Newsletter Vol 17 Issue 1 Spring-Summer 2014

The Renal Pharmacist

Vol 17

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In this issue ...

A biannual Insight into the Renal Pharmacist Network

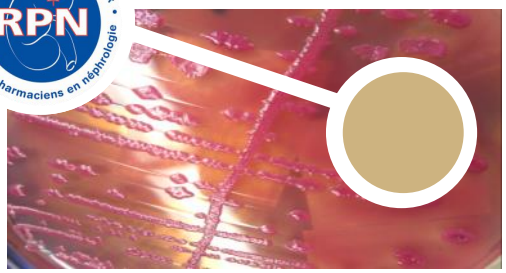
View from the Chair

Being Chair of the Renal Pharmacists Network for 2014, I'd like to reflect on what the RPN has done thus far in 2014 and would like to look ahead on what we wish to accomplish for the rest of the year. It's been a colder than usual winter and spring for most of Canada so I'm sure most of us are looking forward to the summer months ahead.

Our first major event for RPN this year was the Canadian Society of Nephrology (CSN) annual meeting held during the week of April 22 in Vancouver, BC. On April 24th, thirty-five members of the RPN attended the annual Education Day at CSN, including 2 RPN bursary recipients, Cali Orsulak and Elena Sze. The RPN had a wide assortment of pharmacist and physician speakers from across Canada. I was extremely pleased at the quality and variety of the topics presented – I was also proud to see the original research / innovative roles renal pharmacists across Canada are involved. It was truly a successful event. For those who could not attend the CSN this year, a selection of presentation handouts are available on the website.

"look ahead on what we wish to accomplish"

On April 25th, the RPN had a joint education workshop with the CSN, with Marisa Battistella moderating. Dr. Michael Sebag, oncologist from McGill University and Dr. Shirin Abadi, clinical associate professor from UBC had a joint presentation discussing multiple myeloma and the management of medication issues in myeloma patients with renal impairment. Dr. Michael Zappitelli, pediatrician from McGill University, discussed the issues of acute kidney injury in an oncology setting. Dan Martinusen, from the Vancouver Island Health Authority, presented on rasburicase for the prevention and treatment of tumour lysis syndrome. The CSN will have next year's annual general meeting in Montreal. Hope to see you next year!



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Other upcoming events to keep on the agenda: RPN education days in Vancouver, Calgary and Toronto this fall. The RPN Education Committee is working hard to organize the venue and book the speakers, so periodically check the renalpharmacists.net website as well as email updates (for those registered to receive electronic correspondence) for these upcoming events. Looking forward to meet other RPN members at these future outings!

On an organizational level, I'd like to personally welcome Carlee Balint, from Alberta Health Services, to join the RPN Executive Committee as Chair-Elect for 2014. If anyone has any interest being more involved with RPN (i.e. help organize / coordinate educational events) or serve on the Executive Committee, please contact us directly or through the renalpharmacists.net website.

Have a great summer!

Derrick Soong,
RPN Chair 2014

RPN Executive from left to right: Elaine Cheng, Grace Leung, Derrick Soong, Marisa Battistella, Judith Marin

Do you want to get involved with the Renal Pharmacists Network?

We are currently taking nominations for the **2015 Chair Elect**.
Find out more by contacting one of our executive members!

www.renalpharmacists.net

Highlights: The Renal Pharmacists Network Nephrology Education Day, 2014, Vancouver, British Columbia

The RPN Nephrology Education Day was held in Vancouver on April 24th, 2014, prior to the CSN conference. There was a great turn out of pharmacists from Vancouver as well as many pharmacists from across the country. Dr Lavern Vercaigne, Professor and Associate Dean (Academic), from the Faculty of Pharmacy, University of Manitoba, started off the day with the much awaited preliminary results from his 30% ethanol/4% citrate study. He tested the safety and efficacy of this novel catheter locking solution to prevent hemodialysis catheter-related infections and thrombosis. Please see the posted presentation slides on the RPN website.

