APSN/HKSN
CME Course 2019
Saturday, 28 September 2019
Cheung Kung Hai Conference Centre, William MW Mong Block,
Li Ka Shing Faculty of Medicine, the University of Hong Kong
## Programme

### APSN/HKSN CME Course

**Saturday, 28 September 2019**

**Venue:** Cheung Kung Hai Conference Centre, William MW Mong Block, Li Ka Shing Faculty of Medicine, the University of Hong Kong

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<td><strong>Opening Address</strong>&lt;br&gt;(1) Chairman, Hong Kong Society of Nephrology&lt;br&gt;(2) President, Asian Pacific Society of Nephrology&lt;br&gt;(3) President, Hong Kong College of Physicians</td>
<td>Dr. Yuk Lun CHENG&lt;br&gt;Prof. Masaomi NANGAKU&lt;br&gt;Prof. Philip KT LI</td>
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<td>14:10 – 14:35</td>
<td>Chairs: Dr. Chun Yu YUNG and Dr. Terence PS YIP&lt;br&gt;Slowing Progression of Chronic Kidney Diseases</td>
<td>Prof. Mark ROSENBERG (USA)&lt;br&gt;Prof. Sydney CW TANG (Hong Kong)</td>
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<td>14:35 – 15:00</td>
<td>Chairs: Dr. Sunny SH WONG and Dr. Siu Ka MAK&lt;br&gt;IgA Nephropathy: The Landscape after 50 Years</td>
<td>Prof. Frederick WK TAM (UK)&lt;br&gt;Dr. Jack KC NG (Hong Kong)</td>
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<td>17:10 – 17:35</td>
<td>Chairs: Dr. Siu Fai CHEUNG and Dr. Kai Ching HAU&lt;br&gt;Importance of Nutritional Status in Chronic Kidney Disease and Dialysis Patients</td>
<td>Prof. Eiichiro KANDA (Japan)</td>
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<td>17:35 – 18:00</td>
<td>Case Record from the HKSN Interhospital Meeting&lt;br&gt;The Controversy on PPI Induced Hypomagnesaemia</td>
<td>Dr. Ping Nam WONG (Hong Kong)</td>
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Abstracts

Slowing Progression of Chronic Kidney Diseases

Early identification of CKD provides an opportunity to implement therapies to improve kidney function and slow progression. This presentation will first review the epidemiology of progression to define the normal and CKD related rate of decline in kidney function and discuss the competing risks between death and development of ESRD. Common pathophysiologic mechanisms underlie the progression of most kidney diseases including glomerular capillary hypertension, renal fibrosis, podocyte loss, proteinuria and activation of systems such as the renin-angiotensin-aldosterone system and intrarenal activation of developmental and injury pathways. These pathophysiologic factors present potential targets for therapy. The current state of art regarding treatment will be discussed and will include a careful review of the supporting evidence for different treatments. The importance of controlling blood pressure will be discussed along with the target blood pressure in CKD patients. Therapy directed at inhibiting the renin-angiotensin-aldosterone system remains a mainstay of treatment with single agent inhibition of this system being as good as dual blockade with fewer adverse effects. More recently evidence indicates that SGLT2 inhibitors and endothelin antagonism slow progression of diabetic kidney disease and this evidence will be discussed. Other therapies include glycemic control, correction of metabolic acidosis, and dietary protein restriction. Emerging therapies targeting uric acid, kidney fibrosis, oxidant stress, and kidney augmentation hold promise for the future and will be presented.

Prof. Mark ROSENBERG (USA)
Vice Dean of Education and Academic Affairs, Professor of Medicine, University of Minnesota Medical School, United States of America
President, the American Society of Nephrology
IgA Nephropathy: The Landscape after 50 Years

IgA nephropathy (IgAN) was first described in 1968 and over the last 50 years, much progress has been made on its epidemiology, pathology and classification, pathogenesis, and genetics. However, as of 2019, there is no approved specific treatment for IgAN, although a number of RCTs have been published on various forms of immunosuppressive approaches with variable outcomes.

