HKSN
Annual Scientific Meeting 2018

Sunday, 30 September 2018
Hung Hom Room, Kerry Hotel, Hung Hom, Hong Kong

APSN/HKSN CME Course

Saturday, 29 September 2018
Hong Kong Academy of Medicine Jockey Club Building, Hong Kong

PROGRAM

APSN/HKSN CME Course — Saturday, 29 September 2018

Venue: Pao Yue Kong Auditorium, Hong Kong Academy of Medicine

Time	Program	Speakers
13:45 – 14:00	**Registration**	
14:00 – 14:10	**Opening Address**	
	(1) Chairman, Hong Kong Society of Nephrology	Prof. Sydney TANG
	(2) President, Asian Pacific Society of Nephrology and Hong Kong College of Physicians	Prof. Philip LI
	Chairs: Dr. Hon Lok TANG and Dr. Desmond YAP	
14:10 – 14:35	Immunoregulatory Role of Tissue Microenvironment in Kidney Injury	Prof. Mark OKUSA (USA)
14:35 – 15:00	Basic Epidemiology presented in a Palatable Way: the problem of Bias	Prof. Kitty JAGER (the Netherlands)
	Chairs: Dr. Samuel FUNG and Dr. Terence YIP	
15:00 – 15:25	Blood Pressure Targets in CKD Patients in the post SPRINT Era	Prof. Carmine ZOCCALI (Italy)
15:25 – 15:50	Hypertension Metrics in Renal Transplantation	Prof. Francesca MALLAMACI (Italy)
15:50 – 16:15	**Group Photo** Coffee break / Exhibition	
16:15 – 16:20	**Address from the Chair of the APSN CME Committee**	Prof. Xueqing YU (China)
	Chairs: Dr. Koon Shing CHOI and Dr. Chun Yu YUNG	
16:20 – 16:45	The AKI to CKD Continuum: a Clinical Perspective	Prof. Masaomi NANGAKU (Japan)
16:45 – 17:10	CVD in PD Patients: Prevalence and Risk Factors	Prof. Xueqing YU (China)
	Chairs: Dr. Sing Leung LUI and John CHAN	
17:10 – 17:35	A Decision for the Elderly with Renal Failure: take an old kidney or remain on dialysis	Prof. Julio PASCUAL (Spain)
17:35 – 18:00	Case Record from the Interhospital Meeting The First ABO-incompatible Kidney Transplant in Hong Kong	Dr. Maggie MA (HK)

ABSTRACTS

Immunoregulatory Role of Tissue Microenvironment in Kidney Injury

Acute kidney injury (AKI) is one of many initiating events that contribute to chronic progressive disease. Some patients with AKI fully recover renal function, while in others development of CKD is accompanied by a progressive decline in kidney function leading ultimately to end-stage renal disease (ESRD). Regardless of the cause of CKD (nephrotoxic kidney injury, ischemia, infection, genetics; paraneoplastic, immunological processes), there is a stereotypical response leading to interstitial fibrosis, tubular atrophy and peritubular rarefaction and inflammation. Incomplete repair following AKI contributes to the progression to CKD and ESRD. During kidney recovery, renal and extrarenal cells participate in the wound healing response and can initiate fibrosis. Immune cells of the mononuclear phagocyte system, including macrophages and dendritic cells not only contribute to injury but have emerged as important cells in the recovery of kidney function during adaptive repair or fibrosis during maladaptive repair. It is the balance between wound healing and progressive fibrosis that dictates the final outcome. The intrinsic plasticity of monocytes/macrophages and dendritic cells as well as attempts to relate in vitro studies to in vivo findings makes the functional definition and phenotype of this myeloid population in kidney pathophysiology complex. In vitro studies have led to two well-defined mononuclear phagocytes. Classically activated macrophages (M1 mononuclear phagocytes consisting of macrophages and dendritic cells) are produced by exposure to LPS or INF-g and are largely thought to be pro-inflammatory and contribute to initial kidney injury. Alternatively activated macrophages (M2 mononuclear phagocytes) are produced by IL-4 and IL-10 appear later after AKI and have a genetic signature associated with wound healing and/or fibrosis. These mononuclear phagocytes phenotypes depend on the complex local tissue microenvironment, which may induce phenotype switching.

