

The Renal Pharmacist



2018

Issue 1

View from the Chair

2017 has been another successful year for the RPN. The RPN hosted education events in Montreal, Calgary, and Toronto. In an attempt to include renal pharmacists from coast to coast, web broadcasting was used for the November 2017 Toronto RPN education event. For those of you interested, the RPN website has a library of many of the presentation slides from these past events. This year, the RPN signature education day will be held in Vancouver in conjunction with the CSN 2018 AGM (May 3-5th), which happens to be celebrating its 50th anniversary. As well, of special note, the 17th Congress of the International Society for Peritoneal Dialysis will be held immediately following CSN. The RPN education coordinators, Judith and Jenny have been working incredibly hard and are organizing a top notch RPN education day for May 4th- you don't want to miss it. Check the RPN website for registration and updates.

Joanne Breckles from Toronto East General Hospital and Kathryn Haubrich from B.C. Children's Hospital join the RPN executive as our secretary/treasurer and chair elect, respectively. Departing from the RPN executive are Cali Orsulak, Past RPN Chair and Julianne Kim, secretary/treasurer. Huge thank you to both of you for your dedication to the RPN

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and years of volunteer work- we will miss you.

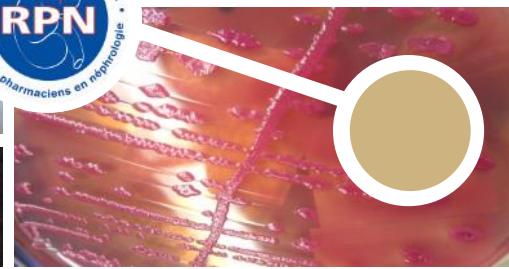
I would like to thank the RPN executive members, Clifford, Jenny, Judith, Marisa, Elaine, and Andrea for your amazing commitment to the network, so many hours spent planning education events, publishing newsletters and maintaining our website. The aforementioned activities keep renal pharmacists connected across Canada and supports our pursuit to provide excellent renal care to our patients. I feel fortunate to be part of this dream team!

On a final note, we are always looking for colleagues to join the RPN Executive Committee. Please let us know if you are interested in getting more information.

Hoping to see all of you at the RPN education day in May 2018 in Vancouver.

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BScPharm, ACPR, PharmD
Chair, Renal Pharmacist Network 2018



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SGLT2 Inhibitors and cardiorenal complications in type 2 diabetes

Author: Andrea Narducci-Swanson, BScPhm, ACPR, PharmD

On November 27, 2017, the Toronto members of the RPN hosted an educational evening discussing the sodium–glucose cotransporter (SGLT) 2 inhibitor class of antihyperglycemics. We were delighted to have Dr. David Cherney, MD, PhD as our guest speaker. Dr. Cherney is an Associate Professor in the Department of Medicine, University of Toronto and a Clinician Scientist at the University Health Network and Mount Sinai Hospitals, where he is the director of the Renal Physiology Laboratory.

The evening started with a review of diabetic kidney disease and the pharmacology of SGLT2 Inhibitors. Under normal physiological conditions, nearly all filtered glucose undergoes renal tubular reabsorption, and this process is enhanced in diabetes.¹ Two distinct sodium-dependent transport systems, SGLT1 and SGLT2 have been characterized. SGLT2 transporters are located almost exclusively in the proximal tubular epithelial cells of the kidneys and are responsible reabsorbing greater than 90% of filtered glucose.² The remainder is subsequently reabsorbed by SGLT1 in the more distal tubular segments. SGLT1 transporters are also located in cells isolated from the intestine, heart and skeletal muscles. SGLT2 inhibitors decrease the capacity of the renal tubules to reabsorb glucose, resulting in glucosuria and reduced plasma glucose concentration. There are three SGLT2 inhibitors available on the Canadian market: Canagliflozin (Invokana®), Dapagliflozin (Forxiga®) and Empagliflozin (Jardiance®).

