



Armadale Health Service

RENAL NURSE PRACTITIONER CLINICAL PROTOCOL

TITLE: MANAGEMENT OF HYPERPHOSPHATAEMIA

This Clinical Protocol is devised for an exclusive use by the Nurse Practitioner,
Renal Dialysis Unit, Armadale Health Service.

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PURPOSE OF THIS PROTOCOL

To provide clinical practice guidelines for the Renal Nurse Practitioner (RNP) in the management of hyperphosphatemia, including indications for aggressive management, prescription of relevant medications, and diagnostic investigations that may be needed, under supervision of the Renal Physician.

EXPECTED CLIENT OUTCOMES

Maintenance of predialysis serum levels of calcium, phosphate and thus calcium x phosphate (CPP) within target ranges recommended by *Caring for Australians with Renal Impairment* (CARI) guidelines in hemodialysis patients, to enable prevention and control of hyperparathyroidism, renal osteodystrophy, and systemic calcification.

SCOPE OF THIS PROTOCOL

This protocol covers the hemodialysis patients at Armadale Dialysis Unit under the care of RNP and the supervising renal physician.

BACKGROUND

Dialysis patients have increased mortality, the majority due to cardiovascular deaths when compared to patients not on dialysis. This remains when corrected for age, sex, race and modification of traditional risk factors for cardiovascular disease (CVD). Modification of non-traditional risk factors such as dialysis efficacy, control of hyperparathyroidism and systemic calcification, hyperhomocystinemia have thus attracted attention so as to improve morbidity and mortality in this population. Complications of chronic kidney disease (CKD) are evident very early in the disease process. These include increasing cardiovascular events, anaemia and calcium/phosphate/parathyroid hormone abnormalities. The majority of dialysis patients have manifestations of CVD at the initiation of dialysis. (Sarnak & Levey, 2000).

As renal function declines, the majority of patients have abnormal serum phosphorus and calcium concentration, decreasing vitamin D levels, and increased levels of parathyroid hormone (PTH). Abnormalities of phosphorus and calcium metabolism, in particular, hyperphosphatemia contributes to cardiovascular and systemic calcification (Ribeiro et al, 1998; Block et al, 1998; Cozzolino et al, 2001), which is associated with increased mortality (Block et al., 2000; Ganesh et al., 2001). Hyperphosphatemia (S PO₄ > 2.12 mmol/L) was associated with a higher total mortality (RR 1.20), death from coronary artery diseases (RR1.41) and sudden deaths

(RR 1.20) (Nolan, 2005; CARI, 2005). Thus, a target serum phosphate <2.2 mmol/liter (and preferably below 1.8 mmol/litre) on a predialysis blood sample is suggested with a target Calcium Phosphate Product (CPP) below 5.8 mmol²/liter² (preferably below 4.0 mmol²/liter²) by the Caring for Australians with Renal Insufficiency (CARI) Guidelines, 2006.

Hyperphosphatemia may influence vascular calcification by various mechanisms that include worsening of secondary hyperparathyroidism which in turn facilitates calcification, promoting calcium-phosphate deposition in pre-formed endothelial plaques and in the artery walls, effects of loss of mineralisation inhibitors, induction of bone formation via Pit1 channels causing release of mineralisation competent matrix vesicles, altered ALP, decrease matrix Gla, and increased fetulin.

Using ultrafast electron beam computed tomography (CT), young patients undergoing dialysis have commonly been found to have coronary artery, mitral and aortic valve calcification (Goodman et al., 2000). The presence of calcification is associated with time from commencement of dialysis (Raggi et al., 2002), increased age, hyperphosphatemia and high calcium phosphate product (Goodman et al., 2000), high intake of calcium-based phosphate binders and lower alkaline phosphatase levels (Goodman et al., 2000). Coronary artery calcification is thus a marker for atherosclerotic burden and is associated with prevalence of myocardial infarction in End Stage Renal Disease (ESRD) (Raggi et al., 2002).

