Montallerg Kid Leaflet

Size: A5 148mm

210mm

Montallerg Kid Syrup

Montelukast Sodium and Levocetirizine Dihydrochloride

Quantitative composition:
Each 5ml contains: Montelukast Sodium 4mg & Levocetirizine Dihydrochloride 2.5 mg
Pharmaceutical form: Oral Syrup
Pharmacology:
As Montallerg Kid Syrup is combination of montelukast and levocetirizine: the pharmacology properties of both the molecules are given separately:
ATC code -R03DC53 — Montelukast, combinations
Levocetirizine: Pharmacotherapeutic group: Antihistamine for systemic use, piperazine derivatives Levocetirizine, the (R) enantiomer of cetirizine, is a potent and selective antagonist of peripheral H1-receptors. Binding studies revealed that levocetirizine has high affinity for human H1-receptors (Ki = 3.2 nmol/l). Levocetirizine has an affinity 2-fold higher than that of cetirizine (Ki = 6.3 nmol/l). Levocetirizine dissociates from H1-receptors with a half-life of 115 ± 38 min. After single administration, levocetirizine shows a receptor occupancy of 90% at 4 hours and 57% at 24 hours Pharmacodynamic studies in healthy volunteers demonstrate that, at half the dose, levocetirizine has comparable activity to cetirizine, both in the skin and in the nose.
Pharmacokinetic /pharmacodynamics relationship 5mg levocetirizine provide a similar pattern of inhibition of histamine-induced wheal and flare than 10mg cetirizine, the action on histamine-induced skin reactions was out of phase with the plasma concentrations. EGGS did not show relevant effects of levocetirizine on QT interval
Montelukast: Pharmacotherapeutic group: Leukotriene receptor antagonist
Mechanism of action the cysteinyl leukotrienes (LTC4, LTD4, LTE4) are potent inflammatory eicosanoids released from various cells including mast cells and eosinophils. These important pro-asthmatic mediators bind to cysteinyl leukotriene receptors (CysLT) found in the human airway and cause airway actions, including bronchoconstriction, mucous secretion, vascular permeability, and eosinophil recruitment.
Pharmacodynamic effects: Montelukast is an orally active compound which binds with high affinity and selectivity to the CysLT1 receptor. In clinical studies, montelukast inhibits bronchoconstriction due to inhaled LTD4 at doses as low as 5 mg. Bronchodilation was observed within 2 hours of oral administration. The bronchodilation effect caused by a β-agonist was additive to that caused by montelukast. Treatment with montelukast inhibited both early- and late-phase bronchoconstriction due to antigen challenge.
Pharmacokinetics:
Levocetirizine
The pharmacokinetics of levocetirizine are linear with dose- and time-independent with low inter- subject variability. The pharmacokinetic profile is the same when given as the single enantiomer or when given as cetirizine. No chiral inversion occurs during the process of absorption and elimination
Absorption: Levocetirizine is rapidly and extensively absorbed following oral administration. Peak plasma concentrations are achieved 0.9 h after dosing. Steady state is achieved after two days. Peak concentrations are typically 270 ng/ml and 308 ng/ml following a single and a repeated 5 mg o.d. dose, respectively. The extent of absorption is dose-independent and is not altered by food, but the peak concentration is reduced and delayed.
Distribution: No tissue distribution data are available in humans, neither concerning the passage of levocetirizine through the blood-brain-barrier. In rats and dogs, the highest tissue levels are found in liver and kidneys, the lowest in the CNS compartment. Levocetirizine is 90% bound to plasma proteins. The distribution of levocetirizine is restrictive, as the volume of distribution is 0.4l/kg.
