NEW ZEALAND DATA SHEET

1 PRODUCT NAME

Potassium Citrate 10, potassium citrate 10 mEq (1080 mg) modified release tablet

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

Each tablet contains 10 mEq (1080 mg) potassium citrate (equivalent to 390 mg potassium).
For the full list of excipients, see section 6.1.

3 PHARMACEUTICAL FORM

Modified release tablet.

Tan to yellowish colour, oval shaped, biconvex uncoated tablets, debossed with "537" on one side and plain on the other side.

4 CLINICAL PARTICULARS

4.1 Therapeutic indications

Potassium citrate is a urinary alkaliniser indicated for the management of:

- Renal tubular acidosis (RTA) with calcium stones.
- Hypocitraturic calcium oxalate nephrolithiasis of any etiology.
- Uric acid lithiasis with or without calcium stones.
- For the symptomatic relief of dysuria associated with mild urinary tract infections.

4.2 Dose and method of administration

Treatment with extended release potassium citrate should be added to a regimen that limits salt intake (avoidance of foods with high salt content and of added salt at the table) and encourages high fluid intake (urine volume should be at least two liters per day). The objective of treatment with potassium citrate extended-release tablets is to provide potassium citrate in sufficient dosage to restore normal urinary citrate (greater than 320 mg/day and as close to the normal mean of 640 mg/day as possible), and to increase urinary pH to a level of 6 or 7.
Monitor serum electrolytes (sodium, potassium, chloride and carbon dioxide), serum creatinine and complete blood counts every four months and more frequently in patients with cardiac disease, renal disease or acidosis. Perform electrocardiograms periodically. Treatment should be discontinued if there is hyperkalemia, a significant rise in serum creatinine or a significant fall in blood hematocrit or hemoglobin.

For the symptomatic relief of dysuria associated with mild urinary tract infections, the recommended dose is 1 tablet 3 times a day (up to a maximum of 2 tablets 3 times a day). Use in this indication is intended to be short term (see section 4.4).

Severe hypocitraturia (urinary citrate < 150 mg/day):
Therapy should be initiated at 60 mEq per day; a dose of 20 mEq three times per day with meals or within 30 minutes after meals or bedtime snack.
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. Doses of potassium citrate extended-release tablets greater than 100 mEq/day have not been studied and should be avoided.

Mild to moderate hypocitraturia (urinary citrate >150 mg/day):
Therapy should be initiated at 30 mEq per day; 10 mEq three times per day with meals or within 30 minutes after meals or bedtime snack.
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. Doses of potassium citrate extended-release tablet greater than 100 mEq/day have not been studied and should be avoided.

Paediatric population:
Safety and effectiveness in children have not been established.

4.3 Contraindications

Potassium citrate extended-release tablets are 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).
- 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.
- In patients with peptic ulcer disease because of its ulcerogenic potential.
- 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.

4.4 Special warnings and precautions for use

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:
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
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.

Urinary tract infections
Raising urinary pH may reduce the effectiveness of some antibiotics (such as nitrofurantoin) and may promote further bacterial growth. Use with caution in urinary tract infections requiring antibiotic treatment.

When used for the symptomatic relief of dysuria associated with mild urinary tract infections, potassium citrate does not treat the underlying bacterial infection. Short term symptomatic relief may be provided through alkalinisation of the urine. Worsening, recurrence, or persistence of symptoms should prompt clinical review.

4.5 Interaction with other medicines and other forms of interaction

Potential Effects of Potassium Citrate on Other Medications
Potassium-sparing diuretics: concomitant administration should be avoided since the simultaneous administration of these agents can produce severe hyperkalemia.

Potential Effects of Other Drugs on Potassium Citrate
Drugs that slow gastrointestinal transit time: These agents (such as anticholinergics) can be expected to increase the gastrointestinal irritation produced by potassium salts.

Renin-angiotensin-aldosterone inhibitors:
Drugs that inhibit the renin-angiotensin-aldosterone system (RAAS) including angiotensin converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), spironolactone, eplerenone, or aliskiren produce potassium retention by inhibiting aldosterone production.
Closely monitor potassium in patients receiving concomitant RAAS therapy.