A key feature is the activation of extracellular matrix-producing myofibroblasts. Other factors important in CKD progression include endothelial cell damage and vascular damage in AKI, hypoxia-HIF, innate and adaptive immunity, cell cycle arrest and epigenetic mechanisms. While some injured tubules may undergo repair and regeneration, injury may also be accompanied by inflammation, maturation and proliferation of fibroblasts, and extracellular matrix deposition as part of the process of fibrosis. The source of fibroblasts in the injured kidney - fibrocytes, epithelial cells through EMT, intrinsic fibroblasts and pericytes - remains controversial.

Reference:

1. Venkatachalam MA, Griffin KA, Lan R, Geng H, Saikumar P, Bidani AK. Acute kidney injury: a springboard for progression in chronic kidney disease. Am J Physiol Renal Physiol 2010.
2. Schrimpf C, Duffield JS. Mechanisms of fibrosis: the role of the pericyte. Curr Opin Nephrol Hypertens 2011;20:297-305.
3. Huen SC, Huynh L, Marlier A, Lee Y, Moeckel GW, Cantley LG. GM-CSF Promotes Macrophage Alternative Activation after Renal Ischemia/Reperfusion Injury. J Am Soc Nephrol 2015;26:1334-45.
4. Zhang MZ, Yao B, Yang S, et al. CSF-1 signaling mediates recovery from acute kidney injury. J Clin Invest 2012;122:4519-32.
5. Yang L, Besschetnova TY, Brooks CR, Shah JV, Bonventre JV. Epithelial cell cycle arrest in G2/M mediates kidney fibrosis after injury. Nat Med 2010;16:535-43, 1p following 143.
6. Bechtel W, McGoohan S, Zeisberg EM, et al. Methylation determines fibroblast activation and fibrogenesis in the kidney. Nat Med 2010;16:544-50.
7. Wynn TA. Fibrosis under arrest. Nat Med 2010;16:523-5
8. Humphreys BD, Lin SL, Kobayashi A, et al. Fate tracing reveals the pericyte and not epithelial origin of myofibroblasts in kidney fibrosis. The American journal of pathology 2010;176:85-97.

Prof. Mark OKUSA

John C. Buchanan Distinguished Professor of Medicine;
Chief, Division of Nephrology;
Director, Center for Immunity, Inflammation and Regenerative Medicine, University of Virginia Health System, USA

ABSTRACTS

Basic Epidemiology presented in a Palatable Way: the problem of Bias

Bias is a systematic error in the design, conduct or analysis of a study. There are hundreds of types of biases. As a reader of clinical research papers one needs to be aware of the potential of bias in a study and see if authors have done all they could to address the problem. This presentation provides some examples of the most important form of bias, i.e. selection bias induced by physicians, patients or researchers. Finally, it discusses information bias and lead time bias.

Dr. Kitty JAGER

Managing Director, ERA-EDTA Registry;
Professor, Department of Medical Informatics, Amsterdam UMC, Location AMC,
The Netherlands