Next, Robin Cho, a Clinical Pharmacy Specialist in Nephrology from Fraser Health Authority, BC presented on the rare but often fatal complication of long-term peritoneal dialysis called encapsulating peritoneal sclerosis. Below, he gives a brief summary of his talk and RPN members may also download his slides at the RPN website.

Dan Martinusen and Cliford Lo discussed the question, "Is warfarin good for dialysis patients and if it is, how well are we managing it?" A controversial topic that stimulated good questions. Our own RPN executives presented various topics in the Short and Snappy session. Piera Calissi reviewed the use of hydralazine in CKD, Marisa Battistella discussed her novel role as a Pharmacy Clinician Scientist at the Leslie Dan Faculty of Pharmacy and Derrick Soong discussed a new approach to IV iron. Also below, Derrick has provided a brief summary of his presentation.

In the afternoon, Dr Vanita Jassal, a nephrologist from the University Health Network in Toronto, gave a presentation on the management of depression in CKD. This was followed up with our Round Table Discussions.

Overall, the day was a great success and provided opportunities for pharmacists from various institutions to network and discuss the latest topics. We look forward to seeing you again next year!

Dr Lavern Vercaigne speaking about antibiotic and ethanol locking solutions.

From left to right: Dan Martinusen and Clifford Lo.

Encapsulating Peritoneal Sclerosis

Submitted by Robin Cho, BScPharm, ACPR, PharmD, BCPS, Fraser Health Peritoneal Dialysis Program, New Westminster, BC

Encapsulating peritoneal sclerosis (EPS) is an infrequent complication of long-term peritoneal dialysis (PD). This condition was reported in Japan and Australia using this terminology. Also known by other names, such as sclerosing encapsulating peritonitis (SEP), peritoneal chronica fibrosa incapsulata, and "abdominal cocoon," it involves clinical symptoms and the encapsulation of bowel loops. EPS is associated with significant morbidity and mortality, with mortality rates in the literature ranging between 26 and 58%.

The incidence of EPS has varied among countries. For example, reports from Australia have described incidence rates of 6% after being on PD for 5 years, and 19% after being on PD for 8 years. Incidence rates in Japan have been reported to be 1-2% after 5 years

of PD, and 2-6% after 8 years of PD. In Scotland, reported incidence rates are 4% after 3-4 years of PD, and 6% after 4-5 years of PD. Generally, the risk of EPS is believed to be relatively low in patients who have been on PD for less than 5 years, and appears to increase with extended length of time on PD. A multicentre study of 1958 patients in Japan reports the incidence of EPS to be 17% in patients who have been on PD for more than 15 years.

The development of EPS is believed to be multi-factorial; however, a consistent risk factor appears to duration on PD. Other potential risk factors include inflammation, prior episodes of severe peritonitis, exposure to foreign agents such as plasticizers or chemicals, and exposure to glucose-based PD solutions. A "two-hit" mechanism is believed to be implicated in the development of this condition. The first hit involves long-term PD exposure, leading to the breakdown of cells, increased fibroblast and macrophage activity, and defective fibrinolysis at the level of the peritoneal membrane. This ultimately leads to progressive peritoneal fibrosis, thickening, and adhesion. The second hit involves inflammatory stimuli, which acts as an accelerant of the processes that occur during the first hit.

The diagnosis of EPS requires two components: a clinical syndrome, which may involve gastrointestinal complaints such as anorexia, nausea, vomiting, weight loss, or malnutrition, and the encapsulation or encasement of bowel loops. Although markers of inflammation in laboratory findings, such as increased CRP, low albumin, and anemia are typical of EPS, there is no specific diagnostic effluent or blood marker for this condition. The diagnosis of EPS is most often conferred via a combination of assessing the patient's clinical symptoms, and the discovery of classical findings of EPS on CT, laparotomy, or laparoscopy.

To date, there have been two sets of guidelines that been be written and disseminated about EPS. The first set of guidelines originated in Japan and were published by Kawaguchi et al in 2005. Subsequent to that, the EPS Clinical Guidelines Group from the United Kingdom developed a set of guidelines in 2009.

