

**RANIPETS
TABLETS
GASTROENTERIC**

It antagonizes histamine h2 receptors in the parietal cells of the stomach. It inhibits basal and stimulated gastric acid secretion and reduces pepsin production.

APPROVAL NUMBER Q – 7972 – 118

Each tablet contains:

Ranitidine HCl 45 mg.

Excipient c.b.p 1 tablet.

PRODUCT PRESENTATION

RANIPETS TABLET box with 30 tablets.

Ranitidine is a histamine H₂ receptor antagonist. It inhibits both basal and stimulated gastric acid secretion (histamine, pentagastrin and food). After the administration of ranitidine, the volume of gastric juice is reduced, the acid fraction and the pepsin fraction in a greater proportion the first than the second, ranitidine is between 5 and 12 times more potent than other drugs of the same family such as Cimetidine as an H₂ antagonist and shows a lower affinity towards the hepatic enzyme system of cytochrome P450, therefore it has fewer interactions with other drugs than cimetidine. Ranitidine is indicated in the treatment of gastrointestinal disorders in which gastric acid secretion is increased.

INDICATIONS

It acts as a competitive antagonist of histamine H₂ receptors that reduces gastric acid secretion. Indicated in the treatment of gastritis, gastric ulcer, duodenal ulcer, esophagitis, gastroesophageal reflux, drug gastritis, or in any state where an effective control of gastric acid secretion is required.

PHYSICOCHEMICAL CHARACTERISTICS

Synonyms: RanitidinI hydrochloridum; Ranitidine hydrochloride

Appearance: Crystalline powder.

Color: White to pale yellow.

Smell: Characteristic.

Flavor: Acid and bitter.

Polymorphic

Formula: C₁₃H₂₂N₄O₃S, HCL

CAS: 66357-59-3

Molecular weight: 350.9

Solubility: Very soluble in water, methanol and dilute hydrochloric acid. Slightly soluble in alcohol and chloroform; very slightly soluble in methylene chloride. A 1% aqueous solution has a pH 4.5 - 6.0.

Melting point: 133 - 134 °C

HOW IT WORKS

Ranitidine competitively inhibits the binding of histamine to gastric parietal cell receptors (called H₂ receptors) by reducing secretion of basal acid and stimulated by food, caffeine, insulin, or pentagastrin. Ranitidine reduces the volume of acid excreted in response to stimuli thereby indirectly reducing pepsin secretion.

They also inhibit the secretion stimulated during shock situations, periods of stress or by the administration of insulin, caffeine and non-steroidal anti-inflammatory drugs. This inhibition is manifested by a reduction in the volume and concentration of hydrogen ions and by a corresponding decrease in pepsin. Ranitidine has no effect on gastrin, nor does it affect gastric emptying, motility, intraesophageal pressure, peristalsis, or bile and pancreatic secretions. It also has no anticholinergic properties. Ranitidine shows a healing effect on the gastrointestinal mucosa, protecting it from the irritant action of acetylsalicylic acid and other non-steroidal anti-inflammatory drugs.

H₂ antagonists alone do not eradicate *Helicobacter pylori* and should always be used in conjunction with an adequate antibiotic regimen with 2 or more antibiotics such as amoxicillin + clarithromycin, amoxicillin + metronidazole, or other combinations. Ranitidine slightly stimulates prolactin secretion, but has no effect on gonadotropin, TSH, or GL secretion. It also does not affect plasma levels of cortisol, aldosterone, androgens, or estrogens. They also inhibit the other effects due to stimulation of H₂ receptors: the part of vasodilation and hypotension that corresponds to the H₂ component, as well as inotropic and chronotropic stimulation of the heart. Ranitidine has some cholinomimetic activity, probably due to an anticholinesterase action, but this activity does not appear to be relevant for its therapeutic utility.

PHARMACOKINETICS AND PHARMACODYNAMICS

Ranitidine can be administered orally or parenterally. Intramuscular and intravenous administration show a bioavailability of 90-100%, while orally, the bioavailability is less than 80% because the drug undergoes a first-pass metabolism, although food does not appreciably alter the extent absorption or peak level. The half-life in serum is 2.2 hours and the volume of distribution is 2.6 L / kg. with a net systemic bioavailability of approximately 50%. Peak levels occur 2-3 hours after oral dosing.