ABSTRACTS

Blood Pressure Targets in CKD patients in the post SPRINT Era

The BP target for in hypertensive CKD patients remains poorly defined. The Systolic Blood Pressure Intervention Trial (SPRINT) is a landmark study that tested a systolic BP target of <120 mm Hg in a large population of hypertensive patients (n=9361) including a subgroup of non-diabetic CKD patients with proteinuria <1g and without ADPKD (intensive treatment to reach the <120 mmHg target; n=1330) or <140 mm Hg (standard group; n=1316). After a median follow-up of 3.3 years, the primary composite cardiovascular outcome (myocardial infarction, other acute coronary syndromes, stroke, heart failure, or death from cardiovascular causes) occurred in 112 intensive group and 131 standard group CKD participants (hazard ratio [HR], 0.81; 95% confidence interval: 0.63 to 1.05). The intensive group also had a lower rate of all-cause death (HR, 0.72; 95% CI, 0.53 to 0.99). Treatment effects did not differ between participants with and without CKD. The composite renal end point of ≥50% decrease in eGFR from baseline or ESRD, occurred in 15 intensive group and 16 standard group participants (HR, 0.90; 95% CI, 0.44 to 1.83). After the initial 6 months, the intensive group had a slightly higher rate of change in eGFR (−0.47 versus −0.32 ml/min per 1.73 m2 per year; P<0.03). The overall rate of serious adverse events did not differ between treatment groups. However, hypokalemia and hyperkalemia were more common in the intensive group, likely related to more frequent use of medications, such as diuretics and inhibitors of the renin-angiotensin system. Acute kidney injury was also more common in the intensive group. These complications can potentially be prevented or managed by changing the medications or decreasing the intensity of antihypertensive therapy. Thus, among patients with CKD and hypertension without diabetes, targeting an SBP<120 mm Hg compared with <140 mm Hg reduced rates of major cardiovascular events and all-cause death without evidence of effect modifications by CKD or deleterious effect on the main kidney outcome.

Prof. Carmine ZOCCALI

President, European Renal Association – European Dialysis and Transplant Association (ERA-EDTA)

Institute of Clinical Physiology of Pisa, National Research Center (CNR-IFC), Clinical Epidemiology and Physiopathology of Renal Diseases and Hypertension Unit, Reggio Calabria, Italy

ABSTRACTS

Hypertension Metrics in Renal Transplantation

The prevalence of Hypertension in renal transplant patients is extremely high being close to 90%. Hypertension is a risk factor for renal dysfunction and it was documented an association between Blood Pressure (BP) levels and an increase in the risk of renal deterioration in renal transplant patients. Being hypertension an important and potentially modifiable risk factor it should be properly treated in renal transplant patients.

BP target in renal transplant patients remains unclear. Of note, there are no recommendations for BP targets in renal transplant recipients in the latest European Society of Hypertension (ESH)/European Society of Cardiology (ESC) guidelines BP target in renal transplant patients. Current KDIGO guidelines for kidney transplant patients formally recommend that the diagnosis and the treatment of hypertension be made by conventional, office BP measurements. On the other hand, 24h ambulatory monitoring provides detailed information on the circadian BP profile as well as separate estimates of average day-time and night-time BP and the NICE guidelines now recommend 24h ABPM for the diagnosis of hypertension at community level. Perhaps, one reason cautioning against formal recommendation for the adoption of ABPM for the diagnosis and the monitoring of hypertension in transplant patients in current guidelines is the fact that there is still scarce evidence that the altered BP profile and hypertension, as measured by ABPM, reflect CV burden.

In a paper published in Transplantation in 2014, 24h ABPM and office BP were assessed in CKD and renal transplant patients and it was found that renal transplant patients had a higher prevalence of nocturnal hypertension than CKD patients while no difference was documented in office BP in the two cohorts. Therefore exploring the potential of a better metrics, such as 24hABPM, to assess BP is of paramount importance to better clarify the relevance of it for the care of renal transplant patients.

Prof. Francesca MALLAMACI

Chief, Department of Nephrology, Dialysis and Renal Transplantation, in Reggio Cal;
Head of the Hypertension Unit, European Society of Hypertension Excellence Center;
Research Supervisor at the Institute of Clinical Physiology of the National Research Council (CNR);
Section of Epidemiology and Physiopathology of Renal Diseases and Hypertension of Reggio Calabria, Italy

ABSTRACTS

The AKI to CKD Continuum: a Clinical Perspective

Previously acute kidney injury (AKI) had been believed to be a transient event, and recovery from AKI had been thought to be perfect with no long-term consequences. However, recent epidemiological studies revealed that AKI can result in chronic kidney disease (CKD) and eventually in end-stage kidney disease in the long term.

