Learning Package

Health
Hunter New England
Local Health District

Renal: Renal Bone Disease

Sites where Learning package applies	Clinical areas where care is provided to patients with renal disease
Description	This learning package explores renal bone disease and its relationship with Chronic Kidney Disease (CKD) and End Stage Kidney Disease (ESKD) patients.
Target audience	Nephrology and Non-nephrology clinical staff who provide care to people who have or are at risk of developing renal bone disease.
Learning Outcomes, On completion of this package you will be better able to:	• Understand the concept and clinical manifestations of renal bone disease • Be aware of the targets set by the CARI Guidelines for renal bone disease • Initiate nursing interventions to assist in the treatment of complications associated with renal bone disease • Provide patient education around the treatment of renal bone disease
Keywords	Renal; Bone Disease; Dialysis; CKD; MBD
Document registration number	LP2018:08
Replaces existing document?	Yes

Related Legislation, Australian Standard, NSW Ministry of Health Policy Directive or Guideline, National Safety and Quality Health Service Standard (NSQHSS) and/or other, HNE Health Document, Professional Guideline, Code of Practice or Ethics:

- Infection Prevention and Control Policy [PD2017_013]
- NSW Health Policy PD 2005_406 Consent to Medical Treatment http://www.health.nsw.gov.au/policies/PD/2005/pdf/PD2005_406.pdf
- Renal Guidelines and Procedures http://ppg.hne.health.nsw.gov.au/

Is this package recorded in MyHealth Learning?	Renal: Renal Bone Disease (177725384)
Learning package contact person & contact details:	Gemma Fogarty ACNC Nephrology Department Ext 48815 Gemma.Fogarty@hnehealth.nsw.gov.au Lynn Brown CNE Nephrology Department Speed Dial 68396 Lynn.Brown@hnehealth.nsw.gov.au
Date authorised by Learning package committee:	14/02/2018
Issue date	2018
Review date	2021

Renal: Renal Bone Disease

Learning Package

2018

NSW GOVERNMENT Health
Hunter New England
Local Health District

Learning Package Overview

Purpose: *This package is designed to provide baseline information and to guide staff through the resources on renal bone disease. It will be useful for both Enrolled Nurses and Registered Nurses working in the area of nephrology nursing as well as those who would like to revise their knowledge on the subject*.

Revised and Updated by:
Gemma Fogarty (ACNC Nephrology Dept. JHH) &
Lynn Brown (CNE Nephrology Dept. JHH)

Peer reviewed by:
Leanne O'Grady ((CNC Renal Service Manning Hospital)
Cheryl Wertheim (Dialysis Nurse Educator Peel, Mehi & Tablelands Sectors)
Kim McNamara (CNC Renal Service Peel, Mehi & Tablelands Sectors)

Date: 19/01/2018

Acknowledgements: This learning package is an adaptation of original work by:
- Ginger Chu 2012 (Renal CNC/Lower Hunter & Greater Newcastle sector).
- Gemma Fogarty 2012 (Renal CNE Nephrology Dept. JHH).

Date for Learning Package Review: 2020

Contact details: Gemma Fogarty ACNC Nephrology Department Ext 48815
Gemma.Fogarty@hnehealth.nsw.gov.au

Lynn Brown CNE Nephrology Department Speed Dial 68396
Lynn.Brown@hnehealth.nsw.gov.au

Table of Contents **Page**

Introduction

This package is one of a suite of packages aimed at offering guided learning for nephrology nurses. The package aims to enhance theoretical knowledge leading to improved clinical practice as they care for patients who are at risk and those with renal bone disease.

Disclaimer

This learning package has been prepared by health professionals employed in Hunter New England Local Health District in the Renal Services. While all care has been taken to ensure that the information is accurate at the time of development, the authors recommend that all information is thoroughly checked before use if utilised by another unit, context or organisation.

Naming Convention

Renal: Renal Bone Disease

Aim

To enhance knowledge of Nephrology and Non-Nephrology clinical staff who provide care to patients who have or are at risk of developing renal bone disease.

Learning Outcomes or Learning Objectives

Completion of this learning package will enable the learner to:

- *Understand the concept and clinical manifestations of renal bone disease*
- *Be aware of the targets set by the CARI Guidelines for renal bone disease*
- *Initiate nursing interventions to assist in the treatment of complications associated with renal bone disease*
- *Provide patient education around the treatment of renal bone disease*

Pre-requisites

There are no formal prior learning requirements to undertake this package. However this forms part of the learning pathway for a nephrology nurse following completion of

- Haemodialysis or Peritoneal Dialysis SDLP
- Renal Anatomy and Physiology SDLP

Learning Package Outline

The package is designed to be a self-directed learning experience that will guide you through the literature and clinical issues related to renal bone disease.

