diarrhoea, joint pain, back pain, weakness, orthostatic hypotension, tachycardia, ECG changes (non specific ST-T changes, U waves, left ventricular strain), modification of libido, polyuria, gout, tinnitus, malaise, fainting, BUN increase, blood creatinine increase, hypochloraemia and hyponatraemia.

Overdose & Its Treatment

Symptoms of overdosage include nausea, vomiting, weakness, gastrointestinal disorders and electrolyte imbalance. In severe instances, hypotension and depressed respiration may be observed. If this occurs, support of respiration and cardiac circulation should be instituted. Treatment would be symptomatic, directed at correcting electrolyte abnormalities and gastric lavage or emesis should be considered. In cirrhotic patients, overdosage might precipitate hepatic coma. Discontinue drug; induce emesis or perform gastric lavage; correct dehydration, electrolyte imbalance, hepatic coma and hypotension by established procedures.

Pharmaceutical Particulars

Incompatibilities : None reported

Shelf life : 24 months

Storage precautions : Store at or below 25°C, in a dry place, protected from light.

Keep out of reach of children.

Presentation :

Box of 100 tablets (10 x 10's blister strip).

Manufactured by
MODI-MUNDIPHARMA PVT. LTD.,
Mfd. at : Modipuram - 250 110, U.P., India
Regd. Off. : 1400, Modi Tower,
98, Nehru Place, New Delhi - 110 019, India

TM : Trade Mark

INC1-PTM1-E00/0607-GLO

For the use of a Registered Medical Practitioner or a Hospital or a Laboratory.

Rx

Controlled Release Tablets of Indapamide

INDICONTIN™

CONTINUS™ controlled release system

Description

Each yellow, film-coated caplet embossed with 'INC' on one of the facets contains 1.5mg of Indapamide U.S.P. in Continus™ controlled release system.

Indications

For the treatment of essential hypertension.

Clinical Pharmacology

Indapamide is an oral antihypertensive agent. The mechanism where by indapamide exerts its antihypertensive action has not been completely elucidated; both vascular and renal actions have been implicated. The possible beneficial pharmacological effects of indapamide in the treatment of hypertension include a reduction in cardiac hypertrophy and a reduction in the thickening of arterial walls, prevention of the accumulation of the embryonic isoform of fibronectin in coronary vessels, free radical scavenging leading to stimulation of vasodilator eicosanoid formation, and interaction with renal carbonic anhydrase.

The renal effects of indapamide are minimal and the antihypertensive effect of indapamide has been attributed to a reduction in vascular reactivity to pressor amines. The finding that indapamide retains its antihypertensive activity to functionally anephric patients lends support to the hypothesis.

The renal site of action of indapamide is the proximal segment of the distal tubule. Indapamide appears to have natriuretic properties (sodium and chloride being excreted in equivalent amounts) with less effect on kaliuresis or uric acid excretion. Only at doses greater than 1.5mg an appreciable increase in urinary volume is observed. No significant changes in plasma sodium levels have been observed in clinical studies. Hypokalaemia (plasma potassium level < 3.2 mmo/L) occurred in 4-11% of the recipients of controlled release tablets of indapamide 1.5 mg per day.

Indapamide kinetics is linear. The fraction of indapamide released is rapidly and totally absorbed via the gastrointestinal digestive tract. Ingestion with food slightly increases the rate and extent of absorption. These changes are unlikely to be clinically significant. Peak serum level following a single dose occurs about 12 hours after ingestion; repeated administration reduces the variation in serum levels between 2 doses. Indapamide is widely distributed throughout the body, with extensive binding to some specific sites. In blood, it is highly bound to red blood cells and, more specifically, to carbonic anhydrase without having any significant inhibiting activity on this enzyme. Binding of indapamide to plasma proteins is 79 %. The plasma elimination half-life is 14 to 24 hours (mean 18 hours). The drug has a volume of distribution of approximately 60 L. Steady state is achieved after 7 days. Repeated administration does not lead to accumulation.