Most of the current literature on the management of EPS is in agreement in that there is no definitive approach in the management of this condition. However, most experts agree that treatment should be initiated promptly upon discovery. An appropriate management plan generally includes discontinuation of peritoneal dialysis, bowel rest, nutritional support, medications, and consideration for surgical intervention. Reports have been published on the use of various medications in the treatment of EPS. Corticosteroids are believed to suppress the inflammatory processes that occur at the peritoneal membrane and inhibit collagen synthesis and maturation. Prednisone or prednisolone may be tried, at starting doses of 0.5-1 mg/kg/day. Alternatively, pulse-dose methylprednisolone at initial doses of 500-1000 mg daily may be reasonable. A prospective study published by Kawanishi et al in 2004 reported recovery from EPS in 15 out of 39 patients treated with prednisolone or methylprednisolone (alone or in combination with other interventions), compared to 0 out of 3 patients treated with TPN alone, and 7 out of 12 patients treated with surgery/enterolysis. However, it is important to note that only 6 out of these 39 patients were on corticosteroids alone.

Tamoxifen, which is believed to have antifibrotic properties in EPS, may also be reasonable at a dose of 10-40 mg daily. The Dutch Multicentre EPS Study by Korte et al was a retrospective study on survival involving 63 patients, comparing patients who received tamoxifen to those who did not. Patients who were treated with tamoxifen had a mortality rate of 46%, compared to 74% in the group that did not receive tamoxifen (p=0.03). Lastly, there have also been reports of using azathioprine, sirolimus, mycophenolate mofetil, or colchicine to treat EPS in the literature, often in combination with steroids. Regardless of the medication used, the optimal duration of treatment is yet to be established.

Lastly, the surgical enterolysis of abdominal adhesions may be indicated where clinical symptoms persist despite nutritional and medical therapy. The goal of surgical intervention is to maximize the removal of sclerotic tissue while avoiding accidental bowel perforation. Certain challenges exist with surgical intervention. Post-surgical mortality rates in EPS patients have varied from 4 to 80%. Other challenges include finding a surgeon experienced in the management of EPS and establishing an optimal time for surgery in relation to other therapies.

In summary, EPS, although a relatively rare condition, is associated with significant health, social, and societal effects. Despite published reports of therapies which appear to be effective, there still remains a lack of high-quality controlled trials to guide the management of this condition. This is one area of research which may derive substantial benefit from contributions of those involved in the study or management of these cases.

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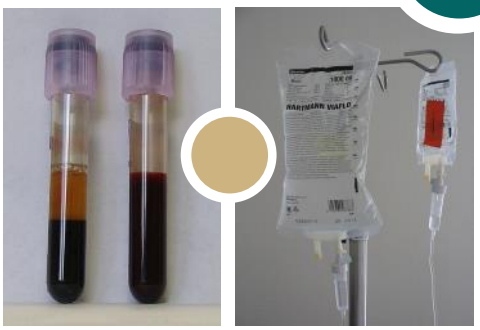
Short & Snappy

IV Iron: A New Approach to an Old Drug

Submitted by Derrick Soong, PharmD, Windsor Regional Hospital, Ontario



The use of IV iron to decrease erythropoietin stimulating agent (ESA) doses in hemodialysis patients is a standard of practice in North America, however, the effect of the frequency of IV iron dosing is not well studied. Our research team decided to conduct a retrospective, single centre cohort chart review to compare traditional one gram IV iron loads (as per the product monograph) versus a once-weekly maintenance dose (100 mg or 125 mg elemental iron, depending on which agent was used) for iron-deficient, ESA-requiring hemodialysis patients, as defined by an iron saturation (TSAT) less than twenty percent and a ferritin level less than 200.