Biotransformation: The extent of metabolism of levocetirizine in humans is less than 14% of the dose and therefore differences resulting from genetic polymorphism or concomitant intake of enzyme inhibitors are expected to be negligible. Metabolic pathways include aromatic oxidation, N- and O- dealkylation and taurine conjugation. Dealkylation pathways are primarily mediated by CYP 3A4 while aromatic oxidation involved multiple and/or unidentified CYP isoforms. Levocetirizine had no effect on the activities of CYP isoenzymes 1A2, 2C9, 2C19, 2D6, 2E1 and 3A4 at concentrations well above peak concentrations achieved following a 5 mg oral dose. Due to its low metabolism and absence of metabolic inhibition potential, the interaction of levocetirizine with other substances, or vice-versa, is unlikely.
Elimination: The plasma half-life in adults is 7.9 ± 1.9 hours. The mean apparent total body clearance is 0.63 ml/min/kg. The major route of excretion of levocetirizine and metabolites is via urine, accounting for a mean of 85.4% of the dose. Excretion via feces accounts for only 12.9% of the dose. Levocetirizine is excreted both by glomerular filtration and active tubular secretion.
Renal impairment the apparent body clearance of levocetirizine is correlated to the creatinine clearance. It is therefore recommended to adjust the dosing intervals of levocetirizine, based on creatinine clearance in patients with moderate and severe renal impairment. In anuric end stage renal disease subjects, the total body clearance is decreased by approximately 80% when compared to normal subjects.
Montelukast
Absorption: Montelukast is rapidly absorbed following oral administration. For the 10 mg film coated tablet, the mean peak plasma concentration (Cmax) is achieved 3 hours (Tmax) after administration in adults in the fasted state. The mean oral bioavailability is 64%. The oral bioavailability and Cmax are not influenced by a standard meal.
Distribution: Montelukast is more than 99% bound to plasma proteins. The steady-state volume of distribution of montelukast averages 8-11litres.
Biotransformation: Montelukast is extensively metabolised. In studies with therapeutic doses, plasma concentrations of metabolites of montelukast are undetectable at steady state
Cytochrome P450 2C8 is the major enzyme in the metabolism of montelukast. Additionally, CYP 3A4 and 2C9 may have a minor contribution, although itraconazole, an inhibitor of CYP 3A4, was shown not to change pharmacokinetic variables of montelukast in healthy-subjects that received 10 mg montelukast daily. Based on in-vitro results in human liver microsomes, therapeutic plasma concentrations of montelukast do not inhibit cytochromes P450 3A4, 2C9, 1A2, 2A6, 2C19, or 2D6. The contribution of metabolites to the therapeutic effect of montelukast is minimal.
Elimination: The plasma clearance of montelukast averages 45 ml/min in healthy adults. Following an oral dose of radio labelled montelukast, 86% of the radioactivity was recovered in 5-day faecal collections and <0.2% was recovered in urine. Coupled with estimates of montelukast oral bioavailability, this indicates that montelukast and its metabolites are excreted almost exclusively via the bile. No dosage adjustment is necessary for the elderly or mild to moderate hepatic insufficiency.
Studies in patients with renal impairment have not been undertaken
Indications:
- Relief of symptoms of allergic rhinitis [seasonal or perennial]
- As prophylaxis in seasonal allergic rhinitis and
- Treatment of comorbid asthma and allergic rhinitis in patients 15 years of age and over.