This package is developed within an adult learning framework so not all activities need to be documented but it is expected that you will complete them in order to facilitate your learning.

Problem based learning

This program is based on a problem-based approach to learning. This approach has been chosen to enhance critical thinking, and to create a body of knowledge that the learner can apply to practice.

The package is developed with an adult learning framework so not all activities need to be documented but it is expected that you will complete them to facilitate your learning.

Instructions for participants

- It is estimated it will take an average of 4 hours to complete this package.
- Completion of this package is equivalent to Continuing Professional Development (CPD) hours which is a requirement for National Registration. Evidence of CPD can be generated using the reflection on learning page at the end of the package.
- Self-directed learning will be required to complete this package. Some activities will include essential reading and others will have additional supplementary readings that participants can undertake to further consolidate their knowledge.
- A brief outline of the topic followed by recommended readings & learning activities that will reinforce key points guide participants study.
- There is a suggested reference list but it is by no means complete. Please read widely to facilitate your learning. Journal articles can be accessed through CIAP. The online readings are not provided within this document due to copyright law restrictions. If you have any difficulty locating the readings please seek assistance from your relevant NE/CNE/CNS/CNC or hospital library.

This SDLP uses the following icons:

	**READING** **This icon alerts you to undertake reading related to the topic, which may include Guidelines and Procedures, Journal Articles or Books**
	**LEARNING ACTIVITY** **This icon denotes a learning activity or competency assessment that you will need to complete**
	**GUIDELINES** **This icon alerts you to the presence of a guideline or procedure related to the subject**

Assessment process

When completed, you can return the package to the relevant NE/CNE/CNC/CNS who will discuss it with you.

Reflection tool

At the completion of the learning package there is a reflection form that will assist you in reflecting on the package and how it meets your professional development needs.

Evaluation

A Learning Package Evaluation form when you have completed this package is found on page 22. You will need to return this to the relevant CNE/NE/CNC/CNS. This form is used to inform future updates and modifications of the learning package according to ongoing feedback from the user.

Overview of Renal Bone Disease

- *Understand the concept and clinical manifestations of renal bone disease*

Renal bone disease occurs secondary to changes in mineral metabolism and bone structure and occurs to some degree in all patients with Chronic Kidney Disease (CKD). Renal bone disease can be slowed or perhaps even prevented with early intervention using dietary measures and medications such as phosphate binders and Calcitriol (activated vitamin D). As a result, it is important that nurses working with patients with renal dysfunction have a good understanding of the underlying principles of renal bone disease and its management.

As renal disease progresses, the kidneys are less able to remove phosphate, leading to higher than normal phosphate levels. High phosphate levels stop the conversion of Vitamin D to its most active form (1, 25 Dihydroxycholecalciferol) in the kidneys (Thomas, 2014). Active Vitamin D is involved in regulating the serum level of calcium, by influencing the absorption of calcium from food in the intestines (Thomas, 2014). Levels of calcium in the blood therefore tend to fall when there is not enough active Vitamin D. A low serum level of calcium is a major stimulus to parathyroid hormone (PTH) production. In renal patients, this is known as secondary hyperparathyroidism. The parathyroid glands are 4 small glands in the neck next to the thyroid gland. Parathyroid hormone is released from these glands and the calcium level in the blood rises due to the action of PTH on bone and the kidneys (Thomas, 2014). Secondary hyperparathyroidism develops early in renal failure, even before the need for dialysis and is a "silent disease", just like blood pressure, because it is often unrecognised until symptoms occur. However, side effects of this imbalance (with elevated PTH and phosphate levels, and normal or low calcium levels) can cause itching, loss of mineral from the bones, pain, fractures and calcium deposits in blood vessels.

Normal Calcium and Phosphate

The kidneys play an important function in the maintenance and structure of healthy bone mass due to their role in balancing plasma calcium and phosphorus levels.

Calcium

Calcium is important for muscle and nerve function as well as the formation of bone. Less than 1% of total body calcium is in the extracellular fluid (ECF) the rest being within the bones.

There are three forms of ECF calcium (Grossman, 2014)

1. Ionised/free serum calcium (50%)
2. Non-ionised/complexed (bonded to Cl, PO4, and Citrate) (10%)
3. Bound to protein (Albumin) (40%)

Calcium is absorbed in the gastrointestinal tract (GIT). Approximately 40% of oral intake is absorbed and 15% of intake is then secreted into the intestine as part of digestive fluid. Hence only 25% of consumed calcium is absorbed. The kidneys freely filter ionised calcium. Most of the calcium that is filtered is reabsorbed, so balance is maintained in normal adults.