The primary endpoints were the absolute hemoglobin level and the average weekly ESA dose of the traditional VS once-weekly IV iron groups. Secondary endpoints included: TSAT, total iron, ferritin level, and total IV iron dose over a 3 month

period. The study period was between 2010 and 2012, where 2011 was the switchover from the traditional IV iron dosing as per the product monograph to the once-weekly IV iron dosing schedule. A total of 567 charts were reviewed, of which 419 were excluded. Exclusion criteria were: age less than 18 years, non-hemodialysis patients, IV iron use less than a total of 3 months, allergies or contraindications to IV iron (iron or other ingredients contained in IV iron), patient refusal, non-iron causes of anemia, and patients using darbepoetin. There were 148 charts included in the study, 99 patients in the traditional IV iron dosing and 49 patients in the once-weekly IV iron dosing. Patient baseline demographics were similar, except for dry weight, where the once-weekly dosing group was significantly heavier (72.9 kg vs 83.1 kg, $p < 0.0001$).

After analyzing the data, the once-weekly IV iron group had a non-significant difference in hemoglobin, however, the weekly ESA dose was significantly lower ($p = 0.04$), after multivariate analysis. Secondary outcomes found that there was a significant improvement in TSAT ($p= 0.01$) and absolute iron ($p= 0.01$), with a non-significant difference in ferritin or IV iron dose used in 3 months between study groups.

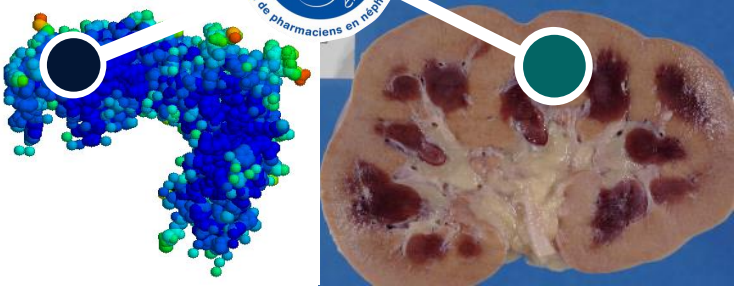
The authors concluded that once-weekly IV iron administration significantly reduced ESA consumption, improved TSAT and total iron, without significantly affecting hemoglobin, ferritin or IV iron consumption. Some of the strengths of the study included the number of patients enrolled and the duration of the observation. Some of the limitations of the study were the retrospective nature, single centre and different IV iron used in the observation period. Future studies in this area should be conducted to remove any potential confounding in the results.

Highlights: Canadian Society of Nephrology 2014, Vancouver, British Columbia

Once again, the RPN had the opportunity to have a joint education session with the CSN during their 2014 annual general meeting. This year's subject was titled "Oncology and the Kidney." The session was divided in two; the first part addressed the risk of renal failure in patients with multiple myeloma. The second part was directed toward the risk of acute kidney injury (AKI) in oncology patients.

Acute Kidney Injury in Oncology patients

Submitted by Judith Marin PharmD, St Paul's Hospital, Vancouver, BC



Dr. Zappitelli gave an interesting talk on the different predisposing factors to acute kidney injury (AKI) in oncology patients, with a focus on the pediatric population. AKI is quite common in patients with cancer and is associated with an increased length of hospital stay, cost and mortality. The approach to AKI differential diagnosis in oncology patients is no different than for any other patients; however, some AKI causes have a higher probability in this population. Here are some factors pharmacists should keep in mind when evaluating these patients:

- Cancer patients are at high risk of volume depletion related to nausea, vomiting and diarrhea due to chemotherapy agents. Make sure these symptoms are under control.
- Hypercalcemia can cause AKI due to direct renal vasoconstriction and natriuresis-induced volume depletion. Saline hydration and biphosphonate/ calcitonin treatment should be considered to correct calcium level.
- Medications are definitely an important factor to consider while evaluating AKI cases. Exposure to nephrotoxic medications, chemotherapy or not, such as methotrexate, cisplatin, ifosfamide, interferon a, amphotericine B, renin angiotensin aldosterone system (RAAS) blockers, intravenous contrast, acyclovir, calcineurin inhibitors, biphosphonates and non-steroidal anti-inflammatory drugs (NSAIDs) to only name a few important ones, should be minimized. In addition, medications that can cause interstitial nephritis should be kept in mind while reviewing these cases (for example antibiotics, allopurinol).