SGLT2 inhibitors effectively lower hemoglobin A1c level (HbA1c) by approximately 0.7% versus placebo.³ The ability to lower HbA1c is attenuated in patients with renal dysfunction, however SGLT2 inhibitors have a variety of favourable nonglycemic effects including renal protective pathways. Dr. Cherney reviewed the potential mechanisms through which this class of medications lowers blood pressure (BP), modulates renal hemodynamics, reduces albuminuria and lowers uric acid. Across studies with the various SGLT2 inhibitors in patients with type 2 diabetes mellitus (DM2), there was a ~5mmHg reduction in systolic BP and a ~2 mmHg reduction in diastolic BP.¹ SGLT2 inhibitors do induce weight loss but this is not thought to be

the main mechanism by which they lower BP. The current hypotheses are that changes in plasma volume and reduced arterial stiffness are the main factors contributing to the BP-lowering effects of the SGLT2 inhibitors.

Upon initiation of SGLT2 inhibitors, there is an acute reduction in eGFR by approximately 5 ml/min/1.73 m²; however, renal function then trends back to baseline and remains stable thereafter.² There is also a consistent 30%–40% reduction in albuminuria across studies. The initial effect on GFR led to concern that these medications may increase the risk of acute kidney injury (AKI), but this was not demonstrated in the results of 2 major clinical trials that considered the impact on long-term kidney outcomes (discussed below).⁴ The glycosuric effects of SGLT2 inhibitors are dependent on glomerular filtration, and thus are diminished in patients with a reduced GFR; however, the BP and albuminuria lowering effects, and impact on eGFR are preserved in patients with CKD. SGLT2 inhibitors have been compared to renin-angiotensin-aldosterone system (RAAS) blockers; however contrary to these agents, the initial decline in eGFR observed with SGLT2 inhibitors is likely related to afferent arteriolar vasoconstriction through a tubuloglomerular feedback mechanism.² SGLT2 inhibitors cause proximal tubular natriuresis, which activates tubule-glomerular feedback through increased macula densa sodium delivery, leading to afferent arteriole vasoconstriction and a reduction in intraglomerular pressure.⁵ These data suggest that this class of diabetes



For more detailed information on this topic please see Dr. Cherney's article:

Sodium Glucose Cotransporter 2 Inhibitors in the Treatment of Diabetes Mellitus Cardiovascular and Kidney Effects, Potential Mechanisms, and Clinical Applications Circulation. 2016;134:752–772.

"We were very pleased to offer a free live stream of our Toronto Education Evening through WebEx and thank those members who joined us from across Canada."

medications may have a kidney protective effect, even in patients with reduced kidney function

At this point in the evening, Dr. Cherney then reviewed the two recent SGLT2 inhibitor cardiovascular outcome trials: the Empagliflozin Cardiovascular Outcome Event Trial in Type 2 Diabetes Mellitus Patients (EmpaReg Outcome) Trial and the Canagliflozin Cardiovascular Assessment Study (CANVAS) Program.^{6,7,8}

The EmpaReg Outcome Trial randomized 7020 patients with DM2 and high cardiovascular (CV) risk to placebo or empagliflozin (either the 10 mg or 25 mg dose) on top of standard care. The primary composite CV endpoint, consisting of CV death, nonfatal myocardial infarction (MI) and nonfatal stroke, was significantly reduced in the group taking empagliflozin (both doses combined) compared to recipients of placebo (10.5% vs. 12.1%; hazard ratio [HR], 0.86; p<0.001 for noninferiority, p=0.04 for superiority). This was driven by a 38% reduction in CV death (p<0.001), without significant reductions in either nonfatal MI infarction or stroke. Empagliflozin therapy was also associated with a 35% relative risk reduction (p=0.002) of hospitalization for heart failure and a 32% relative risk reduction (p<0.001) of total mortality. Participants in the EmpaReg Outcome Trial had an eGFR of at least 30 ml/min/1.73 m², and the secondary, prespecified kidney outcomes were also reported.⁷ Empagliflozin reduced the end point of “new onset or worsening nephropathy” (macroalbuminuria, doubling of creatinine, initiation of RRT, or death from kidney disease). New or worsening kidney disease occurred less frequently (12.7%) in the empagliflozin group compared with placebo (18.8%) (HR, 0.61; 95% CI, 0.53–0.70; P<0.001).² The diuretic effects of SGLT2 Inhibitors (both natriuresis and osmotic diuresis) has been the main mechanism credited with the positive CV and renal outcomes.² However, there are a variety of other hypotheses and conceivably the results of the EmpaReg Outcome trial could be due to an overall beneficial patient profile: reduced BP and plasma volume, weight loss, and modest antihyperglycemic and uric acid–lowering effects.