Phosphate

Phosphate is used in the cellular process to provide energy to cells by combining with lipids to form the cell membrane, deoxyribonucleic acid (DNA) and adenosine triphosphate (ATP). It also combines with calcium required for bone formation. Phosphate also acts as an acid-base buffer as it picks up hydrogen ions in the renal tubule (Thomas, 2014). Approximately 90% of phosphate is filtered via the kidneys (Thomas, 2014).

LEARNING ACTIVITY

1. What is the major role of calcium in the body?

_____

_____

_____

_____

2. What are the major functions of phosphate in the body?

_____

_____

_____

_____

Bone is a dynamic tissue, which constantly undergoes growth and remodelling. Healthy individuals have a complex system of metabolic checks and balances. These are anchored by parathyroid hormone (PTH), active vitamin D and also serum calcium and phosphorus. Together, these factors control the rate and nature of bone growth. Anything that disrupts these cycles can lead to abnormal, weak bone that is prone to pain or even fracture.

Regulation of Calcium and Phosphate

Two major factors that regulate calcium and phosphate are

1. Parathyroid hormone
2. Vitamin D

Parathyroid Hormone (PTH)

PTH is secreted by 4 parathyroid glands (Thomas, 2014). Secretion of PTH is increased by a fall of ionised calcium and decreased by a rise in ionised calcium (Thomas, 2014). The major effect of PTH is on bone.

Actions:

1. Releases calcium and phosphate from bone
2. Increases renal tubule reabsorption of calcium
3. Increases renal tubule excretion of phosphate
4. Stimulates the production of Vitamin D

(Thomas, 2014)

Vitamin D

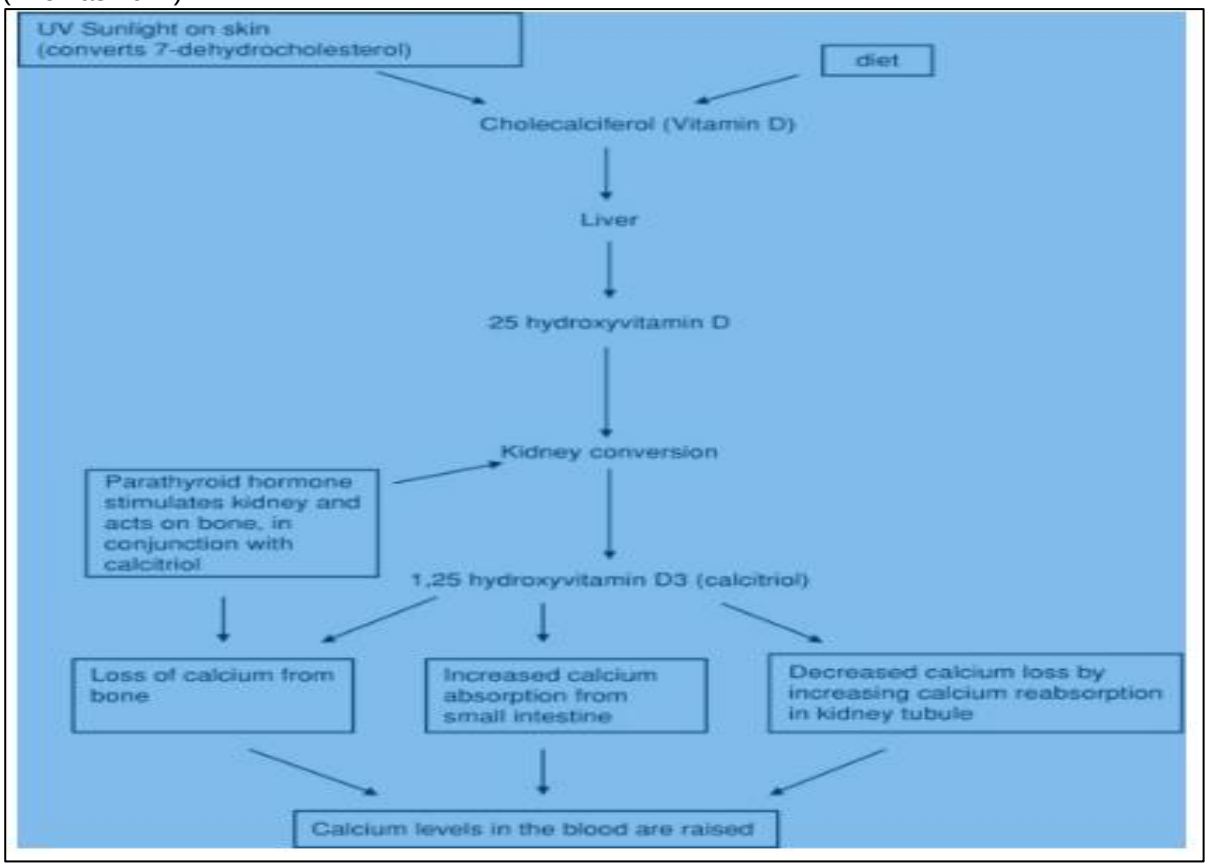
Vitamin D (Cholecalciferol) is a hormone obtained from food or produced by ultraviolet light on skin. In the kidney vitamin D is activated into 1,25 dihydroxycholecalciferol (calcitriol) (Thomas, 2014). Without this action of the kidney Vitamin D cannot work. Vitamin D must be activated. Low phosphate, low calcium and PTH activate Vitamin D. Vitamin D is inhibited by hyperphosphataemia. Hence, activated vitamin D regulates calcium and phosphate absorption from the GIT. If serum calcium or phosphate levels are low absorption is increased, if calcium or phosphate levels are high absorption is decreased (Thomas, 2014).