In the second part of the talk, Dan Martinusen discussed the used of rasburicase in patients with tumor lysis syndrome (TLS). TLS, an oncologic emergency, is also a common cause of AKI in patients with malignancy. TLS is caused by massive tumor cell lysis and the release of large amounts of potassium, phosphate, and uric acid into the systemic circulation. Deposition of uric acid and/or calcium phosphate crystals in the renal tubules can result in acute kidney injury. Clinical trials have not been performed to demonstrate superiority of any specific prophylactic regimen. Usual recommendations are to increase hydration if patient has no volume restriction and the avoidance of any kidney vasoconstrictive substances, such as NSAIDs or iodinated contrast. Prophylactic use of xanthine oxydase inhibitor (allopurinol or febuxostat) is usually recommended for patients with medium to high risk of developing TLS. Once TLS has developed, effort should be made to reestablish normal concentration of extracellular solutes. Volume expansion with the goal to improve solutes renal excretion is the centre of therapy. Allopurinol is a less effective option for TLS treatment since it decreases generation of uric acid, but may not improved current hyperuricemia.

Rasburicase has been used in both treatment and prevention of TLS. Recombinant urate oxidase (rasburicase) catalyzes the conversion of uric acid to allantoin, carbon dioxide and hydrogen peroxide. This treatment was shown to quickly decrease uric acid level, but has never been associated with improved clinical outcomes, like a decrease risk of acute renal failure or the decrease in need for dialysis. Rasburicase should not be used in patients with G6PD deficiency because of an increased risk of severe hemolysis and methemoglobinemia. The manufacturer recommends a dose of 0.15 to 0.2 mg/kg/day for 5 days, starting during the initiation of chemotherapy. Although rasburicase is usually well tolerated, price has been a barrier to its use. No pharmacoeconomic studies have been published on this therapy, which details in Canada at an average of $4,400/dose.

Given the high cost, there is a great interest in alternative dosing strategies. One study demonstrated that a single dose of 0.15 mg/kg before the onset of chemotherapy, with a rescue dose of 0.15 mg/kg as needed was as effective as the 5 day dosing regimen in normalizing the uric acid level. If this therapeutic option is chosen, uric acid levels should be followed closely and uric acid levels should be measured on ice to ensure its accuracy since rasburicase is active ex vivo. Dan agrees with current recommendations to prioritize allopurinol and hydration for TLS prophylaxis, which has a daily cost of about $4.50. However, if a patient presents with acute renal failure or is at high-risk of TLS, rasburicase could be considered. We also need to keep in mind that rasburicase decreases uric acid level, but doesn't resolve the electrolytes disturbances also associated with TLS.

Multiple Myeloma: New Therapies and Renal Consequences

Submitted by: Elena Sze, B.Sc.Phm, The Scarborough Hospital, Toronto, ON.

At the Canadian Society of Nephrology Annual General Meeting in April, Dr. Michael Sebag and Dr. Shirin Abadi co-presented on "Multiple Myeloma: New Therapies and Renal Consequences". Dr. Sebag began by explaining the molecular aspects of multiple myeloma, clarified some of the common terms used in describing the disease, reviewed the staging, and highlighted some of the treatment strategies. Dr. Abadi then followed with a discussion on the renal implications in the treatment of this disease. Below is a summary of their presentation:

In Canada, there are approximately 2200 new cases and about 1300 deaths per year due to multiple myeloma. The incidence is generally higher in males and in those from African descent, although the reason behind this is unclear. Multiple myeloma is a disease that primarily targets the

Common Terminologies Related to Multiple Myeloma:

CRAB Features	These are the clinical features of multiple myeloma: Hypercalcemia Renal failure Anemia Bone disease (e.g. lytic lesions, osteoporosis, fractures)
Monoclonal Gammopathy of Undetermined Significance (MGUS)	This is a benign condition, but has the potential to develop into multiple myeloma. It is characterized by: M protein < 30 g/L Bone marrow plasma cells < 10% Absence of CRAB features
Smoldering Myeloma	This is the asymptomatic form of multiple myeloma, with absence of CRAB features, characterized by: M protein > 30 g/L Bone marrow plasma cells ≥ 10%
Myeloma	This is characterized by: Any amount of M proteins Any amount of bone marrow plasma cells found in biopsies Presence of CRAB features

older population. It rarely occurs before 45 years of age, and the median age of diagnosis is 70 years old. It is a disease of differentiated B cells, and the accumulation of plasma in the bone marrow leads to the problems that we observe in our patients.