Posology and method of administration:
Method of administration: Oral use
Children (2-5 years): 5 ml syrup as measured from the given cup once daily.
Method of administration
Take Montallerg Kid Syrup preferably with meal through oral route.
Contraindications:
Hypersensitivity to the active substance or to any of the excipient
Observed reactions range from urticaria to anaphylaxis.
Also, contradicted in patients with end stage renal disease at less than 10 ml/min creatinine clearance, and patients undergoing hemodialysis.
Children 6 months to 11 years of age with impaired renal function should not be administered.
Special warnings and precautions for use:
Do not exceed the stated dose.
Precaution is recommended if alcohol is taken concomitantly.
Caution in epileptic patients and patients at risk of convulsions is recommended.
Patients should be advised never to use oral montelukast to treat acute asthma attacks and to keep their usual appropriate rescue medication for this purpose readily available.
If an acute attack occurs, a short-acting inhaled beta-agonist should be used.
Montelukast should not be substituted abruptly for inhaled or oral corticosteroids.
There are no data demonstrating that oral corticosteroids can be reduced when montelukast is given concomitantly.
Patients on therapy with anti-asthma agents including montelukast may present with systemic eosinophilia, sometimes presenting with clinical features of vasculitis consistent with Churg-Strauss syndrome, a condition which is often treated with systemic corticosteroid therapy. These cases usually, but not always, have been associated with the reduction or withdrawal of oral corticosteroid therapy. The possibility that leukotriene receptor antagonists may be associated with emergence of Churg-Strauss syndrome can neither be excluded nor established.
Physicians should be alert to eosinophilia, vasculitic rash, worsening pulmonary symptoms, cardiac complications, and/or neuropathy presenting in their patients.
Patients who develop these symptoms should be reassessed and their treatment regimens evaluated.
Treatment with montelukast does not alter the need for patients with aspirin-sensitive asthma to avoid taking aspirin and other non-steroidal anti-inflammatory drugs.
Interaction with other medicinal products and other forms of interaction:
Due to the pharmacokinetic, pharmacodynamic and tolerance profile of levocetirizine, no interactions are expected with this antihistamine. Actually, neither pharmacodynamic nor significant pharmacokinetic interaction was reported in drug-drug interactions studies performed, notably with pseudoephedrine or theophylline
Montelukast may be administered with other therapies routinely used in the prophylaxis and chronic treatment of asthma.
The area under the plasma concentration curve (AUC) for montelukast was decreased approximately 40% in subjects with co-administration of phenobarbital.
Since montelukast is metabolised by CYP 3A4, 2C8, and 2C9, caution should be exercised, particularly in children, when montelukast is co-administered with inducers of CYP 3A4, 2C8, and 2C9, such as phenytoin, phenobarbital and rifampicin.
In drug-interactions studies, the recommended clinical dose of Montelukast did not have clinically important effects on the pharmacokinetics of the following medicinal products: theophylline, prednisone, prednisolone, oral contraceptives (ethinyl estradiol/ norethindrone 35/1), terfenadine, digoxin and warfarin.
No routine dosage adjustment of montelukast is required upon coadministration with gemfibrozil or other potent inhibitors of CYP 2C8, but the physician should be aware of the potential for an increase in adverse reactions.
Co-administration of montelukast with itraconazole, a strong inhibitor of CYP 3A4, resulted in no significant increase in the systemic exposure of montelukast.
Pregnancy and Lactation
There are no adequate and well controlled studies of either montelukast or levocetirizine in pregnant women.
Hence this combination should not be used during pregnancy.
Caution should be exercised when prescribing to pregnant or breastfeeding women because levocetirizine passes into breast milk
Effects on ability to drive and use machines:
Patients should be advised not to drive or use machines until they have established their own response
Patients intending to drive, engaging in potentially hazardous activities or operating machinery should not exceed the recommended dose and should take their response to the medicinal product into account.
Adverse reactions:
Common: Somnolence, Dizziness Headache, Pharyngitis Rhinitis, Abdominal pain Dry mouth Nausea, headache, abdominal pain, Fatigue
Uncommon: Agitation, Paresthesia, Diarrhea, Pruritus Rash, Asthenia, Malaise
Rare: Aggression, Confusion Depression, Hallucination, Insomnia, Movement disorders, Tachycardia, Hepatic function abnormal, Urticaria, Oedema, Weight increased, thirst
Diarrhoea, eczematous, hyperkinesia, asthma, Dysuria Enuresis, Blurred vision, Dysgeusia, Syncope, Tremor Dystonia Dyskinesia, Thrombocytopenia
Overdose and Treatment:
Symptoms observed after an overdose of Levocetirizine are mainly associated with CNS effects or with effects that could suggest an anticholinergic effect.
Adverse events reported after an intake of at least 5 times the recommended daily dose are: confusion, diarrhoea, dizziness, fatigue, headache, malaise, mydriasis, pruritus, restlessness, sedation, somnolence, stupor, tachycardia, tremor and urinary retention.
Management of overdoses There is no known specific antidote to Levocetirizine.
Should overdose occur, symptomatic or supportive treatment is recommended.
Gastric lavage should be considered following ingestion of a short occurrence. Levocetirizine/Montelukast is not effectively removed by dialysis.
Presentation: 60 ml PET Bottle, packed in printed unit carton along with literature insert.
Shelf life: 2years from the date of manufacture.
Storage: Do not store above 30°C. Protect from direct sunlight. Keep all medicines out of reach of children.
Distribution category: Prescription only medicine (POM).

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