Transition of AKI to CKD is mediated by multiple mechanisms, and hypoxia of the kidney is now considered to play a critical role. Transient hypoxia of the kidney induces rarefaction of the peritubular capillaries, inflammation and fibrosis after AKI episodes. It should also be noted that epigenetic changes are closely related to hypoxia, and epigenetic changes induced by hypoxia, called "hypoxic memory" can also explain the AKI-to-CKD transition in the long term after complete recovery from the initial AKI episode. These epigenetic changes include long non-coding RNA expression, histone modifications, and chromosomal conformational changes.

It is important to detect AKI at an early stage and limit the kidney injury with an appropriate therapeutic approach. Animal experiments suggest that targeting hypoxia is a promising strategy to block the transition from AKI to CKD. Hypoxia-inducible factor (HIF) is a master regulator of defensive mechanisms, and a number of compounds to active HIF (PHD inhibitors) are now in the phase 2 or phase 3 clinical trials as a novel therapy against anemia in CKD. Utilizing HIF activators (PHD inhibitors) may improve the long-term outcome of AKI. Epigenetic changes can also be a good target to prevent the transition from AKI to CKD and are focus of intensive researches.

Prof. Masaomi NANGAKU

Professor and Head of the Division of Nephrology and Endocrinology;
Executive Councilor of the Asian Pacific Society of Nephrology;
Executive Councilor of the Japanese Society of Nephrology;
Vice President, the University of Tokyo Hospital;
Professor and Head, Division of Nephrology and Endocrinology, The University of Tokyo Graduate School of Medicine, Japan

ABSTRACTS

CVD in PD Patients: Prevalence and Risk Factors

Cardiovascular disease (CVD) is the leading cause of death in patients undergoing dialysis including peritoneal dialysis (PD) and hemodialysis (HD), accounting for 40%-50% of all-cause mortality in these populations. Numerous risk factors play important roles in the development of cardiovascular complications in dialysis patients, which include traditional and non-traditional risk factors. It is recognized that car¬diovascular risk profiles and their management in chronic PD patients may be different from those of chronic hemodialysis patients in several aspects. Our cohort study demonstrated pre-existing CVD contributes significantly to mortality of PD patients. The incident PD patients with prior stroke, reduced left ventricular ejection fraction (LVEF), pulmonary hypertension, diabetes with CVD at the start of PD are associated with poor survival in PD patients. Faster transporter with pre-existing CVD was with higher risk for death compared to those without any history of CVD. Our studies further identify differential PD-related risk factors associ¬ated with higher CVD mortality, including (1) Loss of residual renal function and fluid overload. Overhydration is prevalent in PD patients and positively correlated with systolic blood pressure and CVD. The development of overhydration after loss of residual renal function is probably the most important cardiovascular risk factor specific to PD. (2) Adverse metabolic effects related to glucose-based PD solutions: higher dialysate glucose exposure, high serum triglyceride to high-density lipoprotein cholesterol (TG/HDL-C) ratio, metabolic syndrome, increased glycosylated hemoglobin (HbA1c) level and decreased Alb-glycated serum proteins. (3) Inflammation: peritonitis in patients with longer PD duration, elevated neutrophil to lymphocyte ratio, higher longitudinal serum high-sensitivity C-reactive protein (hs-CRP) levels, etc. (4) Malnutrition: hypoalbuminemia, body mass index (BMI) decline (>0.80%) during the first year of PD, higher malnutrition-Inflammation Score (MIS) and lower visceral protein concentrations. (5) Other risk factors: hyperuricemia, hypokalemia, higher platelets and plateletcrit, an increasing of red blood cell distribution width, etc. Some of risk factors are readily modifiable, and some are non-modifiable. Assessment and management of cardiovascular risk factors, especially the modifiable factors plays a key role in preventing CVD events in dialysis patients.

