台灣腎臟醫學會
113年度春季學術演講會

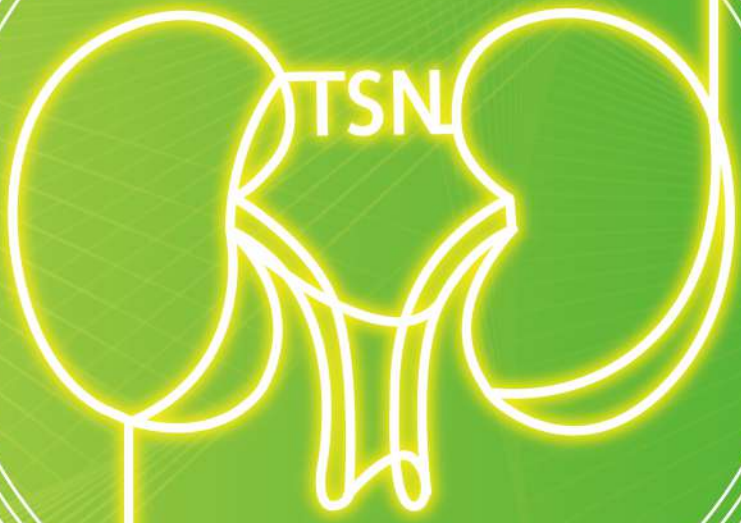
2024 Spring Academic Conference of Taiwan Society of Nephrology

時間 Time

113年04月28日(星期日)
April 28(SUN.),2024

地點 Venue

中國醫藥大學 水湳校區
China Medical University
Shuinan Campus, Taichung, Taiwan



台灣腎臟醫學會
113 年度春季學術演講會

目錄

會員報到、教育積分注意事項

報到：

會員及準會員務必攜帶身份證親自刷卡報到，才給予積分認定。

醫師會員報到

時間：113 年 4 月 28 日(星期日)上午 8:30 至下午 3:00
地點：中國醫藥大學水湳校區卓越大樓 B2 樓報到處

積分認定

✓腎臟醫學會積分：A類15分
✓內科醫學會積分：B類10分

透析護理人員及腎臟照護衛教師報到及刷退

- 已報名且完成繳費者
- 請務必攜帶身分證親自刷到及刷退，才給予積分

【報到及刷退時間】

	刷到時間	刷退時間
4 月 28 日(星期日)	上午 9 時 00 分至上午 11 時 00 分	下午 3 時 00 分至 3 時 30 分

地點：中國醫藥大學水湳校區卓越大樓 B2 樓報到處

積分認定：

✓透析繼續教育積分：1.5 次
✓慢性腎臟病繼續教育積分：15 小時

參展廠商攤位展示區

時間：113 年 4 月 28 日(星期日)
地點：中國醫藥大學水湳校區卓越大樓 B2 樓

午餐資訊

時間：113 年 4 月 28 日(星期日)中午 12:30 至下午 1:20【報到時領取餐券】
地點：中國醫藥大學水湳校區卓越大樓
B201 教室、B202 教室、B203 教室、101 教室、102 教室
各會議室前
【憑券領取餐盒】

Taiwan Society of Nephrology 2024 Spring Academic Conference Program

Date：April 28 (Sunday), 2024
Place：Shuinan Campus, China Medical University, Taichung, Taiwan

Date		April 28, 2024 (Sunday)						
Place / Time		Conference Hall	B201	B202	B203	101	102	B2
AM	08:30 | 15:00	Registration						
	09:00 | 10:30		Kidney Disease Health Prompting Hospital shared course	Symposium 4 Management of Cardiovascular Diseases in Kidney Transplant Recipients	Case Communication 1			Exhibitions
	10:30 | 12:30	【ISN-TSN International Conference】 Advances in Kidney Research: Innovative Approaches and Novel Therapies						
	12:40 | 13:20		Lunch Symposium 1	Lunch Symposium 2	Lunch Symposium 3	Lunch Symposium 4	Lunch Symposium 5	
PM	13:30 | 15:00	Symposium 1 DM, Inflammation, Uremic Toxin Management for CVD	Advanced CME for CKD nurse Interdisciplinary care of chronic kidney disease and cardiovascular disease	Symposium 5 HTN and Hyperlipidemic Control for CVD	Case Communication 2			
	15:10 | 16:40	Symposium 2 Controversies in the Therapeutic Management of Volume in AKI Patients	Symposium 3 Revisit Peritoneal Dialysis with Modern Mindset					

ISN-TSN International Conference I
Topic：Translational Medicine for Future Nephrology

Date ：April 27 (Saturday), 2024
Place：Taipei Medical University, International Conference Hall, 16F

Programme

15:00-15:10 Opening

Prof. Mai-Szu Wu/吳麥斯 理事長
President, Taiwan Society of Nephrology

Chairs：Mai-Szu Wu、Masaomi Nangaku

15:10-15:35 1. Current and future perspectives of telenephrology

Prof. Masaomi Nangaku
Dean, Professor and Head, Division of Nephrology and Endocrinology, The University of Tokyo Graduate School of Medicine, Japan

15:35-16:00 2. Innovative medical device and AI-based software for kidney disease

Prof. Toshio Miyata
Tohoku University Graduate School of Medicine, Japan

16:00-16:25 3. What we learn from multi-omics analyses

Prof. Reiko Inagi
Professor and Chief, Division of CKD Pathophysiology
The University of Tokyo, Graduate School of Medicine, Japan

Chairs：Chih-Wei Yang、Toshio Miyata

16:30-16:45 4. From bench to bedside: Immunometabolic regulation for kidney disease

Huang-Yu Yang/楊皇煜醫師
Division of Nephrology, Linkou Chang-Gung Memorial Hospital

16:45-17:00 5. Expanding biotechnology business landscape: industry-academia collaboration

John Tsung-chun Lee 李宗錞副事業長
Deputy Dean of Office of Business Development,
Taipei Medical University

17:00-17:10 Panel Discussion

17:10-17:15 Closing

Prof. Mai-Szu Wu/吳麥斯 理事長

Taiwan Society of Nephrology
2024 Spring Academic Conference

ISN-TSN International Conference II

Date：April 28 (Sunday), 2024
Place：Conference Hall, Shuinan Campus, China Medical University, Taichung, Taiwan

“Advances in Kidney Research: Innovative Approaches and Novel Therapies”

10:30-10:35 Opening

Prof. Mai-Szu Wu/吳麥斯 理事長
President, Taiwan Society of Nephrology

Chairs：Mai-Szu Wu、Masaomi Nangaku

10:35 – 11:00 1. New drugs for kidney disease; SGLT2 inhibitor, HIF-PH inhibitor, and beyond

Prof. Masaomi Nangaku
Dean, Professor and Head, Division of Nephrology and Endocrinology, The University of Tokyo Graduate School of Medicine, Japan

11:00 – 11:25 2. A kidney disease drug helps fight cancer and aging

Prof. Toshio Miyata
Tohoku University Graduate School of Medicine, Japan

11:25 – 11:50 3. Organelle stress in kidney disease

Prof. Reiko Inagi
Professor and Chief, Division of CKD Pathophysiology
The University of Tokyo, Graduate School of Medicine, Japan

11:50 – 12:15 4. Pericyte-specific targeting for kidney disease and complication

Shuei-Liong Lin/林水龍教授
Division of Nephrology, Department of Internal Medicine, National Taiwan University Hospital

12:15 – 12:25 Panel Discussion

12:25 – 12:30 Photo

【Symposium 1】 Room: Conference Hall

13:30-15:00 DM, inflammation, uremic toxin management for CVD

13:30-13:35
Opening

Chair: 張浤榮/ Horng-Rong Chang
13:35-14:00
1. DM, inflammation, uremic toxin management for CVD
蔡宗翰醫師/ Tsung-Han Tsai
中山醫學大學附設醫院腎臟科
Chung Shan Medical University Hospital

Chair: 王怡寬/ I-Kuan Wang
14:00-14:25
2. The Gordian knot-- Look into the CardioRenal Metabolic Syndrome
張瑋婷醫師/ Wei-Ting Chang
奇美醫院心臟科
Division of Cardiology, Department of Internal Medicine, Chi-Mei Medical Center

Chair: 李玟儀/ Wen-Yi Li
14:25-14:50
3. Targeting endothelium in CKD and related CVD
張芳綺醫師/ Fan-Chi Chang
台大醫院腎臟科
Department of Internal Medicine, National Taiwan University Hospital

14:50-15:00
Panel Discussion

【Symposium 2】 Room: Conference Hall

15:10-16:40 Controversies in the Therapeutic Management of Volume in AKI Patients

慨歎水情，探究急性腎損患者之體液治療

Chair(s): 張智翔/ Chih-Hsiang Chang、林裕峯/ Yu-Feng Lin

15:10-15:15

Opening

15:15-15:40

1. Implementing Green Initiatives in Critical Care: Necessary or Costly?
   施行重症照護理綠色倡議：水到渠成，或是繁費？
   李國華 醫師/ Kuo-Hua Lee
   台北榮民總醫院 腎臟科
   Division of Nephrology, Department of Medicine, Taipei Veterans General Hospital

15:40-16:05

2. Fluid status and fluid responsiveness in AKI patients
   湧泉應變，顧腎患者之流態
   李佳駿 醫師/ Chia-Chun Lee
   國立成功大學醫學院附設醫院 腎臟科
   National Cheng Kung University

16:05-16:30

3. Interdisciplinary treatment strategies: Navigating the challenges and opportunities in volume management for cardiovascular and kidney diseases
   航行河漢，探索心腎疾病之波濤
   邱鼎育 醫師/ Ting-Yu Chiou
   高雄長庚紀念醫院 腎臟科
   Chang Gung Memorial Hospital-Kaohsiung

16:30-16:40

Panel Discussion

【Advanced CME for CKD nurse】 Room：B201

13:30-15:00 **慢性腎臟病與心血管疾病之跨團隊照護**
Interdisciplinary care of chronic kidney disease and cardiovascular disease

Chair: 蔡宜純/ Yi-Chun Tsai

13:30-14:00

1. 心衰竭個案管理照護經驗分享
   Experience of case management of heart failure
   鍾雨珍　心臟衰竭個管師/ Yu-Chen Chung
   台灣大學醫學院附設醫院
   Case manager of heart failure, National Taiwan University Hospital

14:00-14:30

2. 團隊介入提升心肌梗塞病人照護成效 – 藥師角色
   Team intervention improves the care effectiveness for patients with myocardial infarction – the role of pharmacists
   王詩涵 藥師/ Shih-Han Wang
   高雄醫學大學附設醫院
   Pharmacist, Kaohsiung Medical University Hospital

14:30-15:00

3. 運動介入慢性腎臟病衰弱及肌少症之成效
   The effectiveness of exercise on frailty and sarcopenia in chronic kidney disease
   黃心怡 復健治療師/ Hsin-Yi Huang
   台北市振興醫院
   Physiotherapist, Cheng Hsin General Hospital

【Symposium 3】 Room：B201

15:10-16:25 Revisit Peritoneal Dialysis with Modern Mindset

Chairs：吳明儒/ Ming-Ju Wu、楊智宇/ Chih-Yu Yang

15:10-15:15
Opening

15:15-15:45
1. The role of PD in super-aged society
鍾牧圻醫師/ Mu-Chi Chung
臺中榮民總醫院 腎臟科
Division of Nephrology, Department of Internal Medicine, Taichung Veterans General Hospital