Nonsteroidal Anti-inflammatory drugs (NSAIDs)
NSAIDs may produce potassium retention by reducing renal synthesis of prostagladin E and impairing the renin-angiotensin system.
Closely monitor potassium in patients on concomitant NSAIDs.

4.6 Fertility, pregnancy and lactation

Pregnancy:
Animal reproduction studies have not been conducted. It is not known whether potassium citrate can cause foetal 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 breast feeding only if clearly needed.

Fertility:
No data available.

4.7 Effects on ability to drive and use machines

Not relevant.

4.8 Undesirable effects

Postmarketing Experience
Some patients may develop minor gastrointestinal complaints during potassium citrate therapy, such as abdominal discomfort, vomiting, diarrhoea, 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 faeces.

Reporting of suspected adverse reactions
Reporting suspected adverse reactions after authorisation of the medicine is important. It allows continued monitoring of the benefit/risk balance of the medicine. Healthcare professionals are asked to report any suspected adverse reactions https://pophealth.my.site.com/carmreportnz/s/

4.9 Overdose

Treatment of Overdosage: 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 to 500 mL/hr of 10% dextrose solution containing 10 to 20 units of crystalline insulin per 1,000 mL.
6. Correction of acidosis, if present, with intravenous sodium bicarbonate.
7. Hemodialysis or peritoneal dialysis.
8. Exchange resins may be used. However, this measure alone is not sufficient for the acute treatment of hyperkalemia.

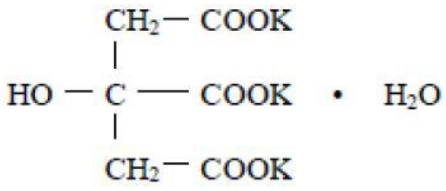
Lowering potassium levels too rapidly in patients taking digitalis can produce digitalis toxicity.

For risk assessment and advice on the management of overdose please contact the National Poisons Centre on 0800 POISON (0800 764766).

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: potassium ATC code: A12BA02
Potassium citrate is a citrate salt of potassium. Its molecular formula is K C H O • H O, and it has the following chemical structure:



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 ultrafilterable 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.

Potassium citrate therapy does not alter the urinary saturation of calcium phosphate, since the effect of increased citrate complexation of calcium is opposed by the rise in pH-dependent dissociation of phosphate. Calcium phosphate stones are more stable in alkaline urine.

Clinical efficacy and safety

The pivotal potassium citrate trials were non-randomized and non-placebo controlled where dietary management may have changed coincidentally with pharmacological treatment.

5.2 Pharmacokinetic properties

In the setting of normal renal function, the rise in urinary citrate following a single dose begins by the first hour and lasts for 12 hours. With multiple doses the rise in citrate excretion reaches its peak by the third day and averts the normally wide circadian fluctuation in urinary citrate, thus maintaining urinary citrate at a higher, more constant level throughout the day. When the treatment is withdrawn, urinary citrate begins to decline toward the pre-treatment level on the first day.

The rise in citrate excretion is directly dependent on the potassium citrate dosage. Following long-term treatment, potassium citrate at a dosage of 60 mEq/day raises urinary citrate by approximately 400 mg/day and increases urinary pH by approximately 0.7 units.

In patients with severe renal tubular acidosis or chronic diarrheal syndrome where urinary citrate may be very low (<100 mg/day), potassium citrate may be relatively ineffective in raising urinary citrate. A higher dose of potassium citrate may therefore be required to produce a satisfactory citraturic response. In patients with renal tubular acidosis in whom urinary pH may be high, potassium citrate produces a relatively small rise in urinary pH.

5.3 Preclinical safety data

Not available.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Carnauba wax
Magnesium stearate.

6.2 Incompatibilities

Not applicable

6.3 Shelf life

24 months

6.4 Special precautions for storage

Store at or below 25°C

6.5 Nature and contents of container

Plastic bottle containing 100 tablets with child proof closure.

6.6 Special precautions for disposal

No special requirements.

7 MEDICINE SCHEDULE

Pharmacy Medicine

8 SPONSOR

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Pt Chevalier, Auckland 1246
Telephone: (09) 815 2664.

9 DATE OF FIRST APPROVAL

24 July 2025

10 DATE OF REVISION OF THE TEXT

24 July 2025

SUMMARY TABLE OF CHANGES

Section Changed	Summary of new information
	• New