Prof. Xueqing YU

Dean, Guangdong Academy of Medical Science; President, Guangdong Provincial Hospital, Guangzhou, China

ABSTRACTS

A Decision for the Elderly with Renal Failure: take an old kidney or remain on dialysis

Given the worse results with an expanded criteria donor (ECD) kidney than with a standard criteria donor (SCD) one, it is important to clarify if there is better patient survival after kidney transplantation (KT) using ECD kidneys compared to remaining on the waiting list on dialysis. This is difficult to assess as the comparison between both populations implies unbridgeable biases. The average increase in life expectancy for recipients of ECD kidneys (usually those procured from old donors, with co-morbidities such as hypertension or diabetes or with prolonged cold ischemia time) compared with the waiting list non-transplanted dialysis cohorts is relevant. Classical literature shows that due to excess mortality in the perioperative period, the ECD recipient survival equals the survival observed with SCD or remaining on the waiting list at least 3 years after KT. In other words, according to data published more than a decade ago, it took 3.5 years to justify an ECD KT in terms of survival when this practice was compared to waiting until an SCD was available. The subgroups that show significant ECD survival benefit included old patients, non-Hispanics, unsensitized, recipients with hypertension and diabetics, particularly in those programs with long (>4 years) waiting times.

Albeit the benefits are clear for certain patient populations, patient survival is limited when an ECD KT is performed in high-risk recipients such as retransplantation. Patients 60 years old or older with associated co-morbidities have particularly suboptimal survival results when receiving an ECD KT compared with SCD KT.

In an attempt to minimize confounding factors in a comparison between patients listed who remained on dialysis and those who are transplanted, our group performed a paired-matched analysis between 823 recipients from donors over 65 years and counterparts listed with the same comorbidity. The risk for death was 2.6-fold higher in the dialysis group. Consequently, ECD-KT shows survival advantage over dialysis in the elderly, although undoubtedly SCD offers better survival. In a further analysis, a cohort of 389 KT recipients from donors ≥75 years was analyzed and compared to those who remained listed on dialysis. Even using these extreme aged kidneys, the benefit in survival over dialysis was clear, with 60% less mortality in the transplanted group.

A particular effort should be made in selection of adequate elderly recipients and allograft kidneys with enough quality. But the message is clear: take this old kidney and avoid dialysis.

Prof. Julio PASCUAL

Medical and Patient Care Director, Nephrology Department and Kidney Transplantation Program, Hospital del Mar, Barcelona, Spain