15:45-16:15
2. Elderly patients on PD
顏介立醫師/ Chieh-Li Yen
林口長庚醫院 腎臟科
Department of Nephrology, Chang Gung Memorial Hospital

16:15-16:25
Panel Discussion

【Symposium 4】 Room：B202

09:00-10:30 **腎臟移植病人之心血管疾病之治療**
Management of Cardiovascular Diseases in Kidney Transplant Recipients

Chairs： 賴彬卿/ Ping-Chin Lai、陳呈旭/ Cheng-Hsu Chen

09:00-09:30

1. 腎臟移植病人心率不整藥物及介入性治療
   Medical and intervention therapy of arrhythmia in kidney transplant recipients
   謝育整醫師/ Yu-Cheng Hsieh
   台中榮民總醫院
   Taichung Veterans General Hospital

09:30-10:00

2. 腎臟移植病人心衰竭藥物及介入性治療
   Medical and intervention therapy of heart failure in kidney transplant recipients
   郭弘典醫師/ Hung-Tien Kuo
   高雄醫學大學附設中和紀念醫院 腎臟科
   Division of Nephrology, Kaohsiung Medical University Hospital, Kaohsiung Medical University

10:00-10:30

3. 腎臟移植病人冠心症之診斷及介入性治療
   Diagnosis and interventional therapy of coronary heart disease in kidney transplant recipients
   游棟閔醫師/ Tung-Min Yu
   台中榮民總醫院 腎臟科
   Division of Nephrology, Taichung Veterans General Hospital

【Symposium 5】 Room：B202

13:30-15:00 HTN and hyperlipidemic control for CVD

13:30-13:35
Opening

Chair：陳呈旭/ Cheng-Hsu Chen

13:35-14:00
1. News about blood pressure and lipid control in managing cardiovascular disease
   王奇彥醫師/ C.Y. Wang
   台中榮民總醫院心臟內科主治醫師
   Taichung Veterans General Hospital

Chair：吳勝文/ Sheng-Wen Wu

14:00-14:25
2. Optimizing blood pressure management in chronic kidney disease and diabetes: addressing controversies and evidence gaps
   王威傑醫師/ Edy Kornelius
   中山醫學大學附設醫院內分泌暨新陳代謝科主治醫師

Chair：邱炳芳/ Ping-Fang Chiu

14:25-14:50
3. The neurovascular consequences of cardio-renal syndrome: Exploring the pathways to cognitive impairment and stroke
   陳彥中醫師/ Yen-Chung Chen
   彰化基督教醫院神經醫學部主治醫師
   Changhua Christian Hospital

14:50-15:00
Panel Discussion

【腎臟病健康促進機構共學課程】 Room：B201

09:00-10:00 提升腎臟病健康促進機構辦理腎臟病前端防治作業能力

Chair: 蔡宜純/ Yi-Chun Tsai

9:00-9:20
早期腎臟病篩選、診斷及轉介
蔡宜純醫師
高雄醫學大學附設中和紀念醫院 腎臟科

9:20-9:40
提升腎臟前端防治能力_院所經驗分享
杜柏村院長
瑞東診所

9:40-9:55
提升腎臟前端防治能力_院所經驗分享
蕭仕敏衛教師
高雄醫學大學附設中和紀念醫院 腎臟科

Case Communication/病例報告 1

09:00-10:30 Room: B203

【病例討論 1】 主持人：李文欽/ Wen-Chin Lee

09:00 – 09:12 1. 移植腎動脈狹窄
Transplant Renal Artery Stenosis
謝運芳*、徐愷翔
亞東紀念醫院腎臟內科

09:12 – 09:24 2. 以超高效率血液透析，治療透析患者的後天穿透性皮膚病
Use of super high-flux hemodialysis，for treatment of HD Patients with Acquired perforating dermatosis
彭正清
彰化市冠華醫院血液透析中心

09:24 – 09:36 3. 糖尿病腎病變與子癲前症之鑑別診斷考量: 一個病例報告
Differential diagnosis consideration between diabetic nephropathy and preeclampsia: a case report
王昱珩
台中榮民總醫院

09:36 –09:48 4. 抗嗜中性白血球血管炎併發瀰漫性隱球菌感染: 一個病例報告
Disseminated Cryptococcosis Complicating ANCA Associated Vasculitis: A Case Report
陳維哲
台中榮民總醫院

09:48 – 10:00 5. 腎移植病患葡萄球菌相關性腎絲球腎炎: 一個病例報告
Staphylococcus infection-associated glomerulonephritis in a renal transplant patient
王偉陵 徐佳鈿
台中榮民總醫院

10:00 – 10:12 6. 23 歲女性接受血型不相容活體腎臟移植後發生血栓性微血管病變
A 23-year-old young woman with de novo thrombotic microangiopathy after ABO incompatible living donor kidney transplantation
尹玉聰，洪麗玉，廖家德，李明哲，吳美儀
衛生福利部雙和醫院內科部腎臟科、臺北醫學大學醫學院醫學系內科部腎臟科、衛生福利部雙和醫院外科部一般外科、臺北醫學大學泌尿腎臟研究中心

10:12 － 10:24　7．雙側多囊腎栓塞作為復發性腎囊腫出血患者移植前腎臟切除術的替代方案

Embolization of bilateral polycystic kidneys as an alternative to pre-transplantation nephrectomy in a patient with recurrent cyst hemorrhage

劉小櫻，呂岳勳，李明哲，吳美儀，高芷華
衛生福利部雙和醫院內科部腎臟科、衛生福利部雙和醫院放射科、衛生福利部雙和醫院外科部一般外科、臺北醫學大學醫學院醫學系內科部腎臟科、臺北醫學大學泌尿腎臟研究中心

Case Communication/病例報告 2

13:30-15:30 **Room: B203**

【病例討論 2】 主持人：林柏松/ Paik-Seong Lim

13:30 – 13:42 8. 隱藏於糖尿病內的細節：白蛋白-球蛋白比例異常之詮釋
The Details Hidden within Diabetes: Interplay of Albumin-to-Globulin Ratio Reversal in Diabetes Mellitus and IgG4-Related Kidney Disease
柳向芃[1] 丁瑞聰[1] 巫宏傑[1] 王偉傑[1] 陳冬英[2]
[1]衛生福利部桃園醫院內科 腎臟科 [2]台北馬偕紀念醫院 病理科

13:42 – 13:54 9. 36 歲女性，下泌尿道症狀感染合併有雙側膀胱輸尿管逆流
A 36-year-old female with lower urinary symptoms infection and bilateral vesicoureteral reflux
黃雅琳，陳清揚
義大醫院內科部腎臟科

13:54 – 14:06 10. 高敏感腎臟移植的減敏治療
Desensitization in a highly sensitized living donor kidney transplant
陳宜宏、徐愷翔、楊如燁、彭渝森
亞東紀念醫院腎臟內科

14:06 – 14:18 11. 一例以腎周圍腫瘤合併噬血症候群疑似侵襲性淋巴
A case with hemophagocytic lymphohistiocytosis with perirenal tumor suspected aggressive lymphoma
黃翊安 洪毓權

14:18 – 14:30 12. 腹膜透析病人的沉默殺手: 腹膜橫膈膜交通
Pleuroperitoneal communication: A silent killer in peritoneal dialysis patient
侯順方
台中榮民總醫院

14:30 – 14:42 13. 一位二次切片結果為類澱粉沉積症之局部節段性腎絲球硬化症病人
A case of cyclosporine-resistant FSGS revealed renal amyloidosis in a second renal biopsy
鄭佳芸、徐愷翔
亞東紀念醫院腎臟內科

14:42 – 14:54 14. 淋巴癌所引起之免疫複合體沉積腎炎
Immune-complex mediated glomerulonephritis secondary to lymphoma
林佩萱[1], 陳泰迪[2], 陳永昌[1], 田亞中[1], 方基存[1], 楊智偉[1], 塗昆樺[1]
[1]林口長庚醫院腎臟科, [2]林口長庚醫院病理科

14:54 – 15:06 15. C3 glomerulonephritis 伴隨快速進行性腎絲球腎炎
C3 glomerulonephritis with rapid progressive glomerulonephritis
黃怡雯[1], 陳泰迪[2], 陳永昌[1], 田亞中[1], 方基存[1], 楊智偉[1], 塗昆樺[1]
[1]林口長庚醫院腎臟科, [2]林口長庚醫院病理科

Lunch Symposium 1

12:30-13:20 **Room: B201**

Chair: 林俊良/ Chun-Liang Lin

Topic : How does the HVHDF changes dialysis patient QOL and the clinical application?

Speaker: Peter J. Blankestijn
University Medical Center Utrecht

Sponsored by：台灣費森尤斯醫藥股份有限公司/ Fresenius Medical Care Taiwan Co., Ltd.

Lunch Symposium 2

12:30-13:20 **Room: B202**

Navigate CKD care: Empower patients with sustainable management strategies
領航慢性腎臟病: 持續提供策略性慢性腎臟病照護

Chair: 吳明儒/ Ming-Ju Wu

Topic: Management of complications in CKD: One more think while using reno-protective therapy
慢性腎臟病照護：延緩腎功能惡化藥物之使用注意

Speaker: 蔡尚峰醫師/Shang-Feng Tsai
台中榮民總醫院 腎臟科
Taichung Veterans General Hospital

Sponsored by：台灣阿斯特捷利康股份有限公司/ AstraZeneca Taiwan Limited

Lunch Symposium 3

12:30-13:20 **Room: B203**

Chair: 賴彬卿/ Ping-Chin Lai

Topic: The Benefits of Semaglutide to People with Diabetes and Chronic Kidney Disease

Speaker: 楊智超醫師/Chih-Chao Yang
高雄長庚醫院 腎臟科
Chang Gung Memorial Hospital-Kaohsiung

Sponsored by：台灣諾和諾德藥品股份有限公司/Novo Nordisk Taiwan

Lunch Symposium 4

12:30-13:20 **Room: 101**

Chair: 陳呈旭/ Cheng-Hsu Chen

Topic: Unlocking Therapeutic Potential of Renal Anemia in Dialysis Patients: Vadadustat, a Novel HIF-PH Inhibitor

Speaker: 吳家麟醫師/ Chia-Lin Wu
彰化基督教醫院腎臟科
Changhua Christian Hospital

Sponsored by：台田藥品股份有限公司/ Tai Tien PHARMACEUTICALS Co., Ltd.

Lunch Symposium 5

12:30-13:20 **Room: 102**

Chairs : 吳麥斯/ Mai-Szu Wu

Topic: Practical guidance for the use of SGLT2 inhibitors in patients with Cardio-Renal-Metabolic disease

Speaker: 鍾牧圻醫師/ Mu-Chi Chung
台中榮民總醫院腎臟科
Taichung Veterans General Hospital

Sponsored by：台灣禮來股份有限公司/ Eli Lilly and Company (Taiwan)

專題演講
及
Lunch Symposium
摘要

ISN-TSN Internation conference I

Current and future perspectives of telenephrology

Prof. Masaomi Nangaku
Dean, Professor and Head, Division of Nephrology and Endocrinology, The University of Tokyo Graduate School of Medicine, Japan

Telemedicine, the delivery of health care and/or health information using electronic systems, is patient centered and can deliver primary and specialized health care to geographically isolated patients. Telemedicine can also be a solution for shortage of the number of doctors. The systems of telemedicine include synchronous direct physician-patient care through clinical videoconferencing, and asynchronous modalities such as electronic consultation and video telehealth to educate internists about specialized clinical topics.