Serum calcium, phosphate and parathyroid hormone (PTH) levels have interlinked regulatory control with each other, thus the management of all above parameters need to be considered in an individual patient. Serum phosphate is a result of a complex balance of dietary intake, phosphate binders, bone turnover, and dialysis and non-dialysis losses. Phosphate binders have limited capacity to bind intestinal phosphate, thus dietary phosphate intake needs restriction in majority of the patients. Calcium-based phosphate binders such as Caltrate, Cal-Sup are widely used due to their availability on Pharmaceutical Benefits Scheme (PBS). Their use in large doses is accompanied by non-compliance, hypercalcemia, PTH suppression, adverse effects such as constipation and increased risk of systemic calcification. While non-Calcium based binders such as Alu-Tabs are used sparingly due to their risk profile of Aluminium toxicity or availability on PBS.

There is also a complex interplay of dietary PO₄ with protein intake. Patients with poor dietary protein intake may have normal serum phosphate at the cost of malnutrition, which in itself is a risk factor for mortality and morbidity. On the contrary, high protein intake may be associated with high PO₄ intake needing further input from dietician, adjustment of binders and dialysis dose.

Serum phosphate has direct effect on progression of hyperparathyroidism, due to increased PTH syntheses, skeletal resistance, reduction of ionic calcium and probably a direct posttranscriptional action (Slatopolsky et al., 1996). While hyperparathyroidism itself increases risk of hypercalcemia and hyperphosphatemia due to its effects on bone mineralisation. Treatment of hyperparathyroidism with Vit D metabolites increase dietary absorption of calcium and phosphate, often making hyperphosphatemia worse. Thus the balancing act of control of calcium, phosphate

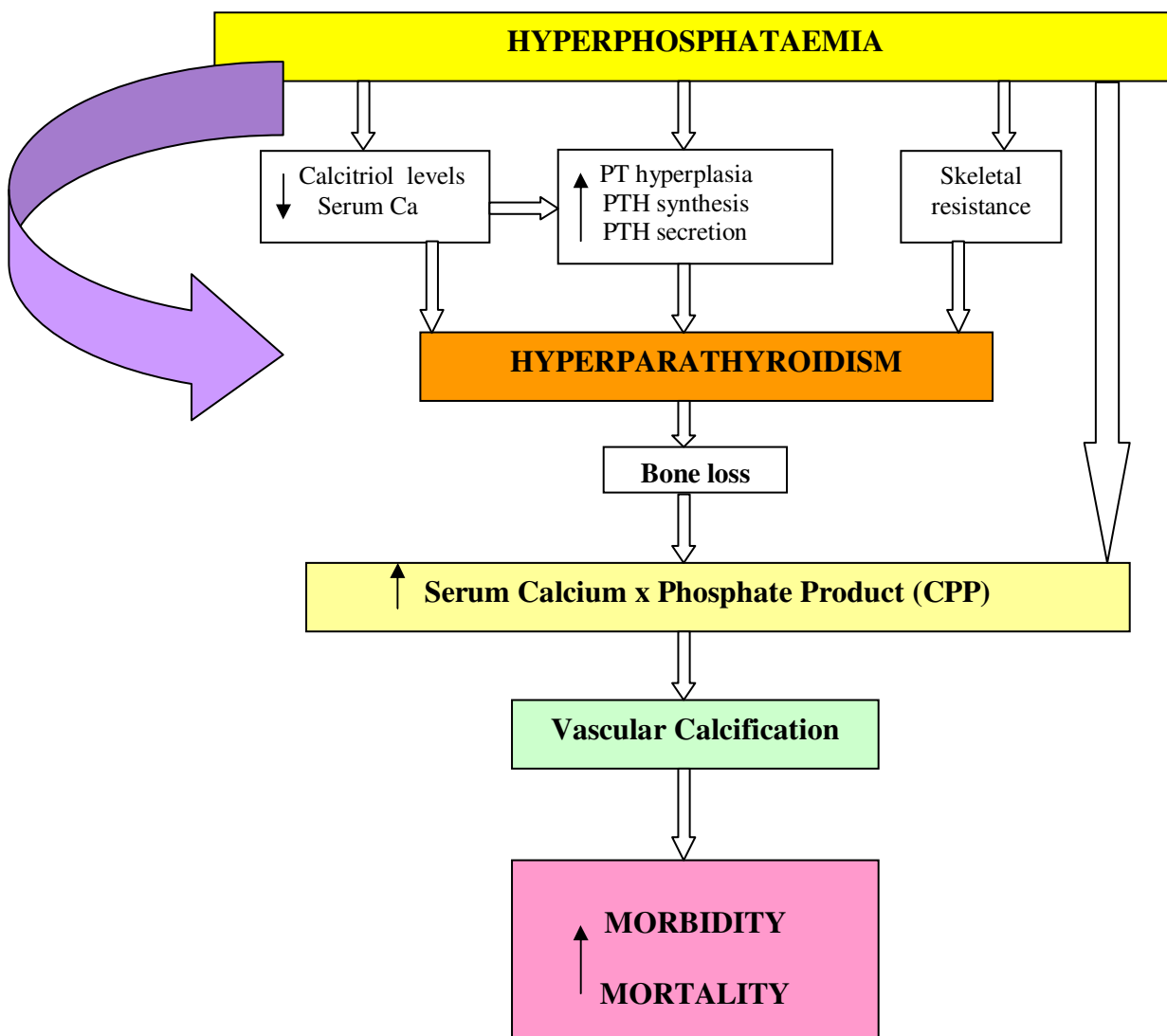
and PTH becomes more challenging needing persistent input, patience, and prolonged followup in an individual patient. Control of hyperphosphatemia has profound independent effect on reduction of hyperparathyroidism, the need for reduction of phosphate binders, and thus facilitates the management of dialysis patient.