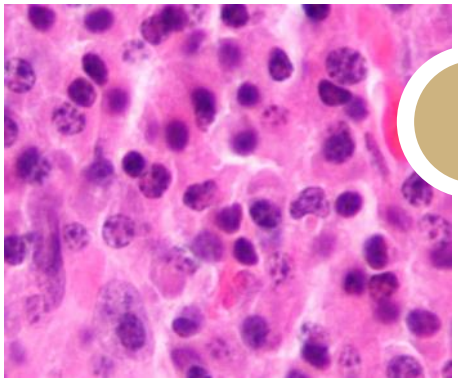
Multiple myeloma is typically staged using the **International Staging System**, which is based on the serum levels of β-2 microglobulin and albumin :

Stage I: Serum β-2 microglobulin level < 3.5 mg/L with normal albumin level
Stage II: Any cases that do not fall under Stages I and III
Stage III: Serum β-2 microglobulin level > 5.5 mg/L

However, nowadays, clinicians also look for genetic abnormalities in patients with multiple myeloma to identify high risk features.

The treatment of multiple myeloma has evolved significantly over time. Prior to the 1950's, quinine, steel, and urethane were used as primary treatments. Melphalan was discovered in the 1950's and it was considered the first effective treatment for multiple myeloma. Corticosteroids (e.g. prednisone, dexamethasone) were later included in the treatment regimens. In the 1980's and 90's, autologous transplantation became available as part of the treatment. More recently, novel therapies were developed, with new classes of drugs such as **proteasome inhibitors** (e.g. bortezomib, carfilzomib) and **immunomodulators** (e.g. thalidomide, lenalidomide, pomalidomide). Myeloma cells are very responsive to proteasome inhibitors such as bortezomib, which is currently available in Canada. Carfilzomib is typically used for patients who failed treatment with bortezomib, but this drug is not available in Canada yet. From a toxicity perspective, lenalidomide and pomalidomide cause less adverse effects than thalidomide. Pomalidomide is used when patient has failed both thalidomide and lenalidomide.

Since there is no complete cure for multiple myeloma, some of the goals of treatment are to maintain remission and to delay disease recurrence. For younger patients, usual treatment consists of induction therapy, followed by stem-cell transplantation, then maintenance therapy. The standard of care for induction therapy prior to stem-cell transplantation consists of a combination of 3 agents: cyclophosphamide, low-dose bortezomib, and dexamethasone (CYBOR-D). This combination produces high remission rates and the effect lasts for many years without recurrence after transplant. In addition, some clinicians may consider using lenalidomide after stem-cell transplant to delay the progression of multiple myeloma and to improve overall survival.



However, in older patients, because they have a lower tolerance to high-dose chemotherapy, and stem-cell transplant may not be an option, their treatments typically consist of the use of novel and traditional medications, followed by maintenance therapy. The addition of a novel agent such as bortezomib to melphalan and prednisone produces better treatment response than using melphalan and prednisone alone. The duration of response with the three-drug regimen lasts about 24 months, which is comparable to the duration of response in a younger patient who has undergone stem-cell transplant.

Dr. Sebag also explained the concept of **clonal tiding**, where the disease exists in "clones". When multiple myeloma returns as a "clone", it may be resistant to the initial therapy and warrants the use of a secondary therapy. However, once the secondary therapy has failed, the patient may once again respond to the original therapy because the original disease has re-emerged. Therefore, the management of multiple myeloma may require reusing agents continuously over time to suppress clones and to manage relapses.