Advantages of telemedicine include less impact on work and employment as well as patient empowerment and engagement in self-care. Barriers to telemedicine include limited availability about biological data, technological difficulty especially for the elderly, resistance to change and preliminary regulations about telemedicine.

In nephrology, telenephrology can bridge distance and deliver kidney care and education to the isolated. Our current system allows us to obtain physiological parameters once per months at hospitals, but telemedicine will enable us to monitor physiological parameters continuously at home. However, in case of telenephrology, we want to monitor kidney function remotely, and this needs technological innovations.

Reference

https://biomse.t.u-tokyo.ac.jp/moonshot/en/

ISN-TSN Internation conference I

Innovative medical device and AI-based software for kidney disease

Prof. Toshio Miyata
Tohoku University Graduate School of Medicine, Japan

ADVERTISEMENT FEATURE

A HOTSPOT FOR RESEARCH AND DEVELOPMENT OF MEDICAL AI

Medical doctors in Japan are WORKING WITH AI EXPERTS TO DEVELOP NEW HEALTHCARE SOLUTIONS from diagnosis through to treatment.

Using artificial intelligence (AI) to improve medicine will bring untold progress, it has already been used extensively in patient diagnosis. Now, researchers at Tohoku University in Sendai, Japan, are using AI to improve treatments including insulin therapy and hemodialysis.

Recognizing the need to involve AI experts and health tech companies from the beginning of the R&D process, the Tohoku University Graduate School of Medicine, has established the 'Medicinal Hub' linking doctors, AI researchers, and health tech companies to develop AI-based medical solutions for use in the real world.

The driving force behind the Medicinal Hub is Toshio Miyata, a professor in medical science and translational research at Tohoku University Graduate School of Medicine. He says the hub provides a framework where doctors, AI researchers and health tech companies can work together to further develop medical AI.

"Collaboration among doctors and AI researchers is important," he says. "Active involvement of medical doctors, who have experience, medical knowledge and data, is the key for success," he says.

Almost half of the researchers working in the hub come from IT companies, such as NEC Corporation. Ryosuke Togawa, an AI researcher with NEC, has been working with Miyata for three years and says that working with medical experts is invaluable for developing medical AI solutions.

"To develop an AI that stands up in medical practice, it is essential that we have input from medical experts, not only to increase its accuracy, but improve its functions for use in different scenarios," he says.

AI FOR INSULIN THERAPY

"AI that has learned the experience of medical specialists can provide valuable information to non-specialists," says Miyata. In an ideal setting, diabetologists are available to provide intensive insulin therapy to strictly control blood glucose levels and prevent diabetic complications. They have the experience and knowledge to set optimal insulin dosages for different patients.

But in reality, non-diabetologists are often the ones providing the insulin, which can be difficult, as the safe insulin dose range is narrow and excessive doses can cause complications such as hypoglycemia.

"There are not enough diabetologists, especially in rural areas," says Miyata, who is working on a project using AI to support non-diabetologists to perform intensive insulin therapy and to select optimal insulin doses for hospitalized patients.

The AI is based on an algorithm known as Behavioral Imitation Learning Technology, which was originally developed by a researcher in NEC Laboratories America to learn repeated skills of professionals and predict behaviour patterns. Miyata's and Togawa's team customized it for medical use and worked with diabetologists to apply it to solve the issues.

After analysing big data from patients admitted to Tohoku University Hospital over five years, the team has developed a cloud-based AI (DM-SAiL) that provides recommended insulin dosage with a very small error margin.

While the technology is being developed for hospital patients, Miyata hopes it will be developed to support insulin therapy at home in the future.

The project has been selected by the Japan Agency for Medical Research and Development (AMED) and the clinical performance test will be conducted in 2024 with the aim of filing for regulatory approval.

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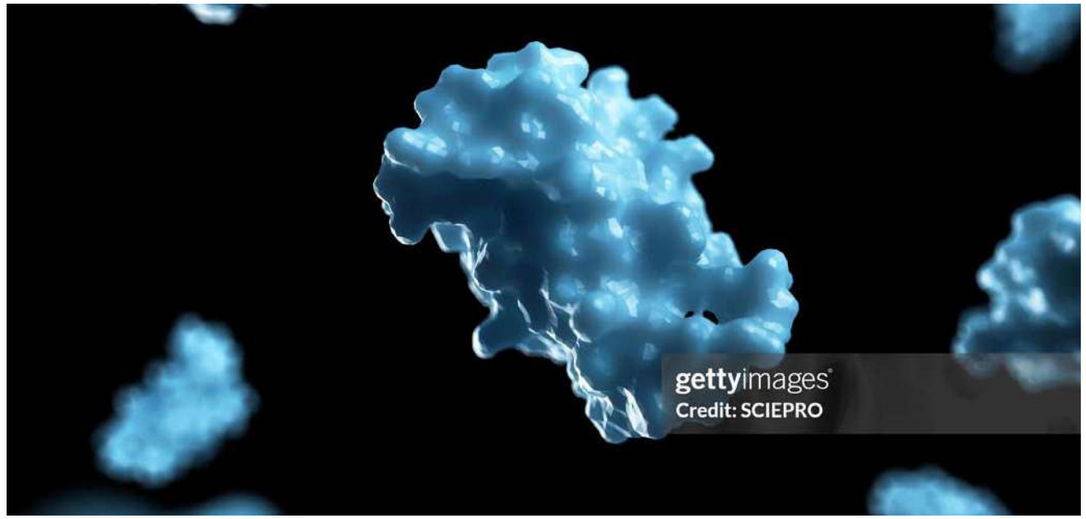
▲ AI could help non-specialist doctors implement hemodialysis treatment (left). The Medicinal Hub at Tohoku University (right).



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▲ AI may be able to help doctors select the optimal insulin (above) dose for patients with diabetes mellitus.

AI FOR HEMODIALYSIS

Another AI project to predict optimal water removal for hemodialysis patients, conducted by Tohoku University, was also selected by AMED.

Led by Miyata and Masaomi Nangaku, a professor at the University of Tokyo Graduate School of Medicine, the project is developing AI which will optimize maintenance hemodialysis and support non-specialist doctors to implement hemodialysis therapy.

In maintenance hemodialysis treatment for people with end-stage kidney failure, blood is drawn from the body and, after correcting electrolytes and removing waste products and excess water, the cleaned blood is returned for circulation.

Appropriate water removal is the most important and difficult part of the procedure — insufficient water removal can impair cardiopulmonary function and excessive water removal can lead to other adverse effects. People on hemodialysis undergo the procedure once every few days.

Using data from 3,000 hemodialysis patients in Japan (approximately 800,000 dialysis sessions), Miyata and Nangaku worked with NEC to train an AI engine to optimize the total amount of water removed and predict the probability of a fall in blood pressure during dialysis. They added personalized data for each patient, such as hemodialysis information, blood test results and patient attributes, to train the model, making it more effective and accurate.

According to Nangaku, the AI can predict the target amount of water removal and the probability of a decrease in blood pressure during dialysis with a high degree of accuracy.

In the future, he hopes the AI can be used for clinical practice as 'software as a medical device' (SaMD) to predict blood pressure lowering and optimal total amount of water removal volume, as well as operating in the cloud and on PCs in medical institutions. Another future development could be an AI equipped hemodialysis machine that controls the amount of water removal and blood flow in real time.

"Development of the AI technology could eventually facilitate home therapy which would reduce pressure on outpatient clinics and improve access in remote areas," he says .

LOOKING AHEAD

While both the insulin therapy and hemodialysis projects are heading for clinical trials in 2024, Miyata says several projects in different fields of medicine are in progress and future initiatives will be selected based on criteria such as abundance of high-quality medical data, involvement of doctors, and potential to commercialize the technology.

"Rather than looking for a medical field where we can utilize a specific AI algorithm, we need to select or develop the optimal algorithm to solve a specific medical issue," he says. "We use the Biodesign principle, where solutions are developed, starting from the needs of the medical field and optimized by envisioning the final product."

Togawa says the emergence of generative AI will have a huge impact on sectors across society, including medical research.

"In the future, generative AI could be used to consolidate existing AI applications. For example, to link diagnostic imaging and electronic medical records to enable consistent analysis that supports human decision making," he says. "But, high-quality data remains essential for developing AI technology. Collaboration with skilled doctors, who can supervise the data, is very important for the research and development of medical AI." ■

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What we learn from multi-omics analyses

Prof. Reiko Inagi
Professor and Chief, Division of CKD Pathophysiology
The University of Tokyo, Graduate School of Medicine, Japan

Recent advanced omics analyses, especially multi-omics analyses, are powerful tools for discovering novel pathophysiological mechanisms of health and disease.
えい In kidney disease, the accumulated data of transcriptomic analysis identifies the alteration of gene expression, which contributes to the development and progression of kidney cell damage, leading to kidney disease. Proteomic and metabolomic analyses robustly validate these findings, elucidating crucial pathophysiological pathways. These advanced discoveries facilitate the development of innovative therapeutic approaches for kidney disease.

In particular, integrated omics data from single-cell RNA sequencing and metabolomic analysis reveal that kidney cells dynamically change the phenotypic population under pathogenic conditions and that these phenotypic changes are often associated with metabolic alteration. Acute and chronic phenotypic changes, such as functional impairment, adaptive responses, and repair reactions, ultimately determine kidney cell fate: cell cycle (senescence), cell death, and pro-inflammatory/fibrotic responses.

Based on omics analysis, recent papers, including ours, demonstrate that tubular cell phenotypic changes are associated with lipid metabolic alterations. In cases of acute tubular cell damage, repaired cells exhibit temporal lipid droplets, suggesting adaptive metabolic alterations for appropriate repair. In contrast, In cases of chronic tubular cell damage, maladaptive tubular damage, such as tubular cell death, is associated with chronic lipid metabolic derangement and organelle stress (mitochondrial stress, ER stress, and altered mitochondria-ER crosstalk), suggesting the lipotoxicity in the progression of tubular cell damage.

The multi-omics approach also provides new insights into the possible mechanisms by which genes or cell types are targets in renoprotective drugs. The latest paper demonstrates that by single-cell expression and chromatin accessibility tools, the renoprotective effects of mineralocorticoid are established through open chromatin and target gene expression primarily in principal and connecting tubule cells and, to a lesser extent, in segments of the distal convoluted tubule cells.
In this presentation, I would like to discuss the advancements in multi-omics analysis, what we have learned from them, and their prospects.

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Expanding biotechnology business landscape: industry-academia collaboration

John Tsung-chun Lee 李宗錞副事業長
Deputy Dean of Office of Business Development,
Taipei Medical University

Modern medicine is advanced through the eye-blazing innovation in technologies, such as telemedicine, artificial intelligence, multi-omics, etc. The translation from bench to bedside and vice versa are gaining more attention and the distance getting shorter. To smoothen the traditional barriers moving from scientific discoveries to practical therapeutics, Taipei Medical University has made tremendous efforts to promote “practical innovation” through (1) “inside-out commercialization of in-house inventions and spinoffs” and (2) “outside-in integrated industry-academia collaboration and newly open Shuang-Ho Biomedical Park”. Shuang-Ho Biomedical Park heralds the unique triple complex of “University-Hospital-Industry” to facilitate the processes from idea to clinics and integrates with the emerging biotechnology ecosystems in Northern Taiwan.

