POTASSIUM CITRATE

CALTA

Rx

1.620 g (15 mEq) EXTENDED-RELEASE TABLET

ANTIUROLITHIC

CYDO

FORMULATION:
Each Extended-Release Tablet contains:
Potassium citrate, USP…….1.620 g

PRODUCT DESCRIPTION:
White to off white, elongated biconvex uncoated extended-release tablets, plain on one side and break line on other side.

PHARMACODYNAMICS:
When Potassium citrate is given orally, the metabolism of absorbed citrate produces an alkaline load. The induced alkaline load in turn increases urinary pH and raises urinary citrate by augmenting citrate clearance without measurably altering ultra filterable serum citrate. Thus, Potassium citrate therapy appears to increase urinary citrate principally by modifying the renal handling of citrate, rather than by increasing the filtered load of citrate. The increased filtered load of citrate may play some role, however, as in small comparisons of oral citrate and oral bicarbonate, citrate had a greater effect on urinary citrate. In addition to raising urinary pH and citrate, Potassium citrate increases urinary potassium by approximately the amount contained in the medication. In some patients, Potassium citrate causes a transient reduction in urinary calcium.
The changes induced by Potassium citrate produce urine that is less conducive to the crystallization of stone-forming salts (calcium oxalate, calcium phosphate and uric acid). Increased citrate in the urine, by complexing with calcium, decreases calcium ion activity and thus the saturation of calcium oxalate. Citrate also inhibits the spontaneous nucleation of calcium oxalate and calcium phosphate (brushite). The increase in urinary pH also decreases calcium ion activity by increasing calcium complexation to dissociated anions. The rise in urinary pH also increases the ionization of uric acid to the more soluble urate ion.

PHARMACOKINETICS:
Potassium citrate is absorbed and the citrate is metabolized to bicarbonate. Citric acid is metabolized to carbon dioxide and water. Oxidation is virtually complete with less than 5% of citrate being excreted unchanged in the urine.

INDICATIONS:
Potassium citrate is indicated for the management of renal tubular acidosis (RTA) with calcium stones, hypocitraturic calcium oxalate nephrolithiasis of any etiology, and uric acid lithiasis with or without calcium stones.

DOSAGE AND ADMINISTRATION:
In patients with severe hypocitraturia (urinary citrate of less than 150 mg/day), therapy should be initiated at a dosage of 60 mEq/day (20 mEq three times/day or 15 mEq four times/day with meals or within 30 minutes after meals or bedtime snack).
In patients with mild-moderate hypocitraturia (>150 mg/day), Potassium Citrate Tablet should be initiated at a dosage of 30 mEq/day (10 mEq three times/day with meals).
Twenty-four-hour urinary citrate and/or urinary pH measurements should be used to determine the adequacy of the initial dosage and to evaluate the effectiveness of any dosage change. In addition, urinary citrate and/or pH should be measured every four months. Or as prescribed by the physician.

CONTRAINDICATIONS:
Potassium citrate Tablet is contraindicated in patients with hyperkalemia (or who have conditions predisposing them to hyperkalemia), as a further rise in serum potassium concentration may produce cardiac arrest. Such conditions include: chronic renal failure, uncontrolled diabetes mellitus, acute dehydration, strenuous physical exercise in unconditioned individuals, adrenal insufficiency, extensive tissue breakdown, or the administration of a potassium-sparing agent (such as triamterene, spironolactone or amiloride). Potassium citrate Tablet is contraindicated in patients in whom there is cause for arrest or delay in tablet passage through the gastrointestinal tract, such as those suffering from delayed gastric emptying, esophageal compression, intestinal obstruction or stricture or those taking anticholinergic medication. Because of its ulcerogenic potential, Potassium citrate Tablet should not be given to patients with peptic ulcer disease. Potassium citrate Tablet is contraindicated in patients with renal insufficiency (glomerular filtration rate of less than 0.7 mL/kg/min), because of the danger of soft tissue calcification and increased risk for the development of hyperkalemia.

WARNINGS AND PRECAUTIONS:
Hyperkalemia:
In patients with impaired mechanisms for excreting potassium, Potassium citrate administration can produce hyperkalemia and cardiac arrest. Potentially fatal hyperkalemia can develop rapidly and be asymptomatic. The use of Potassium citrate in patients with chronic renal failure, or any other condition which impairs potassium excretion such as severe myocardial damage or heart failure, should be avoided. Closely monitor for signs of hyperkalemia with periodic blood tests and ECGs.
Gastrointestinal Lesions:
Because of reports of upper gastrointestinal mucosal lesions following administration of potassium chloride (wax-matrix), an endoscopic examination of the upper gastrointestinal mucosa was performed in 30 normal volunteers after they had taken glycopyrrolate 2 mg by mouth three times a day, Potassium citrate 95 mEq/day, wax-matrix potassium chloride 96 mEq/day or wax-matrix placebo, in thrice daily schedule in the fasting state for one week. Potassium citrate and the wax-matrix formulation of potassium chloride were indistinguishable but both were significantly more irritating than the wax-matrix placebo. In a subsequent, similar study, lesions were less severe when glycopyrrolate was omitted.