ABSTRACTS

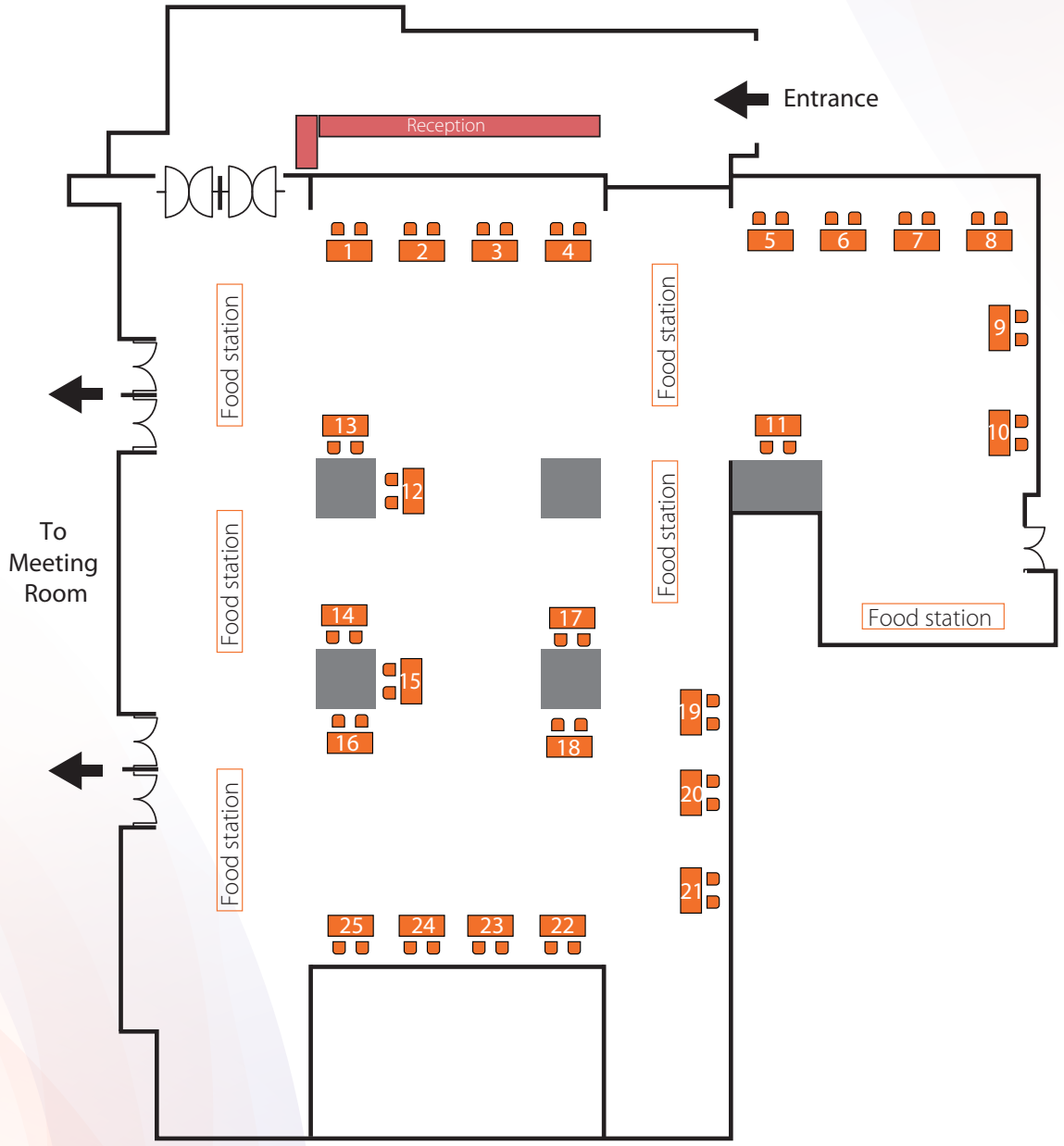
Case Record from the Interhospital Meeting The First ABO-incompatible Kidney Transplant in Hong Kong

ABO-incompatible kidney transplantation is now an established treatment option for patients with end-stage kidney disease. The development of efficient desensitization protocols including apheresis, modulation of B cell immunity and long-term maintenance immunosuppression has enabled ABO-incompatible kidney transplantation to become a safe treatment option that provides comparable patient and graft outcomes to conventional ABO-compatible kidney transplantation. Given the scarcity of deceased organs and long waiting time for deceased donor kidney transplant in Hong Kong, kidney transplantation from blood group incompatible lived donor would be another feasible option to our dialysis patients rather than remaining on dialysis and waiting for deceased donor kidney. Our center had performed the first ABO-incompatible kidney transplantation in Hong Kong. The process of this new service development and clinical progress of the patient would be shared in the presentation.

Dr. Maggie MA

Associate Consultant, Department of Medicine, Queen Mary Hospital, Hong Kong

EXHIBITION FLOOR PLAN



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1-2	Roche
3-4	Astellas
5-6	MSD
7-8	FMC
9-10	Baxter
11	Otsuka
12-13	Novartis
14-16	Kyowa Kirin
17	Boehringer Ingelheim

Booth Number	Exhibitor
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