The use of non-calcium based phosphate binders is limited due to adverse effects of Aluminium based binders (adynamic bone disease, anaemia, dementia) or increased costs of non Calcium- non Aluminium based phosphate binders such as Sevelamar (Renagel: Need for intake of large number of capsules, nonavailability on PBS, interference in absorption of lipid soluble vitamins) and Lanthanum (non-available on PBS, costs, paucity of long term data regarding metabolism of absorbed Lanthanum). Sevelamer treatment has shown to reduce coronary artery and aortic calcification in dialysis patients after fifty-two weeks of treatment.

Thus the current protocol focuses on the control of hyperphosphataemia as a part of unified management of hyperphosphataemia and secondary hyperparathyroidism (SHPT). The management of SHPT is detailed in a separate clinical protocol.

The potential side effects of hyperphosphataemia and SHPT are illustrated in figure 1.

Figure 1.
Effects of hyperphosphatemia and hyperparathyroidism in renal failure



Recommended Biochemical Targets as per CARI guidelines (2006)
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Guideline 1- Serum Calcium

1. Recommended range for serum calcium corrected for serum albumin is within the range for the local laboratory of **2.1-2.4 mmol/L** (Opinion)
2. A lower end of the range is preferred provided it does not worsen hyperparathyroidism (Opinion)
3. A predialysis blood sample should be used (Level III evidence)

Guideline 2- Serum Phosphate

1. Recommended range for serum phosphate is within the range for the local laboratory of **0.8-1.6 mmol/L** (Level III evidence)
2. Higher levels of serum phosphate have been shown to be associated with an increase in mortality (Level III evidence)
3. A predialysis blood sample should be used (Level III evidence)
4. Serum phosphate above 2.2 mmol/L predicts dietary and medication non-compliance and is independently associated with increased mortality (Level III evidence)
5. The dietary/nutritional advice for haemodialysis patient on a phosphorus intake is 1000-1400 mg/day (32-45 mmol/day) (Dieticians Special Interest Group of the European Dialysis & Transplant Nurses Association, European Renal Care Association, 2003)

Guideline 3- Calcium x phosphate product (CPP)

1. Recommended serum albumin corrected calcium x phosphate product is **< 4.0 mmol/L** (Level III evidence)
2. The recommendation should take into account of optimisation of serum calcium and maintenance of serum phosphate below 1.6 mmol/L to avoid hyperphosphataemia (Level III evidence)

Target levels comparison to international renal guidelines

	Serum Calcium mmol/L	Serum Phosphate mmol/L	Calcium x phosphate product (CPP) mmol ² /L ²
CARI	2.1-2.4	0.8-1.6	4.0
DOQI	2.1-2.37	1.13-1.78	4.4
UK RENAL	2.2-2.7	1.2-1.7	Nil