Actions:

1. Enhances Ca and PO4 from GIT
2. Stimulates Ca release from the bones via actions of PTH
3. Increases Ca reabsorption in renal tubules

(Thomas, 2014)

(Figure 1.) The actions of sunlight, vitamin D and parathyroid hormone (PTH) on blood calcium (Thomas 2014)



READING

1. 1. Hallock, A. (2017). Osteoporosis in patients with CKD: A diagnostic dilemma. Nephrology Nursing Journal, 44(1), 13-18. Retrieved from http://search.proquest.com.acs.hcn.com.au/docview/1870849150?accountid=130851
2. 2 Douglas C et al (2013) Chapter 10: Regulation of Calcium, Magnesium, and Phosphate.In: Vander's Renal Physiology, (8th edition) McGraw-Hill, New York, NY. http://accessmedicine.mhmedical.com.acs.hcn.com.au/content.aspx?bookid=2173&section

LEARNING ACTIVITY

3. What triggers PTH secretion?

_____

_____

_____

_____

4. What suppresses PTH secretion?

_____

_____

_____

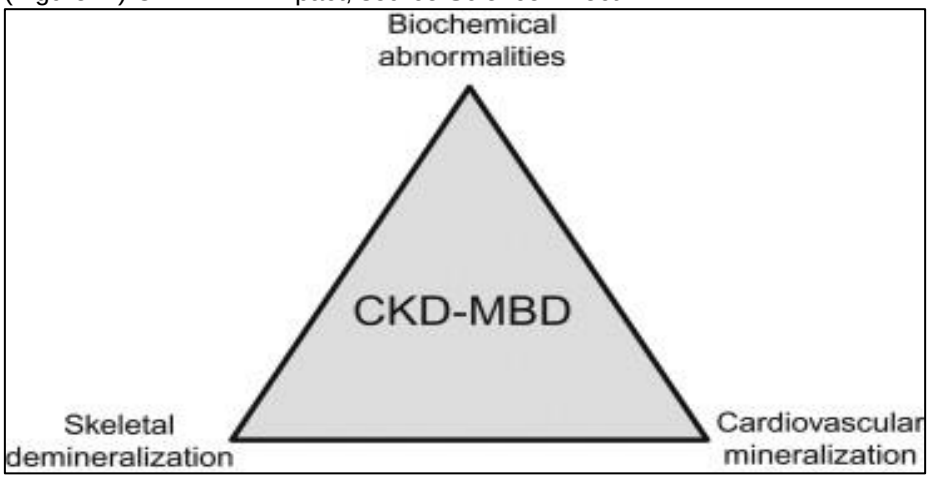
_____

Chronic Kidney Disease Mineral and Bone Disease (CKD-MBD)

- *Be aware of the targets set by the CARI Guidelines for renal bone disease*
- *Ability to initiate nursing interventions to assist in the treatment of complications associated with renal bone disease*
- *Ability to provide patient education around the treatment of renal bone disease*

Bone disease that occurs as a result of chronic kidney disease is termed as CKD-MBD. The bone disease process starts early in CKD and worsens as kidney function declines. Renal osteodystrophy occurs in advanced CKD/ ESKD (Hallock, 2017)

(Figure 2.) CKD-MBD impact, source Science Direct



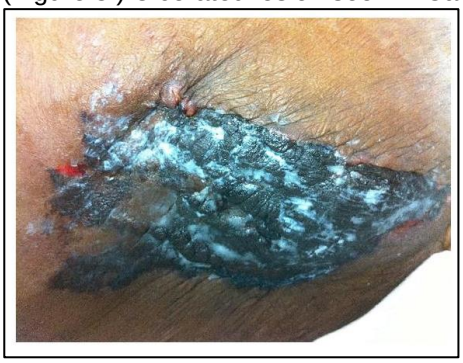
A continuous low serum calcium level and/or a high serum phosphate cause the parathyroid gland to hypertrophy and secrete high levels of PTH resulting in secondary hyperparathyroidism (Hallock, 2017). This is differentiated from primary hyperparathyroidism which occurs as a result of an enlargement of one or more of the

parathyroid glands Renal osteodystrophy occurs from secondary hyperparathyroidism and is referred to as high turnover bone disease as the formation and removal of bone is accelerated (O'Callaghan, 2016). Hence, bones become weaker, leading to bone frailty.