Multiple myeloma patients are prone to develop renal dysfunction. About 13% of patients experience severe renal impairment and they tend to have worse treatment outcomes and increased mortality. There are many contributing factors to the development of renal dysfunction in this group of patients, such as hypercalcemia, infection, nephrotoxic drugs, contrast media, and amyloidosis. However, light chain tubular cast nephropathy appears to be the main cause. In multiple myeloma, high amounts of light chain proteins are excreted in the urine, which leads to the formation of casts in the tubules, causing tubular obstruction and distal tubular dysfunction.

Interestingly, although multiple myeloma can lead to renal impairment, the impairment is reversible in 70% of patients provided that they received timely and effective treatments. Commonly used drugs that require dose adjustment in renal impairment include melphalan and

lenalidomide. Bortezomib has been studied in patients with renal impairment, and dose adjustments are not needed because it is metabolized via CYP 3A4, 2C19, and 1A2. There is limited experience in using thalidomide in the renally impaired, so it is not recommended in patients with severe renal impairment. Currently there are several ongoing prospective trials looking into the dosing for lenalidomide in renal impairment, so it is hoped that their results will provide more guidance on dose adjustments. Until more data become available, clinicians generally follow this dosing for lenalidomide:

- Mild renal impairment: Give regular dose 25mg PO daily
- Moderate impairment: Reduce to 10mg daily
- Severe impairment: Give 15mg every other day
- Dialysis or end-stage renal disease: Give 5mg daily (for hemodialysis patients, give dose after dialysis on dialysis days).

The most commonly used bisphosphonates for reducing bone pain and skeletal-related events in multiple myeloma patients are zoledronic acid (4mg IV over 15 minutes) and pamidronate (30-90 mg IV over 1-2 hours) every 4 weeks until good clinical response is attained or up to 2 years. These medications are cleared renally and can cause acute tubular damage, but dose adjustment guidelines are lacking in terms of use in CrCl < 30 mL/min, thus, practice is mostly based on clinical experience. The BC Renal Agency extends the infusion time of pamidronate (maximum 22.5 mg/hr) and reduces the dose of zoledronic acid and monitors renal function in this group of patients.

Multiple myeloma is a complex disease. The condition itself and the available treatments can potentially lead to renal impairment. Medications used for treating multiple myeloma may require dose adjustments based on renal function. Therefore, careful monitoring is required at all times to ensure the appropriateness of therapy.

Volume and Blood Pressure Control in Hemodialysis

Submitted by Cali Orsulak, BScPharm, Health Sciences Centre, Winnipeg, MB

During the CSN conference in April 2014, I had the opportunity to hear Dr. Ercan Ok of Turkey speak on *Volume and Blood Pressure Control in Hemodialysis*. Dr. Ok stressed that salt intake has a key role in causing hypertension in hemodialysis patients via multiple mechanisms but focusing on volume.

In hemodialysis patients this overconsumption of salt is responsible for increased fluid intake resulting in volume overload and hypertension. If this is continued over a prolonged period of time then end result is left ventricular hypertrophy and dilated cardiomyopathy.

Dr. Ok mentions that often the hypertension in hemodialysis patients is treated with one or more antihypertensive agents or labeled as treatment resistant when the cause (fluid excess) is overlooked. From a pharmacy perspective this is important when trying to optimize a patient's medication regimen.

He also discusses a study that he co-authored retrospective cross-sectional study included 423 HD patients, who had been treated by three times per week HD (scheduled as 12 h/week) at the same centre for at least a year, from two dialysis centers. One of which (Center A) promoted dietary salt restriction (5 g/day; 2 g or 88 mmol sodium) and intensive ultrafiltration for BP control; the other (Center B) used anti-hypertensive medications unless edema was present. Despite the similar session length, dialysate sodium concentration and dialyzer use, Center A had lower IDWG (2.3 versus 3.3 kg; P < 0.001) and less left ventricular hypertrophy (74 versus 88%; P < 0.001) by echocardiographic assessment. However, there were no detectable differences in SBP or diastolic blood pressures between the two centers. [1]

An additional study he authored included series of 218 thrice-weekly maintenance hemodialysis patients were analyzed following adoption of an institutional strategy consisting of dietary salt restriction, cessation of blood pressure medications and intensification of ultrafiltration. Analysis of food consumption patterns estimated a mean daily dietary salt intake of 3.2 g (~1.6 g or 70 mmol sodium). At the end of the observation period (mean 47 months), the mean pre-dialysis SBP had declined from 150 to 121 mmHg and IDWG had declined from 1.44 to 0.93 kg. [2]