Solid dosage forms of potassium chlorides have produced stenotic and/or ulcerative lesions of the small bowel and deaths. These lesions are caused by a high local concentration of potassium ions in the region of the dissolving tablets, which injured the bowel. In addition, perhaps because wax-matrix preparations are not enteric-coated and release some of their potassium content in the stomach, there have been reports of upper gastrointestinal bleeding associated with these products. The frequency of gastrointestinal lesions with wax-matrix potassium chloride products is estimated at one per 100,000 patient-years. Experience with Potassium citrate is limited, but a similar frequency of gastrointestinal lesions should be anticipated.
If there is severe vomiting, abdominal pain or gastrointestinal bleeding, Potassium citrate should be discontinued immediately and the possibility of bowel perforation or obstruction investigated.

PREGNANCY AND LACTATION:
Pregnancy
Animal reproduction studies have not been conducted. It is also not known whether Potassium citrate can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Potassium citrate should be given to a pregnant woman only if clearly needed.

Lactation:
The normal potassium ion content of human milk is about 13 mEq/L. It is not known if Potassium citrate has an effect on this content. Potassium citrate should be given to a woman who is breastfeeding only if clearly needed.

DRUG INTERACTIONS:
Concomitant administration of Potassium citrate and a potassium-sparing diuretic (such as triamterene, spironolactone or amiloride) should be avoided since the simultaneous administration of these agents can produce severe hyperkalemia.

ADVERSE DRUG REACTIONS:
Some patients may develop minor gastrointestinal complaints during Potassium citrate therapy, such as abdominal discomfort, vomiting, diarrhea, loose bowel movements or nausea. These symptoms are due to the irritation of the gastrointestinal tract, and may be alleviated by taking the dose with meals or snacks, or by reducing the dosage. Patients may find intact matrices in their feces.

OVERDOSE AND TREATMENT:
The administration of potassium salts to persons without predisposing conditions for hyperkalemia rarely causes serious hyperkalemia at recommended dosages. It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration and characteristic electrocardiographic changes (peaking of T-wave, loss of P-wave, depression of S-T segment and prolongation of the QT interval). Late manifestations include muscle paralysis and cardiovascular collapse from cardiac arrest.
Treatment measures for hyperkalemia include the following:

1. Patients should be closely monitored for arrhythmias and electrolyte changes.
2. Elimination of medications containing potassium and of agents with potassium-sparing properties such as potassium-sparing diuretics, ARBs, ACE inhibitors, NSAIDs, certain nutritional supplements and many others.
3. Elimination of foods containing high levels of potassium such as almonds, apricots, bananas, beans (lima, pinto, white), cantaloupe, carrot juice (canned), figs, grapefruit juice, halibut, milk, oat bran, potato (with skin), salmon, spinach, tuna and many others.
4. Intravenous calcium gluconate if the patient is at no risk or low risk of developing digitalis toxicity.
5. Intravenous administration of 300-500 mL/hr of 10% dextrose solution containing 1020 units of crystalline insulin per 1,000 mL.
6. Correction of acidosis, if present, with intravenous sodium bicarbonate.

CAUTION:
Foods, Drugs, Devices, and Cosmetics Act prohibits dispensing without prescription.

ADR REPORTING STATEMENT
For suspected adverse drug reaction, report to FDA: www.fda.gov.ph.
Seek medical attention immediately at the first sign of any adverse drug reaction.

STORAGE CONDITION:
Store at temperatures not exceeding 30°C.

Keep all medicines out of reach of children.

AVAILABILITY:
Alu/Alu Blister Pack x 10's / (Box of 30's)

DRP-14484-03
Date of First Authorization: 10 March 2024
Date of Revision of Package Insert: 10 June 2024

Distributed by:
THE CATHAY YSS DISTRIBUTORS CO., INC.
2/F Vernida I Condominium, 120 Amorsolo St.,
Legaspi Village, Makati, Metro Manila

Imported by:
AMBICA INTERNATIONAL CORPPORATION
No. 9 Amsterdam Extension, Merville Park Subd.,
Parañaque City, Metro Manila

Manufactured by:
COOPER PHARMA LIMITED
C3 Industrial Area, Selaqui,
Dehradun-248197, Uttarakhand, India