The more recent CANVAS Program combined data from 2 trials involving a total of 10,142 participants with DM2 and high CV risk (in contrast to EMPA-REG, it also included a primary CV prevention cohort).⁹ Patients were randomized to receive canagliflozin 100 mg, 300 mg or placebo. The rate of the primary outcome (composite of CV death, nonfatal MI, or nonfatal stroke) was lower with canagliflozin than with placebo (HR, 0.86; 95% confidence interval [CI], 0.75 to 0.97; P<0.001 for noninferiority; P=0.02 for superiority). Kidney outcomes were prespecified and patients

allocated to canagliflozin had a lower risk of progression of albuminuria (HR, 0.73; 95% CI, 0.67 to 0.79) and the composite outcome of a sustained 40% reduction in eGFR, need for RRT, or death from kidney causes (HR, 0.60; 95% CI, 0.47 to 0.77). With regards to adverse reactions, they were consistent with those previously reported for other SGLT2 inhibitors except for an increased risk of amputation (6.3 vs. 3.4 participants per 1000 patient-years; HR, 1.97; 95% CI, 1.41 to 2.75); amputations were primarily at the level of the toe or metatarsal.

Despite the exciting and consistent results of the above-cited studies, neither the EmpaReg Outcome nor CANVAS trials were powered to assess kidney outcomes as primary endpoints. Therefore, although the renal analysis was prespecified, it was still exploratory and future studies are required for definitive information on the renoprotective potential of SGLT2 inhibitors. Of note, some trials are already underway [such as the Evaluation of the Effects of Canagliflozin on Renal and Cardiovascular Outcomes in Participants with Diabetic Nephropathy (CREDENCE) trial], and will provide important additional information regarding the safety and efficacy of SGLT2 inhibitors in people with both diabetic and nondiabetic kidney disease.

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Summary of therapeutic options in patients with diabetic kidney disease

Authors: Karen Ng, ACPR candidate and Judith Marin, B.Pharm, M.Sc., PharmD

Diabetes is the main cause of kidney disease in Canada (McFarlane). In the last few years, new diabetic therapeutic options have been approved by Health Canada for treatment of diabetes. The following is a comparison chart of these medications, which is intended to serve as a quick reference guide for healthcare providers. It contains information regarding the usual dosage ranges of the drugs, adjustments required in renal impairment, degree of A1c lowering effect, non-glycemic benefits, adverse effects, and names of landmark studies. I hope some of you will find this useful in your practice.

Medication name (brand name)	Usual dose range	Dosing adjustment for GFR (mL/min/1.73 m ²)			A1c lowerin g effect (%)	Non-glycemic benefits?	Adverse events	Landmark studies to support efficacy or safety	Cost (\$ per 100 days)		
		CKD 3 (30–59)	CKD 4 (15–29)	CKD 5 (<15)							
Biguanides											
Metformin (Glucophage, Glumetza)	1 g–2.55 g/day divided BID–TID	1–1.7 g/day (GFR 45–60); 500–850 mg/day (GFR 30–45)	Avoid use if GFR <20		1–1.5	↓ morbidity and mortality in obese patients	GI: nausea, diarrhea Lyses: lactic acidosis Heme: anemia (due to B ₁₂ deficiency) Endo: ↓ weight	UKPDS-34	\$20–31		
DPP IV inhibitors											
Linagliptin (Trajenta)	5 mg daily	No adjustment			0.4–0.8	N/A	CNS: headache HEENT: sore throat CV: potential worsening of heart failure GI: nausea, diarrhea, constipation Abdo: pancreatitis (rare) Endo: ↓ weight (linagliptin) Heme: possible ↓ lymphocytes MSK: joint pain	N/A	\$253		
Saxagliptin (Onglyza)	2.5–5 mg daily	2.5 mg daily (GFR <50)		Avoid use	0.4–0.8		SAVOR-TIMI 53	\$269–320			
Sitagliptin (Januvia)	25–100 mg daily	50 mg daily (GFR 30–49)	25 mg daily		0.5–1		TECOS	\$318			
GLP-1 receptor agonists											
Dulaglutide ³ (Trulicity)	0.75–1.5 mg weekly	No adjustment			0.8–1.6	N/A	AWARD clinical trials CNS: headache, dizziness GI: nausea, vomiting, diarrhea, constipation, dyspepsia Abdo: pancreatitis (rare) Endo: thyroid CA/tumours (liraglutide, dulaglutide), weight ↓	AWARD clinical trials	\$630		
Exenatide (Byetta, Bydureon)	Immediate release: 5–10 mg sc BID ac Extended release: 2 mg sc weekly	Immediate release: 5 mcg sc BID	Avoid use		0.9–1.5	No benefit in ↓ major adverse cardiac events		EXSCEL	\$468		
Liraglutide (Victoza)	0.6–1.8 mg sc daily	No dose adjusstment	Avoid use		0.8–1.4	↓ all-cause and cardiovascular mortality; ↓ new onset macroalbuminuria		LEADER	\$250–690		
Semaglutide (Ozempic) ⁴	0.25–1.0 mg sc weekly	No adjustment			1.3–1.5	No change in major cardiovascular event		SUSTAIN-6	\$270–550		