ISN-TSN Internation conference II

New drugs for kidney disease; SGLT2 inhibitor, HIF-PH inhibitor, and beyond

Prof. Masaomi Nangaku

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Previously the only treatment we could employ for patients with chronic kidney disease (CKD) was renin-angiotensin inhibitors. However, the situation is dramatically changing. Many clinical trials targeting kidney disease are running, and the number of drugs targeting kidney disease is increasing in the pipeline of pharmaceutical companies. New drugs have been improved in the field of nephrology, such as SGLT2 inhibitors targeting CKD (1) and HIF-PH inhibitors targeting anemia in CKD (2). CKD and other kidney diseases are experiencing a resurgence as targets for drug discovery and development (3, 4). This momentum is driven by the increasing societal and economic burden of CKD and other kidney conditions, improvement in clinical trial systems (5), and key scientific advances in understanding of the pathogenesis of kidney disease (6).

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ISN-TSN Internation conference II

A kidney disease drug helps fight cancer and aging
Prof. Toshio Miyata
Tohoku University Graduate School of Medicine, Japan

ADVERTISEMENT FEATURE

A KIDNEY DISEASE DRUG HELPS FIGHT CANCER AND AGEING

A drug originally developed for treating kidneys, turns out to BE USEFUL AGAINST CANCER.

Toshio Miyata, a nephrologist in Japan, knew he needed to do something when he had run out of treatment options for his patients. "Kidney inflammation and fibrosis can be life threatening, and there were very few renal medicines available," he says. "So 20 years ago, we decided to make some ourselves."

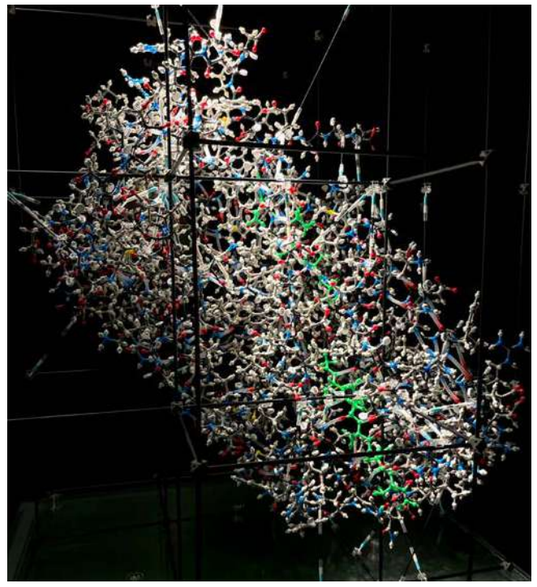
Plasminogen activator inhibitor-1 (PAI-1) — a biomolecule responsible for regulating blood clots — has been on the pharmaceutical industry's list of potential targets for more than three decades. Blood clots, inflammation and fibrosis, and the mechanism that breaks them down are linked to many diseases including kidney, lung, cardiovascular and liver diseases. Patients with inflammation and fibrosis usually get worse. Managing this process could be a game changer.

In 2003, Miyata, a nephrologist and a physician-scientist at the Tohoku University Graduate School of Medicine in Sendai, Japan, became interested in finding small molecules that can inhibit the function of PAI-1. At the time, only a few small-molecule PAI-1 inhibitors had been reported and studies on PAI-1's role in biological function were still in their infancy.

Miyata's lab began searching for PAI-1 inhibitors by performing a virtual screening of a library of chemical compounds against a published structure of human PAI-1. This relies on using 'docking' algorithms to predict the binding between the two structures. After simulating the fit of almost 2.25 million compounds, they finally discovered their first hit, a compound they indexed as TM5007[1].

Initial studies of TM5007 on rodent models for blood clots, showed promise — the compound reduced significant blood clots. But its efficacy was too low to be a clinically useful treatment.

Miyata and his team continued to refine the compound to improve its efficacy, but the journey was long. After 15 years and having synthesized more than 1,400 compounds, they finally arrived at TM5614, a compound 3,000 times more efficacious than their original hit. They are now ready to take this compound to clinical studies.



▲ A crystal structure of human plasminogen activator inhibitor-1 (PAI-1).

STEM CELL INTERACTION

The longer they spent developing TM5614, the more Miyata's team understood PAI-1 biology. While analysing its crystal structure, they learned that PAI-1 binds to a protein called furin, which activates the release of hematopoietic stem cells — immature cells that develop into blood cells — from the bone-marrow environment. When TM5614 binds to a target site on PAI-1, it activates furin, which drives the release of these stem cells.

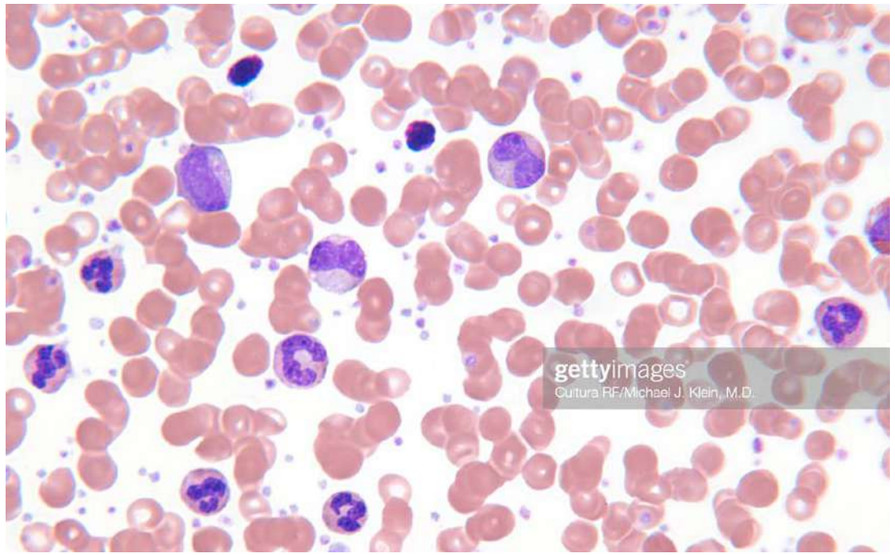
Miyata's team took this idea to Hideo Harigae, a hematologist and director of Tohoku University Hospital, who saw the potential of TM5614 as an adjunct therapy for chronic myelogenous leukemia (CML). "CML is a rare disease," explains Harigae. "It's a type of blood cancer that develops when abnormal genes occur in the hematopoietic stem cells."

Earlier studies on PAI-1 and cancer had focused mostly on its function in thrombotic and fibrinolytic events. While newer findings have hinted at their complex roles in various cancer progression, no-one has ever reported PAI-1 activity on hematopoietic stem cells[2].

CML patients are usually treated with tyrosine kinase inhibitors (TKIs), which act on mature CML cells that differentiated from CML stem cells. While TKIs improve the survival rate of CML patients, they do not address the root cause of the disease. Because TKIs cannot act on CML stem cells that reside in the bone-marrow environment, the cancer often recurs when the treatment is stopped. Meanwhile, long-term TKI use is expensive and carries potentially fatal side effects.

To overcome this, Miyata's team suggested a strategy that combines TKI with a PAI-1 inhibitor to treat CML. The idea is to use PAI-1 inhibitors such as TM5614 to lure CML stem cells out of their bone-marrow environment. Once these cells are released, TKIs can attack the exposed cells.

Miyata's team tested this combination therapy on a CML mouse model and found that the number of CML cells remaining in the bone marrow fell considerably, leading to a greater survival rate.



▲ Blood affected by chronic myelogenous leukemia. Tohoku University researchers have found a useful adjunct therapy for the rare cancer.

Cultura RF/Michael J. Klein, M.D./Image Source/Getty

Following this result, the team proceeded with early and late phase II clinical trials[3] in which 33 patients received a daily dose of TKI. Those who received TM5614 on top of their daily dose of TKI for a year achieved a higher rate of deep molecular remission, a desirable outcome for TKI treatment.

CHECKPOINT INHIBITORS

Other groups had shown that a high expression level of PAI-1 is associated with low survival rates in patients with solid cancer. But the mechanism behind this is poorly understood. Miyata's team further discovered that PAI-1 may have induced the expression of immune checkpoint molecules on cancer cells and suppresses the anti-tumour immune response.

Immune checkpoint molecules such as programmed death-1 (PD-1) and its ligand (PD-L1) have become popular targets for cancer immunotherapy. While these inhibitors do not act directly on cancer cells, they allow immune T-cells to remain active and attack cancer cells. Despite their promise, the therapeutic effect of anti-PD1/PD-L1 is still limited. "An antibody-based drug is expensive and immune-related side effects are a serious problem," says Miyata. "There's a need for a combination drug that increases the response rate of anti-PD-1/PD-L1 with fewer side effects and is less expensive."

Miyata's team have showed that TM5614 can suppress tumour growth and enhance the anti-tumour response of anti-PD-1 antibodies in mouse models of melanoma, non-small lung cancer and colorectal cancer. In a phase II clinical study in malignant melanoma, 7 out of 29 patients who initially did not respond well to nivolumab (an antibody drug) began to respond to the drug after 8 weeks of combination therapy with TM5614[4].

The team has now scheduled other phase II clinical trials for non-small-cell lung cancer and cutaneous angiosarcoma to further confirm the effect of TM5614 on immune checkpoint inhibition in other cancers.

A SURPRISE FINDING

The discovery of TM5614 illustrates what translational medicine could look like in academia. "Previously, the School of Medicine mainly conducted biological research," says Harigae. "But the times have changed." He saw the development as an effort for academic research labs to fill in the clinical needs. "Pharma companies don't develop all the medicines we need," he says. "So we have to do it ourselves."

Miyata's team has collaborated both nationally and internationally and partnered with contract research organizations to accelerate research progress. In 2017, through a collaboration with scientists at Northwestern University in Illinois, United States, they found another surprising role for PAI-1. After surveying the Amish community in the US state of Indiana, they discovered that those without the PAI-1 gene on average lived 10 years on average longer than those with the gene[5].

This unexpected finding was only possible through a worldwide academic collaboration and open resource sharing that his team adopted, Miyata says. The team is currently exploring the potential use of PAI-1 inhibitor as an anti-ageing medication.

"Drug discovery and development is very challenging, but also very important," says Harigae. "I hope young researchers and physician-scientists will follow us in the future." ■

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ISN-TSN Internation conference II

Organelle stress in kidney disease
Prof. Reiko Inagi
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Organelles, including the nucleus, mitochondria, endoplasmic reticulum, Golgi apparatus, and lysosomes, play various important roles that are necessary for the cell to function properly. Recent multi-omics analyses reveal that cellular abnormalities observed in disease phenotypes are linked to organelle dysfunction, such as mitochondrial stress and ER stress. Maintaining organelle homeostasis is crucial for determining cell fate.

We have been studying organelle stress and demonstrated that maladaptive ER stress response, specifically the unfolded protein response (UPR) pathway, contributes to kidney cell damage in both acute and chronic kidney disease. For example, maladaptive UPR pathway-mediated cell damage in tubular cells, podocytes, and EPO-producing cells leads to inflammation and fibrosis. Maladaptive UPR pathway-mediated cell damage also induced metabolic alteration with mitochondrial stress. Our recent study demonstrated that the rapid decline of kidney function in DKD patients is correlated with lipid metabolic alteration (Lysophosphatidylcholine accumulation in the ER) within tubules, which is associated with tubular organelle stress. The connection between organelle stress and metabolic reprogramming suggests a novel approach for developing CKD/DKD treatments.