Secondary hyperparathyroidism causes bone to be formed so rapidly that it is not properly mineralised resulting in osteitis fibrosa. Fractures are uncommon, but muscle weakness, bone pain and itching may be severe. (Hallock, 2017).

Hyperphosphataemia can result in the serum phosphate precipitating with serum calcium, producing calcium phosphate crystals (calcium x phosphate product). These crystals deposit in soft tissue, brain, eyes, joints, blood vessels, heart valves, lungs and skin. This process is known as metastatic calcification (Yao et al, 2015) and it can present as Calciphylaxis (Carter, 2013)

(Figure 3.) Ulcerated lesion seen in Calciphylaxis



Osteomalacia results from the lack of mineralisation of newly formed bone resulting in decreased bone density and mass. This can be caused by metabolic acidosis as calcium from the bone is exchanged with hydrogen ions. Osteomalacia can also occur from aluminum replacing calcium in the bone from long term use of aluminum based phosphate binders. This type of renal osteodystrophy is referred as low turnover bone disease as bone formation is reduced (O'Callaghan, 2016). Fractures to ribs and femoral neck are common symptoms as well as severe bone pain and muscle weakness (Mathew et.al, 2014).
Adynamic bone disease is caused by an over suppression of PTH with Vitamin D analogue (e.g. Calcitriol) and Calcium based Po4 binders (e.g. Caltrate), resulting in the bone not actively being remodeled and causing hypercalcaemia; also referred as to low turnover bone disease (Hallock, 2017).

Other Types of Bone Disease

Bone Disease Following Transplantation

Following transplantation, prednisone, cyclosporine and tacrolimus can reduce bone density. Both calcitriol and bisphosphonates have been shown to reduce loss of bone density following kidney transplantation. Hormone replacement therapy may also be used in some patients to reduce loss of bone density, as well as adequate calcium intake, vitamin D and adequate nutrition and exercise. High calcium levels due to persistent hyperparathyroidism usually resolve after transplantation. If they do not resolve, post-transplant parathyroidectomy is sometimes necessary. Pre-existing bone disease usually improves if good renal function is achieved post-transplant.

Bone Disease Associated with Long Term Dialysis Treatment

A condition known as Secondary Amyloidosis can develop in those who have been on dialysis for many years. This is caused by the deposition of Beta 2 microglobulin (β2M) in the soft tissues and bone around joints. β2M is normally excreted by the kidneys. In the case of dialysis patients, it accumulates, as it is not completely removed during dialysis. Symptoms of amyloidosis, such as pain, stiffness and swelling around the joints, generally occur after 10 to 15 years of treatment, but can occur much earlier. The main joints affected are the hands, the shoulders and the spine, in that order of frequency. The radial nerve to the hand can be compressed at the wrist, as it runs through the carpal tunnel and is termed carpal tunnel syndrome. This can be released through a relatively simple surgical procedure.

Currently, improved dialysis membranes are more efficient in removing β2M. These membranes referred to as high flux are able to remove β2M more effectively than previous low flux membranes. β2M is approximately 11800 Daltons (molecular weight) and therefore not removed by low flux dialyser membranes which have a molecular weight cut off around 5000 Daltons (Daugirdas, Blake & Ing, 2015).

READING

3. Mathew, A. T., Hazzan, A., Jhaveri, K. D., Block, G. A., Chidella, S., Rosen, L., Fishbane, S. (2014). Increasing hip fractures in patients receiving hemodialysis and peritoneal dialysis. *American Journal of Nephrology, 40*(5), 451-457.