Note: DOQI-Dialysis Outcomes Quality Initiatives

Levels of evidence used for the CARI guidelines are as follows:

- Level I: Evidence obtained from a systemic review of all relevant randomised controlled trials
- Level II: Evidence obtained from at least one properly designed randomised controlled trials
- Level III: Evidence obtained from comparative studies (cohort studies, case control studies, pseudo-randomised controlled trials etc)
- Level IV: Evidence obtained from case series (either post-test or pre-test / post-test)

Management

Diagnostic investigations

- Monthly
 - predialysis serum calcium, phosphate, albumin, alkaline phosphate
 - calculation of albumin corrected calcium X phosphate product (CPP)
 - Dialysis adequacy- Kt/V
 - arteriovenous access recirculation test
- 3 monthly
 - parathyroid hormone (PTH)
 - pre dialysis serum aluminium if on Alu-Tabs
 - Dialysis adequacy- Fractional Removal of Urea (FRU)

Dietary assessment (in collaboration with renal dietician)

Protein intake: 1.0-1.2 g/kg/day
Phosphorous: restrict to 1.0-1.4 g/day

The main limitation for effective phosphate restriction in patients undergoing dialysis is that it requires a dietary restriction. A protein intake of 1.0-1.2g/kg/day in hypophosphorous diet is recommended to avoid malnutrition. Phosphorus restriction of 1.0-1.4 g/day is recommended. As dietary intake of phosphorus approaches 2g/day, phosphorus binding medications are less effective, and when dietary phosphorus intake exceeds 2.5g/day hyperphosphatemia develops irrespective of the type and dose of the phosphate binders (Llach, 2005).

Dialysis adequacy

Phosphate clearance is effective only during the first 2 hours of dialysis. Serum phosphorus levels do not change during the second half of dialysis. Haemodialysis removes approximately 900 mg of phosphate three times weekly. (Mucsi et al., 1998; Block & Port, 2000). As a result the patients are left with a positive phosphorus balance of 300-500mg/day. It is thus important to maintain adequate dialysis. The recommended dialysis adequacy parameters are as per CARI guidelines (2005):

- Kt/V > 1.3
- FRU > 0.70
- Arteriovenous access recirculation <10%

Medications (see formulary)

Phosphate binders:

Calcium carbonate-Caltrate, Cal-Sup

Aluminium hydroxide-Alu-Tab

Vitamin D analogue:

Calcitriol-hexatriol, rocaltriol

Calcium dialysate concentration:

Dialysate Calcium is involved in the calcium flux and contributes to multitude of variable factors relating to calcium load. Variation in dialysate calcium can be used in individual patients to achieve a particular goal, as there is no end point and long term data to formulate a guideline for its widespread use in dialysis patients. The definition of “standard dialysate calcium” differs in non calcium based phosphate binders era from the calcium based phosphate binder era. Non calcium based phosphate binders are either have potential toxicity (e.g. Aluminium hydroxide) or are non available on PBS (Renagel, Lanthanum) in Australia.

The current levels of dialysate Calcium used in Armadale Dialysis Unit are:

	STANDARD	LOW	HIGH
Dialysate calcium concentration	1.3 – 1.5 mmol/L	1.0 mmol/L	1.75 mmol/L

Low calcium dialysate (1.0 mmol/L) offers some promise in reducing calcium loading, permitting use of calcium-based phosphate binders in the settings of high-normal serum calcium and in the settings of adynamic bone disease. (Sawyer et al., 1989; Slaptopolsky et al., 1989; Van der Marwe et al., 1990). However, low calcium dialysate concentration may predispose to cardiac arrhythmias, intradialytic hypotension and its use should be avoided especially in hemodynamically unstable patients (Toussaint et al., 2006).

High calcium dialysate (1.75 mmol/L) has limited utility as there is increased risk of over suppression of PTH leading to adynamic bone disease, hypercalcemia, metastatic calcification. (Its used should be discussed with the Renal Physician).