Sulfonylureas										
Gliclazide (Diamicron)	Immediate release: 80–160 mg BID Modified release: 60–120 mg daily	No adjustment	Decrease dose	Decrease dose or avoid use	1–1.5	N/A	CNS: headache GI: nausea, vomiting, diarrhea Endo: hypoglycemia (esp. glyburide), weight gain Derm: rash, photosensitivity	N/A	\$17–50	
Glimepiride (Amaryl)	1–4 mg daily			Max 1 mg daily or avoid use					\$67	
Glyburide (Diabeta)	5–15 mg/day divided BID		Avoid use						\$16–28	
Meglitinides										
Repaglinide (Gluconorm)	0.5–4 mg TID	No adjustment			1–1.5	N/A	CNS: headache HEENT: URTI, sinusitis GI: diarrhea MSK: arthralgia, backache	N/A	\$44–81	
SGLT2 inhibitors										
Canagliflozin (Invokana)	100–300 mg daily	100 mg daily; avoid if GFR <45	Avoid use	0.6–0.8	↓ major adverse cardiac events (non-fatal MI, non-fatal stroke, CV mortality)	General: dizziness, decreased SBP due to diuresis GI: nausea, constipation GU: genital fungal infections , UTI, increased urinary frequency, increased BUN/SCr Fluids & lytes: diuresis and volume depletion MSK: increased fractures and amputations (canagliflozin only)	CANVAS	\$300	No	
Dapagliflozin (Forxiga)	5–10 mg daily	Avoid use			0.4–0.6	N/A				
Empagliflozin (Jardiance)	10–25 mg daily	Avoid use if GFR <45			0.5–0.7	↓ major adverse cardiac events (non-fatal MI, non-fatal stroke, CV mortality); ↓ all-cause mortality; ↓ albuminuria and CKD progression				
Thiazolidinediones (TZDs)										
Pioglitazone (Actos)	15–45 mg daily	No adjustment			<1	↓ composite of all-cause mortality, non-fatal MI, and stroke	CV: edema, heart failure , hypertension Endo: weight gain Liver: ↑ ALT MSK: ↑ fractures	PROACTIVE	\$60–116	Yes – special authority required
Rosiglitazone (Avandia)	4–8 mg daily					No benefit in ↓ CV hospitalization or CV mortality		RECORD	\$192–262	No

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Denosumab (Prolia) for the Treatment of Osteoporosis in Dialysis Patients: What you should know to manage hypocalcemia following an injection

By: Becca Zhang (Pharmacy Student)

Summary

Denosumab (Prolia) is a highly effective antiresorptive agent for the treatment of osteoporosis. It is not renally cleared and can be used (60mg SC q6months) in all stages of chronic kidney disease (CKD), including in dialysis patients. The evidence for fracture prevention is derived from post-hoc analysis of clinical trials in mild-moderate renal impairment, but this has not been studied in dialysis patients.