4. Robbins, K. C. (2017). Journal club: Read it, share it. Nephrology Nursing Journal, 44(4), 361-362. Retrieved from http://search.proquest.com.acs.hcn.com.au/docview/1929678556?accountid=130851

LEARNING ACTIVITY

5. What are the signs and symptoms of high turnover bone disease?

_____

_____

_____

_____

6. What is metastatic calcification and how does it develop?

_____

_____

_____

_____

LEARNING ACTIVITY

7. What is the formula for corrected calcium?

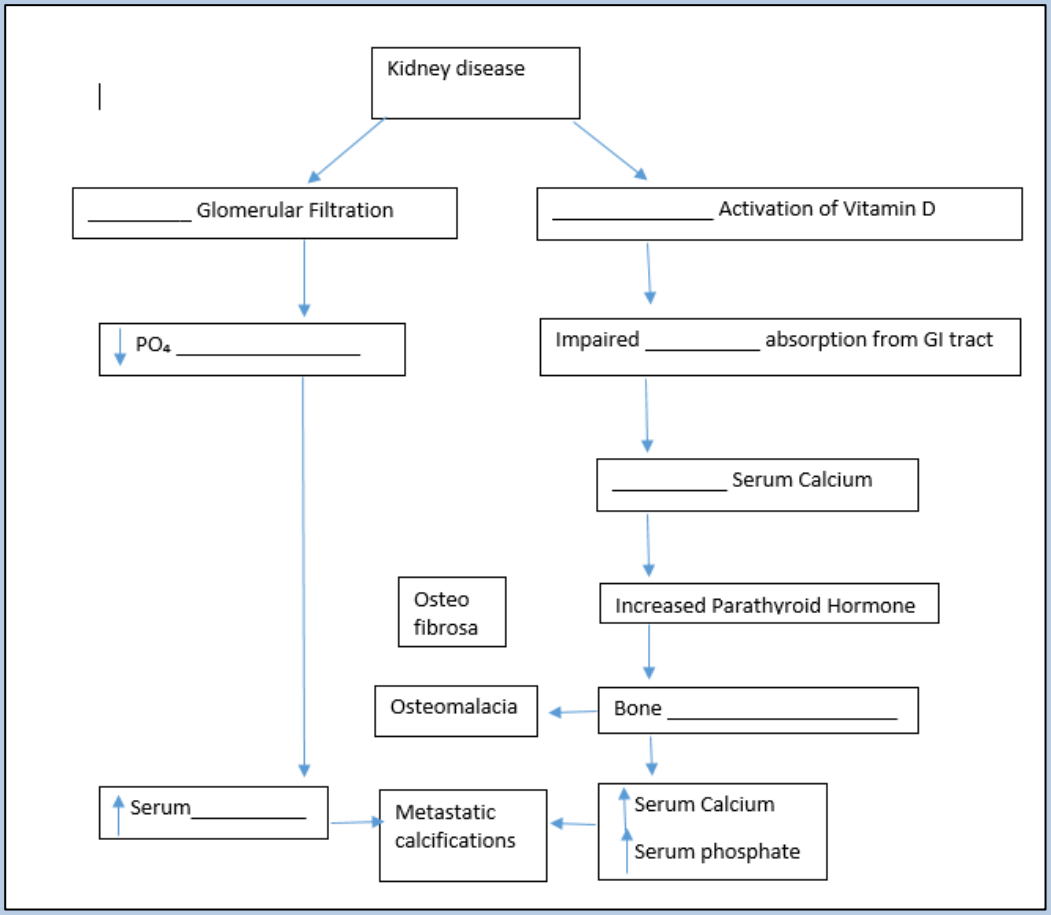
_____

_____

_____

_____

8. Fill in the missing words:



Treatment of Renal Bone Disease

Choice of dialysate

There seems to be no clear indication of the optimum dialysate calcium level. Current dialysate calcium levels seem to have been arrived at by historical means and are no more than an educated guess.

Our dialysate calcium range is from 1.25mmol/L to 1.75mmol. We should avoid low calcium e.g. < 1.5 if the patient's calcium is low or the patient has known cardiac disease or is unstable during dialysis. Patients who have had a parathyroidectomy or are on Cinacalcet usually require higher calcium. Adjusting the calcium dialysate concentration should be considered a short-term measure. The underlying cause of the hypo/hypercalcaemia should be identified and treated.

Diet

Phosphate is found naturally in most foods; unfortunately many foods high in protein are also high in phosphate. Phosphate is also found in processed foods e.g. baked products and cola drinks. Patients who have a high phosphate should be referred to the renal dietitian for review and education as maintaining adequate dietary intake while reducing dietary phosphate is a challenge.

Medications

Activated Vitamin D

For example:

- Calcitriol.

Administration of activated Vitamin D in CKD patients enhances calcium absorption in the small intestine, increasing serum calcium levels. Additionally, two mechanisms result in lower serum levels of PTH. First it inhibits PTH secretion at the messenger RNA involved in the synthesis of parathyroid cells. Secondly, it lowers the calcium – PTH set point (the serum calcium level at which 50% of PTH secretion is shut off).