A number of important publications in 2019 mark a new era in IgAN. First is the availability of a prediction tool to help predict, at the time of biopsy, the risk of a 50% decline in eGFR or ESRD. In this international multiethnic cohorts including 3,927 patients, 2 prediction models, one that included ethnicity, and one that did not, outperformed clinical measures for prediction of kidney disease progression and patient risk stratification. These models could help clinicians improve management in multi-ethnic cohorts. A similar study is also reported from China. A Swedish population-based cohort study investigated mortality in IgAN in which 3,622 patients were compared with 18,041 matched general population controls. On average, patients died 6 years earlier than people without the disease. These findings may have relevance to patient communication and policy development.

In terms of treatment, results of the multi-national spleen tyrosine kinase inhibition in IgAN study have been released and showed no significant change in proteinuria in fostamatinib treated subjects. Another RCT performed in a single centre showed hydroxychloroquine to confer anti-proteinuric effects. And a number of international RCTs have been started investigating the effect of low-dose systemic and regional corticosteroid, complement inhibition and dual endothelin/angiotensin receptor blockade.

Finally, the landscape for IgAN after 50 years will also be shaped by the emergence of a KDIGO guideline updated from the 2012 version, to be released for public review towards the end of 2019, and in a final publication in 2020.

Prof. Sydney CW TANG (Hong Kong)
Chair Professor of Renal Medicine and Yu Endowed Professor in Nephrology,
The University of Hong Kong, Hong Kong
President Elect, the Asian Pacific Society of Nephrology
Inflammatory Cytokines and Fibrotic Factors in Diabetic Nephropathy

Diabetic nephropathy is a common cause of kidney failure worldwide. Specific cytokines and fibrotic factors may have important roles in the progression of diabetic nephropathy.

MCP-1 (also known as CCL2) is a potent chemokine for monocytes and macrophages. Increased amount of urinary MCP-1 has been detected in urine from patients with diabetic nephropathy. Urinary MCP-1 level is prognostic of the rate of loss in kidney function. Recently, MCP-1 and its receptor have been targeted in clinical trials in diabetic nephropathy.

Transforming growth factor-{TGF} beta and connective tissue growth factor (CTGF, also known as CCN2) are important in fibrosis. However, inhibition of these fibrotic factors is challenging. Recent experimental approaches will be discussed.

MCP-1 and CTGF may provide new approaches in assessing prognosis and developing new treatments for diabetic nephropathy.

Prof. Frederick WK TAM (UK)
Ken and Mary Minton Chair of Renal Medicine, Hon. Consultant Nephrologist, Renal and Vascular Inflammation Section, Department of Medicine, Imperial College London, Hammersmith Hospital, London, United Kingdom
Updates on Fluid Management in Peritoneal Dialysis Patients

Cardiovascular disease (CVD) is the leading cause of death in patients on peritoneal dialysis (PD). However, the excessive cardiovascular mortality could not be completely accounted by Framingham risk factors. Fluid overload, which is closely related to hypertension and left ventricular hypertrophy, has been increasingly recognized as a non-traditional risk factor of CVD.

Despite the importance of maintaining euvolemia in PD patients, clinical assessment of fluid status may be subjective and inaccurate. Recently, there is a growing interest in the use of bioimpedance spectroscopy (BIS) because it provides a non-invasive and objective method to estimate hydration status. Importantly, BIS-defined fluid overload was associated with increase in all-cause mortality, technique failure and prolonged hospital stay.

The impact of fluid overload has extended beyond the boundary of CVD. Accumulating evidence revealed that fluid overload is associated with systemic inflammation, malnutrition and even frailty phenotype in PD patients. This lecture will give an overview on the risk factors and clinical outcomes of fluid overload in PD patients, and discuss the role of bioimpedance study in fluid management.