There are **safety concerns associated with severe hypocalcemia (i.e. QTc prolongation, tetany)**. Current monograph recommendations (calcium monitoring at 2 weeks after the injection and if symptoms develop) do not address the risk of hypocalcemia in dialysis patients. In trials involving the use of denosumab 60mg Q6months in hemodialysis patients, **all patients developed hypocalcemia with a nadir occurring between 7 days – 2 months after each injection**. Literature recommends frequent monitoring post-injection and aggressive repletion of calcium by adjusting dosages of calcitriol, calcium carbonate, and increasing the dialysate calcium concentration. Pre-emptive changes in therapy can be considered, although it is unknown if such measures will prevent hypocalcemia. Hemodialysis practitioners should

be aware of the risks of hypocalcemia and its management in patients receiving denosumab for treatment of osteoporosis.

Rationale for Use

Patients on dialysis have an estimated two to four-fold higher fracture risk (Nickolas et al.) and higher risk of morbidity and mortality following a fracture compared to patients without CKD (Lin et al.). Multiple other conditions (e.g. osteoporosis) can also contribute to high fracture risk.

Diagnosing osteoporosis in men and women with advanced CKD is challenging and there is no consensus on the ideal approach. In addition, dialysis patients have disturbed mineral bone metabolism which can lead to extremes in bone turnover (adynamic bone disease to osteomalacia). Patients with high bone turn over may benefit from antiresorptive agents, however the use of antiresorptive agents in patients with adynamic bone disease is theoretically harmful, and thus it would be reasonable to exclude this condition before initiating denosumab.

The Kidney Disease: Improving Global Outcomes (KDIGO) guidelines updated its recommendations for the treatment of chronic kidney disease mineral bone disease (CKD-MBD).