Further, advanced organelle research focuses on intra-organelle interaction and organelle crosstalk. The UPR pathway regulates not only ER function but also mitochondria-ER interaction through gene expression related to lipid metabolism. Tubular mitochondrial DNA induces inflammation via the activation of a sensor molecule, STING, located on the ER lumen, suggesting the novel inflammatory response mediated by the mitochondria-ER interaction. The contact site of mitochondria and ER, named mitochondria-associated ER membrane (MAM), has recently been highlighted because MAM is key for lipid metabolism as well as calcium signaling. We found that proximal tubular cell death in DKD is caused by a decrease in MAM function followed by lipid metabolic alteration, leading to cell death associated with organelle stress.

In my talk, I will summarize the updated pathophysiology of organelle stress and its impact on organelle crosstalk in kidney disease.

ISN-TSN Internation conference II

Pericyte-specific targeting for kidney disease and complication

Shuei-Liong Lin/林水龍教授
Division of Nephrology, Department of Internal Medicine, National Taiwan University Hospital

Pericytes are interstitial mesenchymal cells found in many major organs. In the kidney, microvascular pericytes are defined anatomically as extensively branched collagen-producing cells in close contact with endothelial cells. Although many molecular markers have been proposed, none of them can identify the pericytes with satisfactory specificity or sensitivity. The roles of microvascular pericytes in kidneys were poorly understood in the past. Recently, by using genetic lineage tracing to label collagen-producing cells or mesenchymal cells, the elusive characteristics of the pericytes are illuminated. In the healthy kidney, pericytes are found to take part in the maintenance of microvascular stability. Detachment of the pericytes from microvasculature and loss of close contact with endothelial cells are observed upon kidney injury. Kidney pericytes are shown to be the major source of scar-forming myofibroblasts in progressive kidney disease. Targeting the crosstalk between pericytes and neighboring endothelial cells or tubular epithelial cells may inhibit the pericyte-myofibroblast transition, prevent peritubular capillary rarefaction, and attenuate kidney fibrosis. In addition, kidney pericytes produce erythropoietin in healthy kidneys by sensing the change of oxygenation and hemoglobin concentration. However, the ability of erythropoietin production decreases in pericytes-derived myofibroblasts in chronic kidney disease, leading to renal anemia. Recent advances on epigenetics create a new field to study erythropoietin gene expression at chromatin level. Demethylating agent has shown the restoration of erythropoietin expression as well as downregulation of α smooth muscle actin in myofibroblasts. Through this talk I would like to share the knowledge in the physiology and pathophysiology of kidney pericytes, and our recent research on pericyte-specific drug delivery for kidney disease and complication.

Symposium 1

The Gordian knot-- Look into the CardioRenal Metabolic Syndrome

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奇美醫院心臟科
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CardioRenal Metabolic Syndrome (CRMS) encompasses cardiovascular, renal, and metabolic disorders that exacerbate each other, increasing the risk of cardiovascular events and kidney dysfunction. Risk factors include obesity, hypertension, insulin resistance, dyslipidemia, and chronic kidney disease. Pathophysiology includes impaired insulin signaling, oxidative stress, inflammation, and activation of various systems. Management requires a comprehensive approach including lifestyle modifications, pharmacotherapy, and targeted interventions. Further research is needed for understanding and managing this condition. Updates in the 2023 Standards of Care in Diabetes recommend weight loss, lower blood pressure thresholds, and new medication options like SGLT2 inhibitors and finerenone. Emerging clinical trials explore high-dose oral semaglutide, retatrutide, artificial pancreas, and beta cell replacement therapies, aiming to enhance diabetes care and patient outcomes. In this talk, I will also introduce our research focusing on association between renal dysfunction and cardiovascular diseases.

Symposium 2

Implementing Green Initiatives in Critical Care: Necessary or Costly?

施行重症照護理綠色倡議：水到渠成，或是繁費？

李國華 醫師/ Kuo-Hua Lee
台北榮民總醫院 腎臟科
Division of Nephrology, Department of Medicine, Taipei Veterans General Hospital

在當前全球面臨環境挑戰的背景下，醫療領域，尤其是重症照護，也在尋求可持續發展的道路。實施環保措施在重症照護中變得越來越受到關注，但這一過程是否必要，以及是否成本效益，一直是一個熱烈討論的話題。本文旨在探討在重症照護部門實施環保措施的必要性與成本效益，並提出一系列基於當前研究和案例分析的見解。

首先，實施環保措施對於降低醫療部門的碳耗用量至關重要。重症照護部門因其高耗能、高物資消耗而對環境造成顯著的負面影響。透過節能、節水設備、採用可重複使用的耗材，不僅可以減少對環境的負擔，還能改善醫院內外的空氣品質，間接提升病人和員工的健康狀況。

其次，從長遠來看，環保措施能夠為醫院帶來經濟效益。雖然初期投資可能較高，但通過能源節約和減少資源浪費，可以在長期內節省大量成本。此外，隨著社會對環保意識的提高，環保醫院的形象也能吸引更多患者和優秀醫護人員，從而提高醫院的競爭力。

然而，實施這些措施的成本和實踐挑戰不容忽視。重症照護部門的首要任務是提供高品質的病人照護，任何新措施都不能妨礙這一點。因此，找到既能保障病人照護品質又能實現環境可持續性的策略，是一項挑戰。此外，高額的前期投資和技術更新可能會對醫院的財務狀況造成壓力。

綜上所述，實施環保措施在重症照護中是一項必要且長遠來看可能帶來成本效益的投資。然而，如何平衡病人照護的直接需求與環境可持續性的長期目標，需要進一步的研究和創新解決方案。未來的研究應該集中在量化環保措施的經濟和環境效益，並探索在不妨礙病人照護品質的前提下實施這些措施的最佳方法。

Symposium 2

Fluid status and fluid responsiveness in AKI patients

湧泉應變，顧腎患者之流態
李佳駿 醫師/ Chia-Chun Lee
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Fluid supplementation had been an important treatment during resuscitation for circulatory failure in acute kidney injury (AKI) patients or those at risk of AKI development. Although fluid administration could restore tissue hypoperfusion and microcirculatory impairment, fluid accumulation and resultant fluid overload brought adverse outcome. Past studies had approved that increased mortality, increased ventilator dependence and increased major adverse kidney events were found in critical AKI patients with fluid overload. Since administrated fluid, once given, could not be withdrawn back in the critical AKI patients and could possibly bring poor outcome. In addition, routine fluid bolus therapy was found to be effective in only half of the patients in previous cohort studies and review. Concept of dynamic fluid management strategy for shock patients comprised of “ROSE”, that is “Resuscitation”, “Optimization”, “Stabilization” and “Evacuation”, had been proposed. Thus, we need to evaluate our patients’ fluid responsiveness wisely and precisely.

The methods of fluid responsiveness evaluation could be divided into static and dynamic indices. Static indices, like urine output or central venous pressure, were found to have low prediction value for fluid responsiveness and they were not suggested to be used. On the other hand, dynamic parameters, for example, passive leg raising test (PLRT), pulse pressure variation (PPV), stroke volume variation (SVV) or end expiratory occlusion test (EEOT), etc., were found to have higher prediction value and were recommended to be adopted to evaluate the need of fluid administration. Each of the method had its own strength and limitation and clinician could choose appropriate method based on clinical scenarios and accessibility of available devices. Moreover, fluid intolerance was also suggested to be monitored at the same time via point of care ultrasound to avoid unnecessary fluid administration.

In conclusion, we should evaluate fluid responsiveness in circulatory failure patient with AKI or at risk of AKI and then administered fluid at appropriate timing to maintain hemodynamic stability and avoid fluid overload consequence.

Symposium 2

Interdisciplinary treatment strategies: Navigating the challenges and opportunities in volume management for cardiovascular and kidney diseases

航行河漢，探索心腎疾病之波濤
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Maintaining high quality of care for complex diseases such as kidney and cardiovascular diseases requires a holistic approach based on the concept of multidisciplinary care, in which experts from different disciplines collaborate to optimize management for the physical and psychological needs of the patients. Volume management is especially important for these patients. We will discuss the traditional key members of this multidisciplinary team and their intervention strategies. Emerging new members or tools to meet the challenges and enhance quality of care will also be discussed.

Advanced CME for CKD nurse

心衰竭個案管理照護經驗分享
Experience of case management of heart failure
鍾雨珍　心臟衰竭個管師/ Yu-Chen Chung
台灣大學醫學院附設醫院
Case manager of heart failure, National Taiwan University Hospital

The management of heart failure requires a comprehensive approach that addresses the complex needs of patients, involving both acute treatment and long-term care strategies. Case management in heart failure is an interdisciplinary process designed to ensure that patients receive optimal, coordinated care. Here are some key elements and experiences in the case management of heart failure:
multidisciplinary team approach、patient education and self-management、medication management、monitoring and follow-up、coordination of care、outcome measurement and quality improvement.

Advanced CME for CKD nurse

團隊介入提升心肌梗塞病人照護成效 – 藥師角色
Team intervention improves the care effectiveness for patients with myocardial infarction – the role of pharmacists

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Pharmacist, Kaohsiung Medical University Hospital

高醫心內團隊從 2020 年籌備 JCI 心肌梗塞疾病別認證開始，組成跨領域團隊，包含心內心外醫師、急診部、家醫戒菸團隊、藥學部、營養部、護理部、復健部、呼吸治療組、社會服務室等團隊，透過每個月召開 CV 照護團隊會議，凝聚團隊共識與建立本院臨床照護計畫。本次藉由 30 分鐘課程，和大家高醫 CV 團隊的運作方式，包含收案標準、跨職類介入時間、溝通渠道、系統平台、衛教工具等，藥師的部分還有協助用藥提示系統建置，以提升本院心肌梗塞病人的用藥與照護品質。

Symposium 3

The role of PD in super-aged society

鍾牧圻醫師/ Mu-Chi Chung
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Division of Nephrology, Department of Internal Medicine, Taichung Veterans General Hospital

隨著全球人口老齡化，老年患者需要透析治療的比例不斷增加。腹膜透析作為一種替代性腎替代治療方式，對於許多老年慢性腎病患者來說，提供了更多的靈活性和生活質量。然而，實施腹膜透析在老年患者群體中也面臨諸多挑戰，包括腹膜透析相關感染、患者的生理狀態變化以及透析過程中的管理問題。

本次演講將深入探討腹膜透析在老年患者中的適應症、實施策略以及如何克服相關的臨床挑戰。我們將分析最新的臨床研究數據，並分享一系列成功案例，展示如何有效地管理老年患者的腹膜透析，使其達到最佳的治療效果。此外，本演講還將討論未來腹膜透析技術的發展方向，以及如何在臨床實踐中更好地應用這些技術，以提升老年患者的整體治療效果和生活質量。

Symposium 3

Elderly patients on PD

顏介立醫師/ Chieh-Li Yen
林口長庚醫院 腎臟科
Department of Nephrology, Chang Gung Memorial Hospital

台灣的人口老化增加的速度在全世界可說是名列前茅，在短短的 7 年時間就從高齡化社會進展到超高齡社會。隨著台灣的人口老化，慢性腎臟病進入洗腎的年齡也不斷上升，目前有 6 成的患者開始洗腎的年齡都超過 65 歲，甚至其中 3 成更超過 75 歲，高齡洗腎已經成為我們腎臟科的常態。然而在這群高齡洗腎患者多半都是選擇血液透析，選擇腹膜透析的比例很低，只有百分之五左右。高齡長者不選擇腹膜透析究竟是因為真的預後較差?還是只是臨床醫師以及高齡患者的偏見?本課程將會介紹近期的健保資料庫研究，嘗試回答高齡洗腎患者選擇腹膜透析的優點以及缺點，同時也會介紹在治療高齡腹膜透析病患時，會遇到的困難以及治療策略。

Symposium 4

腎臟移植病人心率不整藥物及介入性治療
Medical and intervention therapy of arrhythmia in kidney transplant recipients

謝育整醫師/ Yu-Cheng Hsieh
台中榮民總醫院
Taichung Veterans General Hospital

Arrhythmias represent a significant challenge in the management of kidney transplant recipients due to the complex interplay between cardiac and renal health. The incidence of arrhythmias is notably higher in patients with renal failure, exacerbating risks associated with kidney transplant procedures. This talk will delve into the multifaceted nature of arrhythmias in the context of chronic kidney disease (CKD), highlighting the enhanced susceptibility of kidney transplant recipients to atrial fibrillation (AF), ventricular tachyarrhythmias, and other rhythm disturbances. The discussion will explore the common and uncommon causes linking CKD to arrhythmogenesis, including the impact of electrolyte imbalances, autonomic dysfunction, and structural cardiac changes.

