg are yellow, round tablets, bisected and debossed with “1” to the left of bisect and “10” to the right of bisect on one side, and plain on the other side, available in bottles of 100, 500 and 1000.

Sulindac is indicated for the treatment of pain and inflammation associated with osteoarthritis, rheumatoid arthritis, and ankylosing spondylitis.

Because clinical studies of sulindac were conducted in patients with osteoarthritis, rheumatoid arthritis, and ankylosing spondylitis, the efficacy of sulindac in other conditions in the absence of osteoarthritis, rheumatoid arthritis, and ankylosing spondylitis is not established.

In osteoarthritis, rheumatoid arthritis, and ankylosing spondylitis, the recommended starting dosage is 150 mg twice a day. The dosage may be lowered or raised depending on the response.

In rheumatic fever, therapy for 7 days is usually adequate. In acute gouty arthritis, therapy for 7 days is usually adequate.

Sulindac Tablets USP, 150 mg are for oral use only.

In clinical trials, the active sulfide metabolite of sulindac has been associated with an increase in cyclosporine-induced toxicity, possibly due to decreased synthesis of cyclosporine's active metabolite in patients with systemic lupus erythematosus (SLE) and mixed connective tissue disease, see PRECAUTIONS (Drug Interactions).

General

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