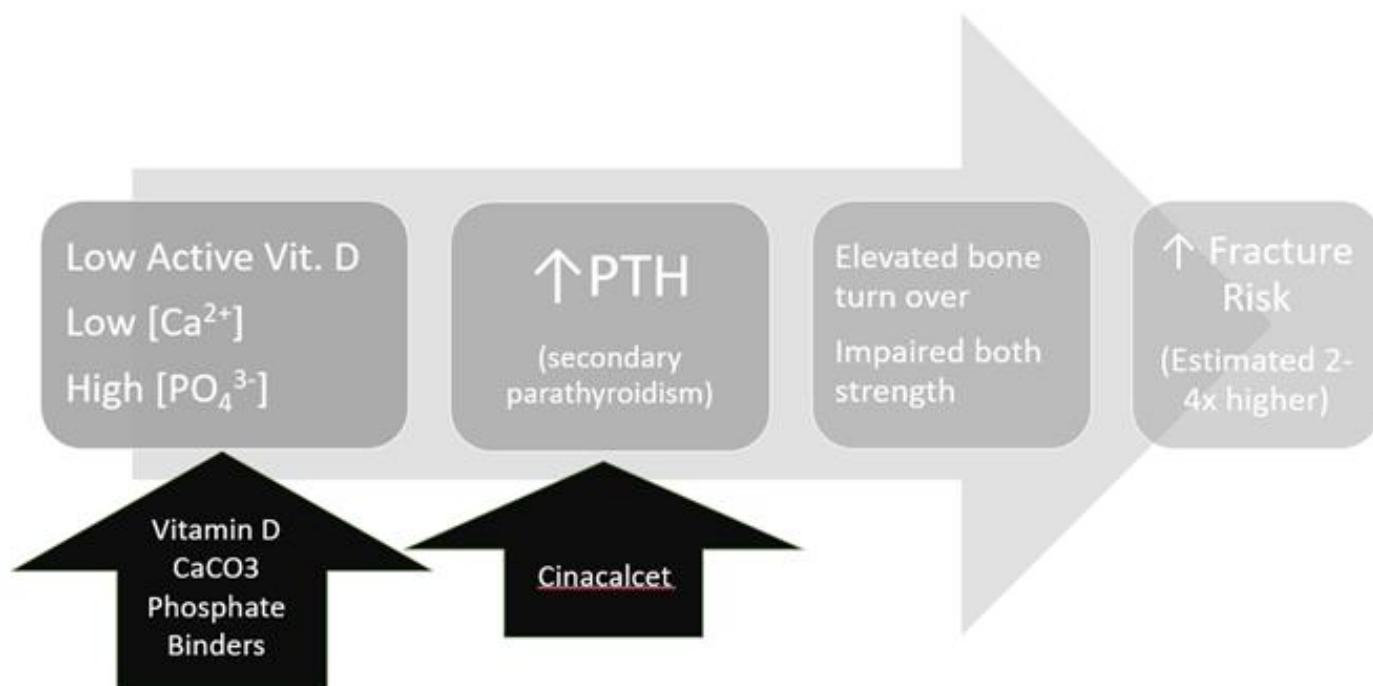


Figure 2. Secondary Hyperparathyroidism leading to High Bone Turnover Disease

The 2009 KDIGO guidelines recommended a bone biopsy prior to initiating antiresorptive agents for patients with CKD 4–5D with biochemical abnormalities, CKD-MBD, low BMD and or fragility fractures. In the recently published 2017 guidelines, there is new evidence to suggest that DXA BMD may predict fracture risk in patients with CKD 3a-5D. Therefore, **the lack of ability to perform a bone biopsy may not justify withholding therapy in patients with a high risk for fracture and in such patients trends in PTH should guide therapy.** A bone biopsy should still be considered with fluctuating PTH trends and if adynamic bone disease is suspected.

Evidence of denosumab's efficacy in CKD

Denosumab is a fully monoclonal antibody that prevents the binding of RANK to RANKL, thereby inhibiting the activity of osteoclasts and resulting in:

1. Reduced bone turn over
2. Rapid mineral deposition in bone matrix

In the pivotal phase 3 clinical FREEDOM trial (Jamal et al.), similar efficacy around improvements in BMD and fracture incidence was seen in patients with moderately reduced eGFR compared to patients with mild or normal renal function. However, no trials have assessed fracture risk in dialysis patients.

Denosumab is available as a 60mg subcutaneous injection typically given every 6 months. **It is not cleared renally and pharmacokinetic studies demonstrate that dose adjustments are not required at any stage of renal impairment** (Block et al.).

Safety Concern: Hypocalcemia

The rapid antiresorptive effect results in rapid reduction in bone resorption and the subsequent efflux of calcium from the circulation in the bones. In the dialysis population, elevated PTH helps to normalize low serum calcium levels by shifting skeletal calcium into the circulation which contributes to high bone turn over. This **pre-existing disturbance in mineral metabolism in CKD-MBD predisposes dialysis patients to hypocalcemia when they are given an antiresorptive agent.** This effect is similar to the “hungry bone syndrome” that is classically seen following a parathyroidectomy or with cinacalcet (Sensipar) use for the treatment of secondary hyperparathyroidism.

Case Reports of Hypocalcemia

There are five case reports describing severe hypocalcemia following Prolia injection for the treatment of osteoporosis in patients with CKD 5D (see Appendix, Table 1). In all cases hypocalcemia nadir occurred between 11 days – 7 weeks from denosumab injection and 3 out of 5 reports indicated

severe hypocalcemia resulting in tetany, QTc prolongation, or dyskinesia. Hypocalcemia was corrected via administration of IV calcium gluconate, adjustments in oral calcium carbonate, calcitriol and changing the dialysate calcium concentration.

Trials Involving Hemodialysis Patients

Three trials reviewed the use of denosumab for the treatment osteoporosis in CKD 5D patients. In all cases patients had well controlled mineral abnormalities (P, PTH, calcium) but there was a progressive and time-dependent decrease in serum calcium which occurred 7-20 days after each denosumab injection. Subsequently, a compensatory rise in PTH was seen, which normalized to baseline levels following the correction of serum calcium. Cases of hypocalcemia ranged from asymptomatic to severe and were corrected in all cases.

Dave et al.’s trial examined 13 patients with CKD4-5 treated with denosumab. Severe hypocalcemia was observed in 8/13 patients (5/7 HD patients) including vasculitis rash, seizure, pulmonary edema, and prolonged QTc.

Festuccia et al.’s retrospective trial involved 12 hemodialysis patients with prior history or high risk of fractures who were monitored over 24 months. In this trial, three patients developed symptomatic hypocalcemia (myalgia, paresthesia) although none required hospitalization.