REFERENCE:

Dr. Jack KC NG (Hong Kong)
Resident Specialist, Department of Medicine and Therapeutics (Division of Nephrology), Prince of Wales Hospital, Hong Kong
Chairman, Young Nephrologists Committee of the Asian Pacific Society of Nephrology
Updates on Anaemia Management in Patients with Chronic Kidney Disease

Anemia is a common complication of chronic kidney disease and is caused by the inability of damaged kidneys to produce adequate amounts of erythropoietin (EPO). Recombinant human EPO and related products (erythropoiesis-stimulating agents, ESA) are the most successful biological reagent and has been widely used to treat anemia in CKD. However, there is a sub-population of patients who show hypo-responsiveness to ESA, and prognosis of these patients is poor.

Studies elucidating the regulation of EPO production led to the identification of hypoxia-inducible factor (HIF), which activates the transcription of genes that mediate adaptive responses to hypoxia. HIF is a heterodimer that consists of an α and β subunit. HIF-α is subjected to ubiquitination and proteasomal degradation under normoxic conditions. This process is mediated by prolyl hydroxylase, the inhibition of which results in an increased expression of hypoxia-induced genes, including EPO. These findings led to the development of prolyl hydroxylase inhibitors as novel therapeutic agents against anemia in CKD. Prolyl hydroxylase inhibition improves iron metabolism, which also contributes to erythropoiesis. Because HIF induces a variety of adaptive responses against hypoxia, HIF activation may also be beneficial in CKD itself and other ischemic diseases. However, there is a theoretical concern that the systemic activation of HIF could also induce some deleterious effects, which demands careful assessments in future clinical studies.

References:
(1) Sakashita M, Tanaka T, Nangaku M. Hypoxia-Inducible Factor-Prolyl Hydroxylase Domain Inhibitors to Treat Anemia in Chronic Kidney Disease. Contrib Nephrol. 2019;198:112-123

Prof. Masaomi NANGAKU (Japan)
Vice Dean, the University of Tokyo Graduate School of Medicine,
Professor and Head, Division of Nephrology and Endocrinology,
The University of Tokyo Graduate School of Medicine, Japan
President, the Asian Pacific Society of Nephrology
Sudden Death in Haemodialysis Patients: Is It Preventable?

From the United States Renal Data System, sudden death is the most common cause of death in patients on hemodialysis (HD), accounting for around one third of all deaths. Risk factors for sudden cardiac arrest (SCA) in the dialysis population are coronary artery disease, left ventricular hypertrophy and rapid electrolyte shift during HD sessions. Risk factors related to dialysis prescription are low potassium dialysate, low calcium dialysate and high ultrafiltration volume. Low magnesium dialysate may also be a risk factor although few data is available. Observational studies suggest that avoiding these modifiable risk factors in dialysis prescription may reduce the risk of SCA in HD patients. Nocturnal hemodialysis, a long and frequent dialysis therapy, minimizes the large volume and electrolyte shifts that occur with conventional thrice-weekly dialysis. This dialysis therapy has also been shown to induce regression of left ventricular hypertrophy. Therefore, nocturnal hemodialysis is a potential therapy that may reduce the risk of SCA in hemodialysis patients.

Dr. Hon Lok TANG (Hong Kong)
Consultant, Renal Unit, Department of Medicine and Geriatrics, Princess Margaret Hospital, Hong Kong;
Honorary Secretary, Hong Kong Society of Nephrology
Importance of Nutritional Status in Chronic Kidney Disease and Dialysis Patients

With aging, kidney function decreases with decreasing functions of other organs. The number of chronic kidney disease (CKD) patients has been increasing among elderly people. Elderly people often have various comorbidities such as diabetes, hypertension, chronic heart failure and CKD. Because of their comorbidities, they have high risks of not only end stage kidney disease but also cardiovascular disease, and death.