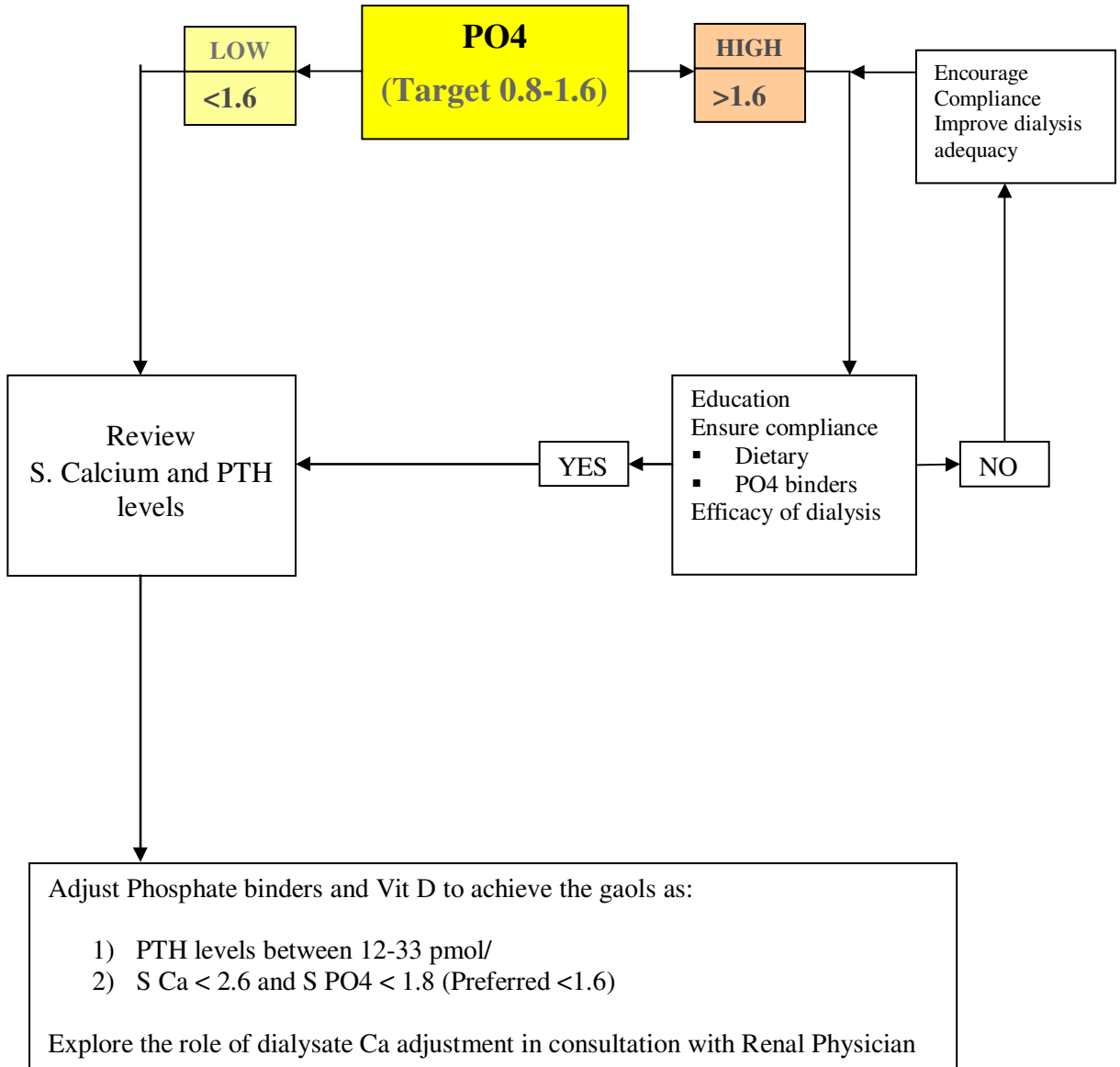
Patient education

- The patients and their carers should be informed about the importance of dietary adherence and medication dosing.
- The risks associated with non-adherence should be highlighted.
- They should be assisted to develop an understanding that phosphate binders should be taken with meals and snacks.
- Enhancement to medication regime adherence could be done in conjunction with providing the patients with information about recent blood biochemistry results.
- They should be informed about the symptoms of hypercalcaemia and hyperphosphataemia .
- The patients should also be asked to consult with their health care provider before using over the counter or complementary medicines.