Furthermore, the talk will examine the current strategies and challenges in treating arrhythmias in this patient group. Pharmacological interventions remain the cornerstone of arrhythmia management, yet their efficacy and safety are significantly influenced by renal function. The application and limitations of non-pharmacological therapies, such as catheter ablation and cardiac implantable electronic devices (CIEDs), will be discussed with an emphasis on procedural risks and the importance of individualized treatment planning.

The coexistence of CKD and arrhythmias complicates medical management, necessitating a holistic and nuanced approach. By addressing these challenges, healthcare professionals can improve outcomes and quality of life for kidney transplant recipients. The talk will underscore the importance of interdisciplinary collaboration and ongoing research to refine therapeutic strategies and optimize care for this vulnerable patient population.

Symposium 4

腎臟移植病人心衰竭藥物及介入性治療

Medical and intervention therapy of heart failure in kidney transplant recipients

郭弘典醫師/ Hung-Tien Kuo
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Cardiovascular disease is the leading cause of mortality following kidney transplantation (KT). Heart failure(HF) is a major cause of morbidity and mortality in patients with end stage renal disease, with a reported prevalence among dialysis patients of 12–36 times that of the general population. Preexisting cardiomyopathy impacts KT outcomes, a functioning KT can also influence the magnitude and clinical course of preexisting cardiomyopathy. KT is associated with improvement in EF over time. Restoration of eGFR and reversal of the uremic milieu may play a role in restoration or improvement of myocardial mechanics and function in patients with preexisting cardiomyopathy. On the other hand, HF post KT remains a major contributor to CVD-related hospitalizations after KT. There are limited controlled data on the optimal pharmacotherapy of HF specific to KT recipients. There is conflicting evidence on the efficacy of RAASi and HF outcomes in KT recipients. Currently, there are limited data on the impact of other goal-directed medical therapies including beta blockers, vasodilators and mineralocorticoid receptor antagonists on HF outcomes after KT, highlighting the need for future studies to define best strategies for the use of these agents in HF after KT. In the section, recent evidence of medical and intervention therapy for HF in KT recipients will be reviewed.

Symposium 4

腎臟移植病人冠心症之診斷及介入性治療

Diagnosis and interventional therapy of coronary heart disease in kidney transplant recipients

游棟閔醫師/ Tung-Min Yu
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腎臟移植病人術後擁有良好的生活品質，但是造成病人死亡的風險因素仍然高於一般人，造成的原因包括感染、 癌症、 心血管疾病。 其中心血管疾病高於其他原因。腎臟移植病人在移植後死亡的原因，無論是移植後短期內死因或是長期追蹤的死因，心血管疾病都是這群腎臟移植病人重要的死亡因素。心血管疾病之中，包含心臟衰竭 、肺動脈高壓 、心律不整 跟 心臟冠狀動脈疾病。而造成腎臟移植病人心臟血管原因的因素包括一般傳統的因子和腎臟移植病人特有的風險因子包括免疫抑制藥物 、 植移植腎功能不佳 、移植後糖尿病等等原因。而在移植手術後，則以移植腎功能延遲恢復、急性排斥、免疫抑制劑、移植後新發生之糖尿病等風險因子為主。

在術後照顧用藥上藥物我要特別注意免疫抑制藥物與 心血管藥物的交互作用 而造成不良的副作用

Symposium 5

Optimizing blood pressure management in chronic kidney disease and diabetes: addressing controversies and evidence gaps

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中山醫學大學附設醫院內分泌暨新陳代謝科主治醫師

Chronic kidney disease (CKD) presents a significant health challenge often compounded by hypertension, which accelerates renal decline and heightens cardiovascular risk. Effective blood pressure (BP) control is pivotal in mitigating these risks and improving outcomes in CKD patients. This presentation aims to explore the rationale behind stringent BP management, discuss optimal targets, and address ongoing debates surrounding BP control to prevent cardiovascular disease (CVD), especially in the CKD and diabetes

Controlling BP in CKD is critical to prevent renal damage and cardiovascular complications. Hypertension exacerbates CKD progression by inducing glomerular hyperfiltration and renal fibrosis, while also contributing to heightened cardiovascular morbidity and mortality. Lowering BP levels has consistently demonstrated benefits in retarding CKD progression and enhancing patient outcomes.

However, determining the optimal BP control strategy remains a subject of debate, particularly in CKD and diabetes. This presentation seeks to elucidate the evidence supporting various BP targets and management approaches in this specific patient population. By synthesizing findings from clinical trials, meta-analyses, and observational studies, it aims to provide insights into the most effective BP control strategies tailored to the prevention of CVD.

Lunch Symposium 1

How does the HVHDF changes dialysis patient QOL and the clinical application?

Peter J. Blankestijn
University Medical Center Utrecht

Several studies have suggested that patients with kidney failure may benefit from high-dose hemodiafiltration compared with standard hemodialysis. Yet, uncertainty prevails, given limitations of the various published studies.
In CONVINCE, patients with kidney failure were randomized to high-dose hemodiafiltration (convection volume of ≥23L/session) or conventional high-flux hemodialysis. Primary outcome was all-cause mortality. Key secondary outcomes included cause-specific mortality and patients reported outcomes.
Overall, 1360 patients were randomized. Median follow-up was 30 (IQI 27 – 38) months. All-cause mortality was 23% lower in the high-dose hemodiafiltration group than in the high-flux hemodialysis group (NEJM 2023).
In this presentation, further results of CONVINCE will be discussed. Additionally, the results will be discussed in the context of the totality of the existing evidence on hemodiafiltration and possible next steps will be summarized. Finally, some practical suggestions on how to implement hemodiafiltration in every day clinical practice will be discussed.

Lunch Symposium 2

Management of complications in CKD: One more think while using reno-protective therapy

慢性腎臟病照護：延緩腎功能惡化藥物之使用注意

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在社會文明的發展下，生活日趨富足。然而，隨之而來的文明病也逐漸增加，其中慢性腎臟病是一個日益嚴重的問題，

根據目前最新 2024 KDIGO 治療指引建議，延緩慢性腎臟病惡化的策略包含包含嚴格的血壓及血脂控制，以及在適當的病人族群與時機接受 SGLT2 抑制劑作為第一線治療，直到需要進行透析或腎臟移植。此外，新型的 MRA 藥物的出現也為慢性腎臟病的治療提供了新的方向。

慢性腎臟病的照護不僅在於延緩洗腎發生、減少死亡風險外，更重要的是不論在藥物或本身疾病進程下預防並控制併發症的發生。這些併發症包括電解質失衡、貧血、骨質疏鬆等，對患者的生活品質和預後都有著重要的影響。

因此，在慢性腎臟病照護不斷的發展下，藉由醫師對於治療指引臨床見解與分析，策略性的減緩未來慢性腎臟病照護的共病症對病人的影響，以改善病人的生活品質。

Lunch Symposium 3

The Benefits of Semaglutide to People with Diabetes and Chronic Kidney Disease

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高雄長庚醫院 腎臟科
Chang Gung Memorial Hospital-Kaohsiung

糖尿病患者的心腎共病在心型降糖藥誤了開發與臨床實證下，逐漸受到治療關注，而糖尿病合併肥胖議題也因為有效的藥物進展引起廣泛的討論。Semaglutide 是一種胰高血糖素樣肽-1 受體激動劑（GLP-1RA），過往的心血管研究證實，Semaglutide 除了具有良好的血糖控制、體重減輕、降低收縮壓的益處外，
Semaglutide 可以顯著減少糖尿病患者尿白蛋白/肌酐比率，改善巨白蛋白尿（UACR >300 mg/g）。

值得注意的是，目前 Semaglutide 可用於治療末期腎臟病（ESRD）的糖尿病患者，不同劑型的 semaglutide 亦能提供患者依其生活型態的個人化治療方案。

未來的大型臨床研究一旦證實 semaglutide 對於減緩腎臟疾病的進展並降低因腎臟或心臟疾病而死亡的風險的影響，將有助於患有糖尿病性腎臟病的患者帶來更多的治療選擇。

Lunch Symposium 4

Unlocking Therapeutic Potential of Renal Anemia in Dialysis Patients: Vadadustat, a Novel HIF-PH Inhibitor

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腎性貧血是慢性腎臟病（CKD）晚期常見的併發症，對患者的生活品質和預後有著深遠影響。本次演講將重點討論腎性貧血的病理機制、目前治療方法的局限性，以及新興療法 Vadadustat 的潛力和臨床應用前景。

腎性貧血的主要原因包括紅血球生成素（EPO）產生不足、鐵利用障礙和尿毒素影響等。目前，促紅血球生成激素（ESA）和鐵劑是治療腎性貧血的主要策略。然而，高劑量 ESA 治療存在增加心血管事件風險的潛在問題，而對於低劑量 ESA 治療延緩透析開始或降低透析患者死亡風險的效果，學界仍存在著不同看法。鐵劑的使用雖然在一定程度上改善了血紅素生成，但其副作用及最佳用量仍需進一步探討。

近年來，缺氧誘導因子脯氨酸羥化酶抑制劑（HIF-PH inhibitor）Vadadustat 的出現，為腎性貧血治療提供了新的方向。Vadadustat 通過模擬低氧環境下的生理反應，穩定低氧誘導因子（HIF），從而促進 EPO 的產生和改善鐵代謝，以達到治療腎性貧血的目的。臨床試驗數據表明，Vadadustat 在提升血紅素水平方面與傳統 ESA 治療相當，且心血管安全性相似，為患者提供了一種口服的治療選項，有望提高患者的便利性和治療遵循性。

然而，儘管 Vadadustat 顯示出良好的治療潛力和安全性，其在實際臨床應用中仍面臨著多方面的挑戰。例如，如何確定最適宜的治療劑量、治療過程中如何監測和管理潛在的副作用、以及如何在透析患者中實施個體化治療等。此外，對於 Vadadustat 長期使用的效果和安全性，仍需更多的臨床數據支持。
總結，腎性貧血的治療是一個不斷發展和變化的領域。Vadadustat 作為一種新型 HIF-PH 抑制劑，其獨特的作用機制和治療潛力使其成為未來腎性貧血治療的有力候選。隨著更多臨床研究的開展，我們期待 Vadadustat 能夠為透析 CKD 患者提供更有效、更安全的治療選項，改善患者的生活品質，並最終實現腎性貧血治療的突破。

病例報告
摘要

病例報告 1

1-1

移植腎動脈狹窄

Transplant Renal Artery Stenosis

謝運芳*、徐愷翔
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亞東紀念醫院腎臟內科
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This 55-year-old man has underlying diseases of hypertension, type 2 diabetes mellitus, non–ST segment elevation myocardial infarction, coronary artery disease: triple vessel disease status post CABG and coronary artery stenting, heart failure with reduced ejection fraction, and end stage kidney disease.