Phosphate Binders

- Calcium based:

For example:

- Calcium Carbonate (Caltrate).

Calcium binds with phosphate in food within the gastrointestinal system and is then excreted in the faeces. This serves to lower serum phosphate levels. These phosphate binders must be taken with food otherwise they serve only as calcium supplements. The amount of phosphate within the meal will influence the amount of calcium bound. All unbound calcium will be absorbed in the gut raising serum calcium levels. Patients taking calcium compounds outside of their meal times are at increased risk of becoming hypercalcaemic. However, some patients are advised, notably post parathyroidectomy, not to take them at meal times and in this instance they are used as a calcium supplement rather than as a phosphate binder.

- Aluminium based:

For example:

- Aluminium hydroxide (Alutabs)

Aluminium binds with phosphate in the GIT and is then excreted in the faeces. It is given with meals with the aim of binding dietary phosphate in the bowel and preventing its absorption. This form of phosphate binder is largely avoided if possible due to the potential for aluminium toxicity due to systemic absorption of aluminum. Some physicians avoid such preparations due to possible aluminium toxicity. Aluminum toxicity can be evidenced through aluminum bone disease (osteomalacia), dementia, myopathy and anaemia.

- Aluminium/Magnesium based:

For example:

- Gastrogel
- Mylanta tables
- Aluminium hydroxide
- Magnesium hydroxide

Aluminium and magnesium binds with phosphate in the bowel and is then excreted. Any that is not excreted is absorbed. Mixtures of aluminium and magnesium have been used with the goal of limiting the aluminium dose and its adverse effects. The mixture however is probably less effective than pure aluminium. The use of lower dialysis solution magnesium may not be adequate in preventing magnesium loading.

Other Preparations

- Non Calcium/Aluminium based:

For example:

- SEVELAMER; Renagel.

Renagel is a relatively new phosphate binder that does not contain calcium or aluminum. It is a cationic polymer that binds phosphate in the bowel through ion exchange and hydrogen bonding and also helps to lower cholesterol levels by lowering LDH cholesterol. A major advantage is the reduced risk of hypercalcaemia relative to the use of calcium containing phosphate binders. Therefore it is especially useful for patients with hyperphosphataemia who also have hypercalcaemia.

Calcinimetics

For example:

- Sensipar/Cinacalcet.

Cinacalcet specifically targets and modulates the calcium sensing receptor (Ca R) on the parathyroid glands mimicking the effect of blood ionised Ca, suppressing PTH secretion. Through the suppression of PTH levels there is no induced hypercalcaemia or hyperphosphataemia. When patients are commenced on Sensipar they should have weekly calcium and monthly PTH levels. When stable monitor calcium monthly and PTH 1-3 monthly.

Surgical Management: Parathyroidectomy

Sometimes, the parathyroid glands will not decrease the production of PTH, regardless of medical management. This condition is called refractory hyperparathyroidism or tertiary hyperparathyroidism if hypercalcaemia is present. If the PTH level is persistently greater than ten times the normal level and severe symptoms of bone disease are evident the patient should be considered for parathyroidectomy. Calcification of blood vessels in the skin with ulceration (calciphylaxis) is also a strong indication for removal of these glands.

Hungry Bone Syndrome is common post-operatively following a parathyroidectomy. It includes sudden falls of calcium and phosphate plasma concentrations in the first hours or days post operatively. The higher the ALP the more likely a severe and more prolonged the fall in calcium will occur.

Patients with hungry bone disease will need replacement with either intravenous calcium or oral calcitriol and calcium dependent on the patient's pathology.

Post parathyroidectomy nursing management

Post parathyroidectomy the patients requires close monitoring for at least the first week or until calcium levels stabilise.

Immediate post-operative management includes:

- Monitoring for signs of hypocalcaemia including ECG changes (prolonged QT interval, bradycardia, ventricular tachycardia or *asystole),* cramps, and numbness and tingling in fingers and toes, paresthesia around the lips and mouth, Chvostek's and Trousseau's signs This includes measurement of serum calcium and albumin six and twelve hours post-operatively. Thereafter, calcium should be measured daily until discharge.
- Consideration must be given to adjusting dialysate calcium concentration to a higher level

The dialysis nurse is expected to monitor and report adverse blood levels on all patients but more regular surveillance of patients who have had a parathyroidectomy is required.

GUIDELINES

Locate and read: the guideline applicable to your unit on parathyroidectomy in renal patients

LEARNING ACTIVITY

9. What is the risk associated with the use of magnesium phosphate binders?

_____

_____

_____

_____

10. When is the best time to take phosphate binders and why?

_____

_____

_____

_____

11. What are the indications for a parathyroidectomy?

_____

_____

_____

_____

12. Describe the mechanisms of Hungary Bone disease

_____

_____

_____

_____

LEARNING ACTIVITY

13. You are caring for a patient who had just had a parathyroidectomy. What nursing management considerations do you need to be aware of?