The drug is widely distributed in the body, with significant levels found in cerebrospinal fluid and breast milk. Ranitidine is widely distributed throughout the body and only 10-19% to plasma proteins. The inhibitory effects on gastric acid secretion last between 8 and 12 hours; It is partially metabolized in the liver and is excreted through urine and feces, a part in the form of metabolites, and a part to be altered. After an intravenous dose, approximately 70% of the dose is excreted in the urine unchanged. The half-life of the drug is 2 to 3 hours, increasing to 5 hours in patients with renal failure (creatinine clearance <35 ml). Renal secretion of ranitidine is carried out by tubular secretion and by glomerular filtration. Ranitidine is metabolized in the liver to ranitidine N-oxide, N-demethylranitidine, ranitidine S oxide, and the analog of furanic acid.

ADVERSE EFFECTS

As with other H₂ antagonists, adverse reactions during ranitidine treatment are rare and, when they do occur, are mild and transient. Hepatitis, jaundice, and elevated transaminases have been reported rarely.

- **Overdose:** Studies in dogs that have received doses of ranitidine greater than 225 mg / kg / day have shown tremor, vomiting and polypne.
- **Chronic and subclinical toxicity (parenteral administration):** No signs of specific local irritation attributable to ranitidine have been observed. All treated animals (mice, rats, rabbits, and dogs) remained healthy. Rats received doses of 13 or 20 mg of ranitidine per kg of body weight, while rabbits received 11.5 mg / kg of body weight per day by I.V., S.C. or I.M. For 5 days. In another study conducted, rats and dogs received doses of 2.5 to 5 mg / kg of body weight daily IV. for 15 and 28 days, respectively. There were isolated cases of dogs with loose stools; conversely, no ranitidine-related changes were observed.
- **Precautions and relationship with effects of carcinogenesis, mutagenesis, teratogenesis and on fertility:** There have been no reports to the Date of carcinogenic, mutagenic, teratogenic or fertility effects due to the use of oral ranitidine.

WARNINGS

Ranitidine is contraindicated in patients with known hypersensitivity to the active substance. It should be used with caution and at a reduced dose in patients with impaired renal function. Adverse effects appear to be very rare in animals.

DRUG INTERACTIONS

Induced ranitidine enzyme inhibition is low, so drugs that are metabolized by liver microsomal enzymes are not affected by doses of less than 300 mg of ranitidine per day. Ketoconazole and itraconazole are weak bases that require an acidic environment for oral absorption, so treatment with ranitidine can affect their bioavailability. Furthermore, due to the sustained action of the H₂ blocker, this interaction can occur even when antifungals are administered at very different times of the day. On the other hand, both ketoconazole and itraconazole are potent inhibitors of the CYP3A4 enzyme system so that a substantial elevation of their blood levels may occur when ranitidine treatment is discontinued.

Antacids can reduce ranitidine absorption by up to 25%, so it is recommended that both drugs be given at least an hour apart. Ranitidine can affect the pharmacokinetics of some oral cephalosporins. Thus, for example, ranitidine reduces the area under the curve of cefuroxime by more than 40% and that of cefodoxime by 29%. Ranitidine reduces the oral bioavailability of some quinolones in humans, there are no studies suggesting the same in animals.

Sucralfate may slightly reduce the bioavailability of ranitidine. Although it was initially thought that reducing gastric acidity might reduce the ability of sucralfate to bind to ulcerated tissues of the digestive tract, animal studies have shown that this interaction does not occur and that sucralfate is equally effective at high pH.

SPECIES

Canines and Felines

DOSES**RANIPETS TABLETS**

- **Canines:** 2 mg / kg every 12 hours for 14-21 days, equivalent to 1 tablet / 22 kg of weight.
- **Felines:** 3.5 mg / kg every 12 hours for 14-21 days, equivalent to 1 tablet / 12 kg of weight.

ROUTE OF ADMINISTRATION

Oral

**MEDICAL PRESCRIPTION IS NECESSARY FOR SELL
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