Management flow chart

The management to achieve target levels of serum calcium, phosphate, CPP is complex and requires the patients' highest degree of medication and dietary compliance, hence patient assessment and education is of utmost priority. The RNP's intervention on blood results outside the recommended target is presented in figure 2.

Figure 2: Management of hyperphosphataemia



DRUG FORMULARY

Drug generic name	Calcium carbonate Product name CALTRATE . Manufacturer Wyeth Consumer
Poison schedule	Unscheduled. Restricted PBS/RPBS
Therapeutic class	Section 21(b) Minerals – Vitamins and Minerals
Preparation & Dosage	Tablets (white, capsule shaped, film coated, scored, marked C/600, CALTRATE on reverse) Calcium carbonate 1500 mg (equiv. Ca 600mg) Pack 600 mg (equiv. Ca) [60]: \$12.73 Pack 600 mg (equiv. Ca) [120]: authority-PBS/RPBS (Rp 1)
Routes	Orally
Administration frequency	1 to 3 tables daily taken with food
Duration of order	Long term, titrate to monthly serum Ca & PO4 levels
Action	As a phosphate binder in chronic renal failure
Indication for use	Hyperphosphatemia associated with chronic renal failure
Contraindications	Hypercalcaemia Hypercalciuria Digoxin toxicity Nephrolithiasis
Interactions	Do not take at the same time as these oral medications: <ul style="list-style-type: none"> ▪ Digoxin-combined use may lead to cardiac arrhythmias ▪ Bisphosphonates-decreased absorption of bisphosphonates; do not take calcium within 2 hours of bisphosphonate administration ▪ Iron, quinolones, tetracyclines, sotalol-calcium may decrease absorption of these drugs. Separate administration by several hours. ▪ Calcitriol-risk of hypercalcaemia; avoid concurrent use unless dietary intake is inadequate
Adverse reactions	Common – belching, flatulence, abdominal distension, Constipation Infrequent- hypercalcaemia, alkalosis, hypophosphataemia, renal calculi Rare - milk-alkali syndrome
Storage	Store below 25 degrees Keep out of reach of children.

Source: Caltrate product information pamphlet, AMH (2005), MIMS (Online)

DRUG FORMULARY

Drug generic name	Calcium carbonate Product name CAL-SUP. Manufacturer : 3M Pharmaceuticals Pty Ltd
Poison schedule	Unscheduled. Restricted PBS/RPBS
Therapeutic class	Section 21(b) Minerals – Vitamins and Minerals
Preparation & Dosage	Ca carbonate 1250 mg (equiv. Ca 500 mg); white spearmint flavoured; gluten free; Pack: 500 mg [60] <u>Private: \$6.74</u> Pack: 500 mg [120] : <u>Authority - PBS/RPBS (Rp 1)</u> [Approved indication(s) for authority: Hyperphosphataemia associated with chronic renal failure.] PBS: \$13.59 Pack: 500 mg [120] : <u>Restricted - RPBS (Rp 1)</u> [Restricted benefit indication(s): Hyperphosphataemia in chronic renal failure; Hypocalcaemia; Osteoporosis; Proven calcium malabsorption.] PBS: \$13.59
Routes	Orally
Administration frequency	2 tabs daily sucked, chewed or swallowed whole. May be taken with or without food
Duration of order	Long term, titrate to monthly serum Ca & PO4 levels
Action	As a phosphate binder in chronic renal failure
Indication for use	Hyperphosphatemia associated with chronic renal failure
Contraindications	Hypercalcaemia Hypercalciuria
Interactions	Calcium may interfere with the absorption of oral iron and tetracyclines
Adverse reactions	Constipation may occur.
Storage	Store below 25 degrees Keep out of reach of children.