Regarding diabetic related end stage kidney disease, he received deceased donor kidney transplantation 2 years ago. Unfortunately, he had 2 episodes of T cell mediated rejection, at 3 months and 2 years post transplantation respectively. Over the course of 2 years post transplantation, he had multiple episodes of heart failure with acute kidney injury requiring temporary dialysis.

He visited the emergency department with sudden onset of shortness of breath on admission morning and was admitted to the cardiac ward for congestive heart failure. Beta blockers, angiotensin 2 receptor blockers (ARB), sodium-glucose transport protein 2 (SGLT2) inhibitors and diuretics were prescribed for heart failure. Oliguria then anuria were noted 2 days later and intermittent dialysis was initiated. He was under prednisolone 5mg and cyclosporine 25mg before and during admission. With the fear of possible calcineurin inhibitor nephrotoxicity, cyclosporine was changed to mycophenolic acid. Acute kidney injury after adding ARB raised the alarm for possible transplant renal artery stenosis. Transplant renal artery angiogram revealed 90% stenosis of the transplant renal artery and stent was deployed. Noticeable renal function recovery and fair urine output were noted after renal artery stenting. Cardiac angiogram showed no in-stent restenosis of coronary arteries and excluded CAD related congestive heart failure. The patient was discharged dialysis-free and is under regular OPD follow-up with both a cardiologist and a nephrologist.

Based on the literature review, transplant renal artery stenosis (TRAS) is a potentially curable cause of hypertension, allograft dysfunction, and graft loss. The definitive diagnosis requires invasive angiographic techniques. Rapid onset of acute renal failure after using ARB is an alarming sign. Percutaneous transluminal angioplasty (PTA) with stent is the treatment of choice in TRAS.

關鍵字: 移植腎動脈狹窄、急性腎損傷、血液透析、腎臟移植
Keyword: Transplant renal artery stenosis, Acute kidney injury, Hemodialysis, Renal transplant

病例報告 1

1-2

以超高效率血液透析，治療透析患者的後天穿透性皮膚病

Use of super high-flux hemodialysis，for treatment of HD Patients with Acquired perforating dermatosis

彭正清
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彰化市冠華醫院血液透析中心

背景: 後天穿透性皮膚病是一種好發於慢性腎衰竭與糖尿病患者的皮膚疾病，大約有4.5-11%的透析患者會罹患此病，患者開始會以許多發癢皮膚突起皮疹表現，典型病灶是紅色或深色丘疹伴隨中央角質突出、好發於軀幹、四肢和臉部，嚴重的會伴隨劇癢，目前傳統療法效果相當有限。最近由於有超高通量膜(Super high-flux membrane)上市，所以我們嘗試以超高通量膜血液透析的方式來治療這類患者。

方法: 這是一位 39 歲的男性患者，因為糖尿病(IDDM)腎病變，已接受高效率血液透析(High-flux HD) 6 年，2 年前開始出現穿透性皮膚病變，最近 3 個月快速惡化且合併嚴重的皮膚搔癢，經口服抗組胺藥及外用藥膏無效後，建議患者改採用超高效率血液透(Super high-flux HD)，來治療此病灶。我們使用的超高通量膜的透析器是 Elisio 21HX Nipro，患者的透析處方及設定與之前的高效率血液透析方式相同。

結果: 這位患者成功的從高效率血液透析轉換成超高效率血液透析，皮膚搔癢的症狀在 2 周後即改善，穿透性皮膚病 2 個月後明顯改善。(附:治療前及治療後照片及說明)

結論: 這是首次嘗試使用透析療法來治療後天穿透性皮膚病，超高效率血液透析，可以有效的治療透析患者的後天穿透性皮膚病和皮膚搔癢症，超高效率血液透析法簡單容易操作，將有助於它的推廣嘉惠這類疾病的患者。

關鍵字: 超高效率血液透析(Super high- flux hemodialysis)、後天穿透性皮膚病(Acquired perforating dermatosis)

病例報告 1

1-3

糖尿病腎病變與子癲前症之鑑別診斷考量：一個病例報告

Differential diagnosis consideration between diabetic nephropathy and preeclampsia: a case report

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Abstracts

Background：

Both diabetic nephropathy and preeclampsia have clinical presentation of proteinuria. A 32 years old woman, who had type 2 diabetes mellitus and pregnancy with gestational age of 20+5 weeks, was referred to our hospital due to generalized edema, acute kidney injury, and heavy proteinuria.

Methods：

Hypertension (185/88 mmHg) was noted. Blood test showed hypoalbuminemia (albumin: 2.2), renal function impairment (BUN/Cr: 38/5.79), hypercholesterol (LDL: 143) and hypogammaglobulinemia (IgG: 212). Urinalysis revealed proteinuria (protein 3+, 24-hour urine UPCR: 25144g). Nephrotic syndrome was impressed.

Results：

Renal biopsy reported severe mesangial proliferative diabetic nephropathy, class IIb. Features of focal segmental glomerulosclerosis and focal active tubulointerstitial nephritis were found. Although there was no evidence of endocapillary damage or fibrin thrombi found under light microscopy, the possibility of preeclampsia cannot be completely excluded. For assessment of possibility and risk of preeclampsia, sFlt-1/PlGF ratio was 53.6 (cut-off value: 38).

Conclusions：

After admission, conservative treatment for blood pressure control and furosemide for edema were kept. Regular hemodialysis was conducted due to fluid overload and uremia. However, due to limited clinical improvement, whereas preeclampsia still could not be ruled out, we had discussed with the patient and she agreed for termination. Afterwards, methylprednisolone was given for suspected secondary FSGS. As renal function had partial improvement, hemodialysis was discontinued, and the patient was discharged.

Key words：

Preeclampsia, Diabetic nephropathy, Nephrotic syndrome, Pregnancy.

病例報告 1

1-4

抗嗜中性白血球血管炎併發瀰漫性隱球菌感染：一個病例報告

Disseminated Cryptococcosis Complicating ANCA Associated Vasculitis: A Case Report

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Abstracts

Background：

ANCA associated vasculitis (AAV) is a severe condition sometimes leading to pulmonary-renal syndrome with devastating outcomes. Cryptococcosis poses a significant complication in AAV patients. We present a case of disseminated cryptococcosis in a 76-year-old female with a history of AAV with pulmonary renal syndrome receiving 2 courses of double filtration plasmapheresis, 8 courses of plasma exchange, and methylprednisolone pulse(250mg) for 3 days following by oral steroid about 4 months ago.

Methods：

The patient's clinical presentation, laboratory findings, imaging results, and management strategy were documented. Blood test showed renal function impairment (BUN/Cr: 38/4.14) and positive cryptococcus antigen. Brain computed tomography did not reveal evidence of intracerebral hemorrhage nor recent cerebral infarction. Lumbar puncture was arranged.

Results：

Lumbar puncture revealed lymphocytosis and positive cryptococcus antigen in the cerebrospinal fluid (CSF), with elevated opening pressure. Cryptococcus neoformans was isolated from both serum and CSF cultures. External ventricular drain placement was necessary due to concerns of increased intracranial pressure. Induction therapy for disseminated cryptococcosis was promptly initiated.

Conclusions：

AAV patients, particularly those with pulmonary-renal syndrome, are at risk for catastrophic outcomes. Complications of AAV treatment, though rare, can be devastating. Despite minimal immunosuppressant use, our case underscores the potential for disseminated cryptococcosis. Timely CSF analysis is crucial for diagnosis and management in such cases.

Key words：

ANCA associated vasculitis, pulmonary renal syndrome, cryptococcosis

病例報告 1

1-5

腎移植病患葡萄球菌相關性腎絲球腎炎：一個病例報告

Staphylococcus infection-associated glomerulonephritis in a renal transplant patient

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Abstracts

Background：

Staphylococcus infection-associated glomerulonephritis (SAGN) is rare in renal transplant recipients and typically exhibits IgA dominance. A 59-year-old male underwent a kidney transplant in 2011 due to diabetic nephropathy. He developed abrupt proteinuria post-cellulitis of the left foot and presented to the emergency department due to progressive generalized edema, decreased urine output, and worsening dyspnea on exertion.

Methods：

Blood tests showed hypoalbuminemia (albumin: 2.4g/dL), renal function impairment (BUN/Cr: 42/1.74mg/dL), and elevated CRP (21.2mg/dL). Urinalysis revealed proteinuria and hematuria (protein 4+, blood 3+), with UPCR of 6488mg/g. IgA elevation was noted (717mg/dL). Left 3rd toe wound culture revealed Staphylococcus aureus (OSSA). Staphylococcus infection-associated glomerulonephritis was suspected.

Results：

The renal biopsy of graft kidney reported proliferative and focal necrotizing glomerulonephritis with mesangial expansion. IF revealed extensive deposition of IgA and C3 in the mesangium and capillary wall. EM showed scant subepithelial hump-like deposits, compatible with postinfectious glomerulonephritis.

Conclusions：

Upon admission, antibiotics (clindamycin and ceftaroline) were administered for infection. Steroid dosage was titrated to Methylprednisolone 16mg BID, but renal function persisted. Subsequently, steroid dosage was adjusted to Methasone 5mg BID. Osteomyelitis scan confirmed the diagnosis, and debridement was performed. Steroids were gradually tapered as renal function improved. The patient was followed up in the outpatient clinic.

Key words：

Staphylococcus-associated glomerulonephritis, Renal transplant, Acute kidney injury

病例報告 1

1-6

23 歲女性接受血型不相容活體腎臟移植後發生血栓性微血管病變

A 23-year-old young woman with de novo thrombotic microangiopathy after ABO incompatible living donor kidney transplantation

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This 23-years-old woman has a medical history of dysfunctional uterine bleeding and end stage kidney disease due to crescentic immunoglobulin A nephropathy. She has been undergoing intermittent hemodialysis for the past six months. The planned course of treatment involves an ABO incompatible kidney transplantation from her father (donor blood group B+, recipient blood group O+, with 3 HLA mismatch). Pre-transplant assessments revealed negative anti-HLA antibodies with anti-A and anti-B isoagglutinin titers of 1/256 and 1/64, respectively. The desensitization protocol included five sessions of double filtration plasmapheresis and administration of rituximab (200 mg given 5 days before transplantation). Tacrolimus, sodium mycophenolate and prednisolone were also initiated seven days prior to transplantation. At the time of transplantation, anti-B isoagglutinin levels was 1/32. Basiliximab induction therapy was administered on days 0 and 4 (20 mg each).

