Lansoprazole

Class: Gastric Acid Secretion Inhibitor, Proton Pump Inhibitor

Pharmacokinetics

Absorption

i.) Non-genetic
   a. Food: Decreased absorption rate and extent. [Note: peak serum levels and AUC values achieved with 30 mg lansoprazole in the presence of food are comparable to those of 15-mg oral doses in the fasting state.]
   b. Concomitant medications/substances: p-glycoprotein inhibitors

ii.) Genetic
   a. Possible: genetic variation in p-glycoprotein gene

Distribution

i.) Non-genetic
   a. Altered serum protein (97-99% protein bound, primarily to albumin)
      i. Renal impairment: protein binding decreased 1-1.5%
   b. Concomitant medications/substances
      i. Drugs highly bound to albumin may displace lansoprazole from protein binding sites; lansoprazole may displace other drugs from albumin binding sites
      ii. P-glycoprotein inhibitors or activators

ii.) Genetic
   a. Possible: genetic variation in p-glycoprotein gene

Metabolism

i.) Non-genetic
   a. Concomitant medications/substances: CYP2C19, CYP3A4 inducers or inhibitors
   b. Age: decreased clearance in elderly (peak levels not increased with once daily dosing)
   c. Hepatic disease: decreased clearance, substantial increase in area under the curve

ii.) Genetic
   a. Genetic variation in drug metabolizing enzyme gene: CYP2C19, CYP3A4

Excretion

i.) Non-genetic
   a. Renal insufficiency: decreased elimination half-life, decreased area under the curve

ii.) Genetic
   a. No clear genetic factors affecting excretion

Pharmacodynamics

Receptors

i.) Non-genetic
   a. Concomitant medications: histamine H2 receptor agonists or antagonists (may block or enhance effects of lansoprazole)

ii.) Genetic
   a. No clear genetic factors affecting receptors

Transporters

i.) Non-genetic
   a. Concomitant medications/substances: p-glycoprotein inhibitors or activators

ii.) Genetic
   a. Possible: genetic variation in p-glycoprotein gene

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