Source: Cal-Sup product information pamphlet, AMH (2005), MIMS (Online)

DRUG FORMULARY

Drug generic name	Aluminium hydroxide Product name: Alu-Tab Manufacture name: 3M Pharmaceuticals Pty Ltd
Poison schedule	Unscheduled
Therapeutic class	Antacid
Preparation & Dosage	Tablets, 600 mg (green, film coated, scored, marked 3M on reverse): 100's.
Routes	Orally
Administration frequency	600-1200mg daily taken with food (Maximum 6 tablets daily)
Duration of order	Long term, titrate to monthly serum calcium levels Serum aluminium must be monitored 3 monthly to avoid aluminium toxicity, encephalopathy
Action	Aluminium hydroxide increases phosphate excretion in the bowel by the formation of nonabsorbable phosphate salts.
Indication for use	Use only when other phosphate binders are inadequate for treatment of hyperphosphatemia associated with chronic renal failure
Contraindications	Aluminium accumulation may result in bone disease and encephalopathy; avoid use as an antacid in renal impairment and minimise use as a phosphate binder.
Interactions	Is reported to interfere with the absorption of some medications including tetracycline, penicillin, sulphonamides, iron, digoxin, indomethacin, naproxen, phenylbutazone and vitamins. Aluminium hydroxide and such medications should be administered 2 hours apart.
Adverse reactions	Common – Constipation, chalky taste Infrequent- hypophosphataemia, Rare - intestinal obstruction, osteomalacia, encephalopathy, anaemia, proximal myopathy
Storage	Store below 25 degrees Keep out of reach of children.

Source: Alu-tab product information pamphlet, AMH (2005), MIMS (Online)

DRUG FORMULARY

Drug generic name	<p>Calcitriol</p> <p>Product name: Citrihexal Manufacturer: Hexal Australia Pty Ltd</p>
Poison schedule	S4
Therapeutic class	6(g) Agents affecting calcium and bone metabolism
Preparation & Dosage	Capsules, 0.25 microgram (red/ orange, off white): 100's.
Routes	Orally
Administration frequency	<p><i>Uraemic osteodystrophy.</i> The recommended initial dose of Citrihexal is 0.25 microgram/day. If a satisfactory response in the biochemical parameters and clinical manifestations of the disease state is not observed, dosage may be increased by 0.25 microgram/day at intervals of two to four weeks.</p> <p>Most patients undergoing haemodialysis respond to doses between 0.5 and 1 microgram daily.</p>
Duration of order	Long term, titrate to monthly serum calcium, phosphate and parathyroid hormone levels
Action	<p>Calcitriol is the most important active metabolite of vitamin D₃. It is normally formed in the kidney from its precursor 25-hydroxycholecalciferol (25-HCC).</p> <p>In patients with marked renal impairment, synthesis of endogenous calcitriol is correspondingly limited or may even cease altogether. This deficiency plays a key role in the development of renal osteodystrophy.</p> <p>In patients with renal osteodystrophy, administration of calcitriol normalises reduced intestinal absorption of calcium, hypocalcaemia, increased serum alkaline phosphatase and serum parathyroid hormone concentration</p>
Indication for use	Treatment of hypocalcaemia in patients with uraemic osteodystrophy, hyperparathyroidism
Precautions	<p>Calcitriol may increase plasma phosphate levels. While this effect is desirable in hypophosphataemic osteomalacia, it may cause ectopic calcification, especially in patients with renal failure. Plasma phosphate levels should be kept normal in such patients by the oral administration of phosphate binding agents</p>
Contraindications	Hypercalcaemia; vit D toxicity; hypersensitivity to other vitamin D analogues

Interactions	Vit D; Ca, Mg (incl antacids); cholestyramine; corticosteroids; digoxin; thiazide diuretics; phosphate binding agents; hepatic enzyme inducers
Adverse reactions	Hypercalcaemia; drowsiness; weakness; nausea; diarrhoea; constipation; pruritus; renal toxicity; ectopic calcification; dehydration; hypersensitivity reactions in susceptible individuals
Storage	Bottles: store below 30 deg. C; blisters: store below 25 deg. C. Protect from light. Keep out of reach of children. Due to the use of a natural colouring agent, discolouration of capsules may occur. This does not affect the quality of the product.

Source: Citrihexal product information pamphlet, AMH (2005), MIMS (Online)

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