Comorbidities affect CKD patients’ nutritional status. “Protein energy wasting (PEW)” is a state of decreased body stores of protein and energy fuels (body protein and fat masses, respectively). It refers to multiple nutritional and catabolic alterations that occur in CKD and is associated with morbidity and mortality. The clinical diagnosis of PEW is based on serum chemistry data, body mass, muscle mass, and dietary intake among others. PEW is caused by various conditions such as decreased protein and energy intake, hypermetabolism, decreased physical activity, and various comorbidities. Malnutrition and inflammation weaken muscular strength and lead to decreased activities of daily living (ADL), which also leads to malnutrition.

To prevent the PEW-related vicious cycle of the above-mentioned conditions in CKD patients, improvements of their nutritional status, muscle mass, and ADL are necessary. Dietary intervention for CKD patients provided by nephrologists and dieticians decreases the risk of death. Dietary and exercise therapies are key strategies to improve the conditions and prognosis of those with PEW. Moreover, to ensure their sufficient nutritional intake and exercise, social services, especially medical and nursing care services, are required.

Prof. Eiichiro KANDA (Japan)
Professor, Medical Science,
Kawasaki Medical School, Japan
Magnesium is the fourth most abundant intracellular ion with numerous biological functions. Its serum concentration is the result of the interplay between intestinal absorption and renal excretion. Mild hypomagnesemia often goes unrecognized while severe hypomagnesemia might present with various neurological and cardiovascular manifestations, such as tetany, seizure and cardiac arrhythmia, with or without the presence of other electrolyte abnormalities, such as hypokalemia or hypocalcemia. There is a recent concern on hypomagnesemia in association with the use of Proton Pump Inhibitors (PPIs)- medications widely prescribed in primary and specialty clinical practice. Even since the first case report in 2006, there have been many observational studies showing a significant association between the use of PPIs and hypomagnesemia. However, there was substantial heterogeneity in the definition of hypomagnesemia and clinical characteristics of the studied populations in the literature. The reported prevalence also varied widely among different studies. While some patients appear more susceptible due to the presence of predisposing factors including poor oral intake, prolonged use of PPIs, advanced age, use of diuretics and genetic predisposition, the prevalence in individual populations of various risk profiles is unknown. Under the circumstances, there has not been any well-designed long term prospective study in the literature to clearly examine and establish the causative association. On the other hand, although a pH-dependent disruption of active intestinal absorption of Magnesium is proposed to be contributory, the exact underlying process or causative mechanism has yet to be further determined. At present, there are still many questions with regard to Proton Pump Inhibitor induced Hypomagnesemia (PPIH) remaining unanswered, which should require further study to clarify. Nevertheless, before the availability of these details, a high index suspicion should be exercised in patients taking PPIs, especially for those at risk, to identify this potentially serious electrolyte abnormality.

Dr. Ping Nam WONG (Hong Kong)

Senior Medical Officer, Department of Medicine and Geriatrics,
Kwong Wah Hospital, Hong Kong
Deputy Chief of Service (Operations), Department of Medicine & Geriatrics, Kwong Wah Hospital, Hong Kong
Director of Dialysis Services, Department of Medicine and Geriatrics, Kwong Wah Hospital, Hong Kong
Back row from left:
Dr. CHOW Chik Cheung Vincent; Dr. YIP Pok Siu, Terence; Dr. CHOW Kai Ming; Dr. HAU Kai Ching; Dr. CHEUNG Siu Fai; Dr. MAK Siu Ka; Dr. CHAN Yiu Han, John; Dr. YAP Yat Hin, Desmond

Front row from left:
Prof. LI Kam Tao, Philip; Prof. TANG Chi Wai, Sydney; Dr. CHENG Yuk Lun; Prof. YU Yue Hong, Richard; Dr. TANG Hon Lok; Dr. YUNG Chun Yu; Dr. FUNG Ka Shun, Samuel; Dr. TONG Kwok Lung, Matthew; Dr. WONG Sze Ho, Sunny

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