Lastly, Hiramatsu et al. conducted a prospective study involving 11 HD patients with osteoporosis. All patients were placed on active Vitamin D prior to denosumab injection and received daily calcium monitoring for 10 days. Although a calcium nadir was reached in all patients (the lowest being 1.38mmol/L), no patients had symptoms of hypocalcemia. Furthermore, BMD T-scores improved after 6 months from the baseline at the lumbar spine (-2.7 to 1.7, p=0.006) and femoral neck (-2.4 – 2.3, p=0.007). Interestingly 4 patients were on cinacalcet (Sensipar) at the beginning of the treatment but by the end of the 1 year period cinacalcet was discontinued in all cases. (There is additive risk of hypocalcemia since cinacalcet alone can cause hypocalcemia.)

Conclusion

Overall, the studies concluded that denosumab can be used in hemodialysis patients although aggressive monitoring of serum calcium with subsequent adjustments in active vitamin D, calcium supplementation and changes in calcium dialysate may be required.

Monitoring was completed on a weekly-to-monthly basis depending on the trial. **Calcium levels returned to baseline within 2-3 months following the injection.** Festuccia et al. and Hiramatsu et al.'s studies suggest denosumab is well tolerated and hypocalcemia can be safely managed with regular monitoring and repletion of calcium and vitamin D. However, Dave et al.'s study had a high number of adverse events and advised to use denosumab cautiously. **In all cases, the monograph recommendation of a one-time measure of calcium 2 weeks after the injection in dialysis patients is not adequate to manage the continuous risk of hypocalcemia.**

Two of the studies are limited by their retrospective design, and all of the studies are limited by not having a placebo comparison group and having a small sample size. In addition, the studies had relatively short follow up periods (the shortest being 3 months and the longest being 2 years) and did not study hard clinical outcomes (eg. fractures, mortality). Furthermore, no definitive diagnosis of CKD-MBD was done prior to denosumab treatment.

Denosumab has promising potential as a treatment to reduce the incidence of fractures in CKD-MBD patients with high bone turn over. Although currently there is limited evidence to suggest denosumab can prevent fracture risk in dialysis patients, future studies may reveal

denosumab's impact on incidence of fractures and mortality. In the meantime, dialysis practitioners should be vigilant for hypocalcemia in patients receiving denosumab and should ensure patients are appropriately monitored and that drug therapy is adjusted based on the biochemical data.

Denosumab (Prolia) results in hypocalcemia in dialysis patients following each injection. Hypocalcemia occurs as early as 7 days post injection and can last up to 2 months. Hypocalcemia can be managed with frequent monitoring of serum calcium and subsequent adjustments in active Vitamin D, calcium supplementation and increases in calcium dialysate concentration.

Appendix

Table 1: Summary of case reports of hypocalcemia following Prolia injection in hemodialysis patients

Author	No of patients	CKD stage	Age	Ca prior to injection (mmol/L)	Lowest serum Ca (mmol/L)	Symptoms	Time point of Ca nadir	Resolution of Hypocalcemia
McCormick <i>et al.</i> 2012	1	HD	61	2.22	1.34	Fatigue	1 month	↑calcitriol dose, ↑dialysate Ca
Ivanov <i>et al.</i> 2013	1	HD	64	2.45	1.97	Not reported	30 days	↓Cinacalcet dose, ↑calcitriol dose
Agarwal <i>et al.</i> 2013	1	PD	58	?	1.57	Tetany	7 weeks	IV Ca gluconate
Talreja <i>et al.</i> 2014	1	CKD5	68	2.22	1.67	Mild confusion, dyskinesia	11 days	IV Ca gluconate ↑PO calcium dose, ↑ active Vit D dose
Sirvent <i>et al.</i> 2014	1	CKD5	75	2.51	1.09	Tremor, muscle spasms	14 days	IV calcium gluconate, IV calcitriol

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The Renal Pharmacists Network

Friday May 4th, 2018

**Marriott Vancouver Pinnacle Downtown Hotel
1128 W. Hastings Street, Vancouver
Grey Point Room**



AGENDA

Morning

Review of Nephro-Oncology
Keynote speaker: Dr. A.Kitchlu

Hyperuricemia in chronic kidney disease
Keynote speaker: Dr. J.Bargman

Short and Snappy Presentations:

REPRISE Trial

Symptom Management; patients and health care professionals point of views
Polypharmacy in dialysis patients

Afternoon

Vaccination

Update on Hepatitis B vaccination in hemodialysis patients
Vaccination in pre-transplantation patients

Round Table Discussions

break out in group discussions with other pharmacists on relevant topics

For full agenda details please refer to our website:

<https://renalpharmacists.net/>

Objectives

This program will provide pharmacists working directly in the nephrology area with an opportunity to gain more insight into issues in renal disease.