_____

_____

_____

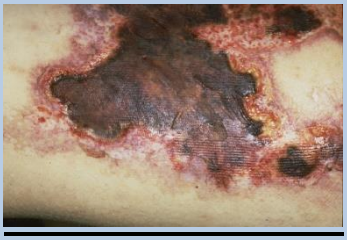
_____

14. What are the mechanism of action for the following medications?

Drug	Action
Aluminium hydroxide	
Calcium carbonate	
Calcitriol	
Sensipar	

LEARNING ACTIVITY

15. What is depicted in this image? Why has this occurred and how could it have been prevented?



_____

_____

_____

_____

_____

References

Carter, T., & Ratnam, S. (2013). Calciphylaxis: A devastating complication of derangements of calcium-phosphorus metabolism - A case report and review of the literature. Nephrology Nursing Journal, 40(5), 431-5.

Daugirdas, J.T., Blake, P.G., & Ing, T.S. (2014). *Handbook of Dialysis* (5th edition). Philadelphia: Lippincott Williams & Wilkins.

Eaton, D.C. & Pooler, J.P. (2013) Chapter 10: Regulation of Calcium, Magnesium, and Phosphate. In: *Vander's Renal Physiology,* (8th edition) McGraw-Hill, New York, NY.

Grossman, S. (2014) Porth's Pathophysiology: concepts of altered health states Ninth edition.

Hallock, A. (2017). Osteoporosis in patients with CKD: A diagnostic dilemma. *Nephrology Nursing Journal, 44*(1), 13-18

Mathew, A. T., Hazzan, A., Jhaveri, K. D., Block, G. A., Chidella, S., Rosen, L., Fishbane, S. (2014). Increasing hip fractures in patients receiving hemodialysis and peritoneal dialysis. *American Journal of Nephrology, 40*(5), 451-457.

O'Callaghan, C. (2016). *The Renal System at a Glance* (4rd edition). UK: Wiley-Blackwell

Robbins, K. C. (2017). Journal club: Read it, share it. *Nephrology Nursing Journal, 44*(4), 361-362.

Scialla, J. J., & Wolf, M. (2015). When there will never be a randomized controlled trial. *Kidney International, 88*(2), 220-222.

Thomas, N. (2014). *Renal Nursing* (4th edition). London: Bailliere Tindall

Yao, L., Sun, Y., Sun, W., Xu, T., Ren, C., Fan, X., Wang, L. (2015). High phosphorus level leads to aortic calcification via beta]-catenin in chronic kidney disease. *American Journal of Nephrology, 41*(1), 28-36.

Learning Package: Reflection on Learning

This document guides your reflection on the extent to which the package meets your professional development needs, and how you plan to apply your learning into practice. This tool is not part of the assessment process, and has been included as a document that you may wish to include in your professional portfolio. Time taken to complete learning package_____

What was your purpose in completing this learning package?

_____

_____

_____

_____

_____

_____

Did you achieve this by completing the learning package?

_____

_____

_____

_____

_____

_____

_____

Reflecting on the content, what key learning have you obtained?

_____

_____

_____

_____

_____

_____

_____

What learning will you apply to your practice immediately? How will you do this?

_____

_____

_____

_____

_____

_____

What learning needs have you identified as a result of completing this learning package?

_____

_____

_____

_____

How do you plan to address these needs?

_____

_____

_____

_____

_____

Signature:_____ Date:_____

Learning Package Evaluation Form

Your feedback regarding this learning package is important to ensure the package meets your learning needs. Please take 5 minutes to answer the following questions to facilitate any change required for future learning packages.

1. The learning outcomes of the learning package were clearly identified	Yes	No
2. The learning outcomes of the package were appropriate	Yes	No
3. The content provided enabled me to meet the learning outcomes?	Yes	No
4.The activities motivated my interest in the topic	Yes	No
5.The activities and workbook questions supported my understanding of the topic	Yes	No
6.The package was presented in a logical manner	Yes	No
7.The assessment process related to this package was clearly outlined (if applicable)	Yes	No

8. My most relevant learning outcomes from this package were:

_____
_____

9. The key learning points from this package I can immediately apply to practice include:

_____
_____

10. The least relevant component(s) of this package were:

_____
_____

11. Some suggestions I would like made to improve the package would be:

_____
_____

12. Further comments:

_____
_____

Thank you for your time to complete the evaluation
Please return to:

The relevant CNE/NE/CNC/NP within your area