A fixed lower dialysate sodium concentration in

combination with dietary salt restriction may also help control hypertension and reduce the requirement for blood pressure medication.[3]

So if salt is the culprit, what does he suggest? Increasing education to patients and their families. Simply telling patients to "quit drinking so much fluid" is an inadequate approach to dealing with the hypervolemic/hypertensive patient. He states that decreasing salt intake is an incredibly difficult adjustment and suggests it will take several weeks to months to" adapt" to a lower ''salt level,'' after which patients will find their previous food choices "too salty". He suggests that education is best done early on, even prior to starting dialysis and using a multidisciplinary approach to help patients and their families become accustom to the new diet.

This presentation should remind pharmacists that suggesting the addition of antihypertensive medication to hemodialysis patients might not be the most appropriate first line treatment in dealing with hypertension. Collaborating with the nephrologist, dietician and the patient are important to determine if salt intake and volume status are primary contributors. This can potentially result in prescribing less "pills" for our patients!

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Upcoming Conferences:

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BC Kidney Days 2014
Hosted by BC Transplant and the BC Renal Agency
Oct 16-17, 2014, Sheraton Vancouver Wall Centre

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American Society of Nephrology
Kidney Week 2014
November 11-16th, 2014 www.asn-online.org
Pennsylvania Convention Center, Philadelphia, PA

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National Kidney Foundation 2015
Spring Clinical Meeting
March 25-29th, 2015, Gaylord Texan, Dallas, Texas
www.kidney.org

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Canadian Society of Nephrology 2015
Annual Scientific Meeting
April 29-May 2nd, 2015, Montreal, Quebec
www.csnscn.ca

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Intensified pharmaceutical care is improving immunosuppressive medication adherence in kidney transplant recipients during the first post-transplant year: a quasi-experimental study.
Joost, R; Dörje, F; Schwitulla, J; Eckardt, KU; Hugo, C
Nephrol Dial Transplant. 2014 Jun 9. pii: gfu207. [Epub ahead of print]

This German study found that intensified pharmaceutical care led by a clinical pharmacist improved daily drug adherence compared to standard care (91% vs 85%, P=0.014) in renal transplant patients at 1 year post transplantation.

Dosing chemotherapy agents in hemodialysis--a focus on multiple myeloma.
Runnels R, Cameron K, Ng P, Battistella M.
CANNT J. 2014 Jan-Mar;24(1):21-5; quiz 26-7.

This review paper discusses the epidemiology and presentation of multiple myeloma and factors to consider for the removal of chemotherapy agents by hemodialysis.

An update on vancomycin dosing and monitoring practices in hemodialysis patients.
Zhang, M; Dresser, L; Battistella, M
CANNT J, vol. 23(4) pp. 25-7; quiz 28-9

This review paper discusses target trough vancomycin serum concentrations and reviews different dosing protocols to achieve these targets. The authors conclude that fixed dose protocols do not consistently achieve target trough levels whereas newer weight-related dosing protocols are more likely to achieve these targets.

Optimized Dosing of Cefazolin in Patients Treated With Nocturnal Home Hemodialysis.
Law, V; Walker, S; Dresser, L; Cardone, KE; Richardson, R; Chan, C; Battistella, M
Am. J. Kidney Dis., 2014;64:479-80.

In this research letter, the pharmacokinetics of cefazolin is described in 15 nocturnal hemodialysis patients. The authors conclude that 2 grams of cefazolin followed by 1 gram after each 8 hour nocturnal hemodialysis session achieves 6 times the minimum inhibitory concentration of S. aureus for greater than 50% of the dosing interval.

Medication Pitfalls in the CKD Clinic: Case Presentations.
Liles, AM
Adv Chronic Kidney Dis, 2014 vol. 21(4) pp. 349-354

A good resource for pharmacy students on a nephrology clinic rotation.

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