Registration

There will be a non-refundable \$50 registration fee. Register at www.renalpharmacists.net

Final registration deadline is April 27th, 2018.

Registration is on a first come, first serve basis – max. 50 people.

**For further information please contact
Judith Marin Judith.Marin@fraserhealth.ca
Jenny Ng jenny.ng@sunnybrook.ca**

Upcoming Nephrology Conferences



NKF's Spring Clinical Meetings

April 10-14, 2018

Austin, TX

<https://www.kidney.org/spring-clinical>



ASN Kidney Week 2018

October 25-28, 2018

(Early programs Oct 23-24, 2018)

San Diego, CA

<https://www ASN-online.org/education/kidneyweek/2018/meeting-overview.aspx>



CSN 2018 AGM - 50th Anniversary

May 4-5, 2018

(Pre-Course May 3-4, 2018)

Vancouver, BC

<https://www.csnsn.ca/agm/>

ISPD 2018 Congress – International Society for Peritoneal Dialysis

May 5-8, 2018

Vancouver, BC

<http://ispdvancouver2018.org/>



Please send any articles
of interest to
renalpharmacistsnetwork@gmail.com



Recent Publications

Opioid Prescription, Morbidity, and Mortality in United States Dialysis Patients

Paul L. Kimmel, Chyng-Wen Fwu, Kevin C. Abbott, Anne W. Eggers, Prudence P. Kline, and Paul W. Eggers
JASN. 2017;28: 3658-3670

Also see editorial: Prescription Opioids for Pain

Management in Patients on Dialysis
Beth Han and Wilson M. Compton
JASN. 2017;28: 3432-3434

Residual Kidney Function and Peritoneal Dialysis–Associated Peritonitis Treatment Outcomes

Whitty R, Bargman JM, Kiss A, Dresser L, and Lui P.
Clin J Am Soc Nephrol. 2017;12(12) 2016-2022

Ambulatory Medication Reconciliation in Dialysis Patients: Benefits and Community Practitioners' Perspectives

Jo-Anne S. Wilson, Matthew A. Ladda, Jaclyn Tran, Marsha Wood, Penelope Poyah, Steven Soroka, Glenn Rodrigues, and Karthik Tennankore.
Can J Hosp Pharm. 2017;70(6):443-9

Warfarin Use in Hemodialysis Patients With Atrial Fibrillation: A Systematic Review of Stroke and Bleeding Outcomes

Chieh Tsai, Laura Quinn Marcus, Priya Patel, and Marisa Battistella.
Can J Kidney Health and Dis. 2017; Vol 4

Effectiveness and Cost of Weekly Recombinant Tissue Plasminogen Activator Hemodialysis Catheter Locking Solution

Brenda R. Hemmelgarn, Braden J. Manns, Steven D. Soroka, Adeera Levin, Jennifer MacRae, Karthik Tennankore, Jo-Anne S. Wilson, Robert G. Weaver, Pietro Ravani, Robert R. Quinn, Marcello Tonelli, Mercedeh Kiaii, Paula Mossop, and Nairne Scott-Douglas.
Clin J Am Soc Nephrol. 2018; Vol 13

Medication Therapy Management after Hospitalization in CKD: A Randomized Clinical Trial

Katherine R. Tuttle, Radica Z. Alicic, Robert A. Short, Joshua J. Neumiller, Brian J. Gates, Kenn B. Daratha, Celestina Barbosa-Leiker, Sterling M. McPherson, Naomi S. Chaytor, Brad P. Dieter, Stephen M. Setter, and Cynthia F. Corbett

Clin J Am Soc Nephrol. 2018;13(2): 231–241

A systematic review of direct oral anticoagulant use in chronic kidney disease and dialysis patients with atrial fibrillation.

Feldberg J, Patel P, Farrell A, Sivarajahkumar S, Cameron K, Ma J, Battistella M.
Nephrol Dial Transplant. 2018 Mar 2. [Epub ahead of print]

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