The patient experienced a prompt recovery of diuresis following reperfusion, but developed severe anemia and thrombocytopenia on postoperative day 1. Hemoglobin level dropped from 7.8 g/dl to 5.3 g/dl and platelet count decreased from 182,000/mm3 to 20,000/mm3, accompanied reticulocytosis and fragmented RBC on blood smear. Lactate dehydrogenase levels was 1132 IU/L and haptoglobin level was < 8 mg/dL, consistent with features of thrombotic microangiopathy (TMA). Anti-B isoagglutinin levels stayed at 1/8 and tacrolimus trough level was 6.1 ng/mL. Despite treatment with double filtration plasmapheresis and intravenous immunoglobulin (6g followed plasmapheresis) for post-transplant TMA, renal function continued to decline (serum creatinine rise from 2.38 mg/dl to 3.08 mg/dl), and thrombocytopenia did not improve. Subsequently salvage therapy with eculizumab 300mg was initiated on postoperative day 6. Following four sessions of plasmapheresis and eculizumab therapy, platelets count increased to 156,000/mm3 and renal function improved (serum creatinine declined to 1.75 mg/dl). The patient was discharged on postoperative day 13 and the immunosuppressive regimen was maintained. The absence of autoimmune disease or antiphospholipid syndrome, along with negative genetic analysis for complement pathway abnormalities, supported the diagnosis of de novo TMA. Two months post-transplantation, the patient demonstrated stable renal function and hematological parameters (serum creatinine 0.8 mg/dl, eGFR 84mL/min/1.73 m2, platelets count 217,000/mm3).

TMA is a rare but serious complication of kidney transplantation, with various etiologies, including antibody-mediated rejection, immunosuppressive drug toxicity, and complement dysregulation. Prompt recognition and intervention are essential in optimizing outcomes for patients at risk of TMA post-transplantation. Our case report demonstrates the successful management of de novo TMA following ABO incompatible kidney transplantation with early initiation of eculizumab therapy.

病例報告 1

1-7

雙側多囊腎栓塞作為復發性腎囊腫出血患者移植前腎臟切除術的替代方案

Embolization of bilateral polycystic kidneys as an alternative to pre-transplantation nephrectomy in a patient with recurrent cyst hemorrhage

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Our patient, a 45-year-old man with blood group A+, presented with gross hematuria and left flank pain for three days. He had experienced similar episodes three times in the past year. As computed tomography showed suspected intracystic tumor bleeding, left nephrectomy was considered. However, renal biopsy revealed a hematoma only. The patient had significant hypertension since age 30 and was diagnosed with autosomal dominant polycystic kidney disease (ADPKD) at 35, with no known family history of any renal cystic disease.

With advancing chronic kidney disease secondary to ADPKD, he was preparing for a living donor kidney transplantation from his 46-year-old sister (with A+ blood). Donor's computed tomography angiography showed suitable vascular anatomy for transplantation. Renal scan revealed reduced estimated glomerular filtration rates (eGFR) in both kidneys: 66.8 mL/min/1.73m² in the left and 50.0 mL/min/1.73m² in the right. Although pre-transplantation evaluation identified three HLA mismatches, the crossmatch was negative. The patient's magnetic resonance imaging revealed numerous bilateral renal cysts, including old hemorrhagic cysts, and new hemorrhage over the left kidney's anterior cysts. The total kidney volume measurements of his left and right kidneys were 17x12.1x23.9cm (2458mL) and 13.9x13.9x27.3cm (2637mL) respectively. Transcatheter arterial embolization (TAE) of both renal arteries before transplantation was recommended as the patient refused nephrectomy.

Upon admission, the patient appeared pale. His body mass index was 22.84 kg/m². Vital signs were normal except for elevated blood pressure (165/90 mmHg). Physical examination revealed bilateral palpable flank masses. Laboratory investigations showed normocytic anemia (Hb, 8.0 g/dL) and impaired kidney function (eGFR, 5.2 mL/min/1.73m²). TAE of the left renal artery was conducted on November 2, 2023, followed by hemodialysis; and TAE of the right renal artery was done on November 6, 2023. The patient experienced post-embolization syndrome, including fever, bilateral flank pain and severe constipation with ileus, which improved gradually after medical management. The kidney transplantation from his sister was successfully performed on November 21, 2023, with immediate urine production noted postoperatively. The patient's graft kidney demonstrated normal function, with a resistive index indicating appropriate perfusion. An immunosuppressive therapy consisting of glucocorticosteroid, tacrolimus and myfortic was initiated. The patient's postoperative course was uneventful. His eGFR was 90 mL/min/1.73m² on POD 4. He was discharged on POD 8. Regular follow-up appointments were arranged to ensure adherence to immunosuppressive therapy and the stability of his condition.

In conclusion, for patients with end-stage ADPKD and recurrent renal cyst hemorrhage requiring kidney transplantation, TAE of polycystic kidneys represents a successful alternative to pre-transplantation nephrectomy. This less invasive approach offers effective control of both hemorrhage and reduced kidney size, facilitating successful renal transplantation and improving patient outcomes.

病例報告 2

2-1

隱藏於糖尿病內的細節：白蛋白-球蛋白比例異常之詮釋

The Details Hidden within Diabetes: Interplay of Albumin-to-Globulin Ratio Reversal in Diabetes Mellitus and IgG4-Related Kidney Disease

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Abstract

We reported a 71 year-old man with type-2 diabetes mellitus, hypertension and dyslipidemia. He had been diagnosed with type-2 diabetes mellitus for 10 years and is under metformin, empagliflozin and insulin glargine for control, with fair blood sugar and proteinuria (UPCR: 300-400 mg/g) But fast declining of renal function was found, with eGFR decreasing from 77 to 9 in past one year. Renal sonography revealed fair kidney size and parenchyma thickness. Glomerulonephritis survey revealed serum albumin-globulin ratio reversal (A/G reverse), and paraprotein (free IgG κ and λ chain) in serum immunofixation electrophoresis. Bone marrow biopsy pathology found no evidence of plasma cell myeloma. Kidney biopsy found lymphoplasmacytic and eosinophil-rich inflammatory infiltrates with irregular fibrosis, IgG4/IgG > 40%, compatible with IgG4-related tubulointerstitial nephritis. Also, segmental thickening of glomerular basement membrane (GBM) (>700 nm), and subendothelial widening with fluffy material was found via electron microscopy, indicating developing diabetic kidney disease (DKD). By the pathology result, corticosteroid was administered for IgG4 related tubulointerstitial nephritis. Gradually improving of renal function was then noted.

The relationship between diabetes and IgG4-related kidney disease involves intricate pathological mechanisms and potential clinical implications. IgG4-related kidney disease, characterized by autoimmune-mediated inflammation and fibrosis in the renal parenchyma, often leading to tubulointerstitial nephritis and renal dysfunction. Although distinct in etiology, overlapping clinical presentations between DKD and IgG4 related kidney disease would be found. The diagnostic challenges lies on the recognition of abnormal renal function declination distinct from nature DKD progression, the initiating of glomerulonephritis survey, and the further interpreting and differentiation diagnosis of albumin-globulin ratio reversal.

Key words: Diabetes mellitus, Diabetic kidney disease, Albumin-to-Globulin Ratio Reversal, IgG4-Related Kidney Disease

病例報告 2

2-2

36 歲女性，下泌尿道症狀感染合併有雙側膀胱輸尿管逆流

A 36-year-old female with lower urinary symptoms infection and bilateral vesicoureteral reflux

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Abstracts

A 36-year-old woman was a case with previous medical history of recurrent urinary tract infection, chronic insomnia and anxiety disorders. She had frequency, urgency and nocturia (2-3 times per night) for lone time. She also had admission history of dilated common bile duct and was diagnosed to have chronic cholangitis in 2018. This time, she presented with fever, dysuria, frequency, urgency, flank pain (Right > Left), bilateral legs and foot edema over one week. Urine frequency and urgency were also complained. She ever visited local clinic and took oral antibiotic for urinary tract infection, but in vain. Then she came to our emergency department and was admitted for suspected acute pyelonephritis.

During admission, kidney echo showed bilateral hydronephrosis, and later abdomen-pelvis CT showed swelling of right kidney, bilateral hydronephrosis and hydroureter, and irregular urinary bladder wall thickening. Dilated biliary tract and common bile duct were also noted. Cystoscope was done and revealed mild trabeculatory bladder wall with erythematous change over left lateral wall, suspect interstitial cystitis with bilateral vesicoureteral reflux was suspected. With the impression of interstitial cystitis and chronic cholangitis, a through history taking and workup will be presented.

Key words：interstitial cystitis, chronic cholangitis, vesicoureteral reflux

病例報告 2

2-3

高敏感腎臟移植的減敏治療

Desensitization in a highly sensitized living donor kidney transplant.

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This 58-year-old woman has a past medical history of diabetic kidney disease with end-stage kidney disease under intermittent hemodialysis thrice weekly since 2017, type II diabetes mellitus, essential hypertension. She has obstetric history of G3P2AA1. There was no history of massive blood transfusion, nor previous allograft. She has been waitlisted for deceased donor kidney transplant since 2020/3. Living donor kidney transplant evaluation was initiated since 2023/6, with her daughter being the eligible donor. HLA matching showed 1A1B1DR mismatch. She is highly sensitized, presented with class I panel reactive antibody (PRA) 100% , and class II PRA 73%. Donor specific antibody was positive for A24, B60 and DR11. Lymphocytotoxic test with complement-dependent cytotoxicity crossmatch (CDC-XM) showed negative for both T cells and B cells. Admission was arranged in 2023/8 for scheduled desensitization and preparation for living donor kidney transplant. Desensitization with double filtration plasmapheresis, IVIG, Rituximab and Bortezomib were prescribed 8/13~8/29. Repeated PRA level was 64% for class I, and 73% for class II. Induction with Basiliximab for kidney transplant was given. Living donor kidney transplant was performed smoothly on 8/29, with cold ischemia time of 4 hours and 25 minutes. The graft kidney presented immediate graft function, with urine output 3230ml on 8/29.

However, urine output decreased to 10~40ml/Hr on post-operative day 2 (POD2, 8/31). Accelerated rejection was suspected. Graft kidney biopsy was performed on 8/31. Pathology showed weak focal positive of C4d on PTC, peritubular capillaritis and interstitial inflammation. Acute antibody-mediated rejection is highly suspected, whilst T-cell mediated rejection can not be fully excluded. Therefore, Methylprednisolone pulse, IVIG, plasmapheresis were administered, and Tacrolimus drug level was titrated up. Urine output improved on POD8 (9/6) after treatment, and serum creatinine level decreased to a nadir of 0.7mg/dL on POD17 (9/15).

Based on literature review, widely accepted desensitization protocol for kidney transplant includes antibody apheresis, intravenous immunoglobulin, and Rituximab. Other agents including Imlifidase, proteosome inhibitor such as Bortezonib, and Eculizumab were also reported

關鍵字: 活體腎臟移植, 去敏治療
Keyword: Living donor kidney transplant, desensitization

病例報告 2

2-4

一例以腎周圍腫瘤合併噬血症候群疑似侵襲性淋巴

A case with hemophagocytic lymphohistiocytosis with perirenal tumor suspected aggressive lymphoma.

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Abstracts

Background：

A 64-year-old man with history of prostate cancer status post treatment, admitted for fever with splenomegaly, bicytopenia(anemia/thrombocytopenia), and elevated serum LDH level.

Methods：

Abdomen CT showed enhancing lymph nodules in left perirenal and periureteral region with left upper ureter encasement, thickening of left Gerota's fascia and peritoneum. Positron emission tomography(PET) scan was arranged, and revealed strong-Fluorodeoxyglucose(FDG)-avid lesion in left supraclavicular nodes, paracardial node, bilateral adrenal glands, spleen, hepatic hilum, upper abdominal para-aortic nodes, left renal fascia/nearby left lateroconal fascia, left peri-renal space, left proximal ureter/peri-ureter region, left external iliac nodes and bone marrow. Bone marrow was arranged, and the pathology reported hemophagocytosis with some atypical lymphocyte.

Results：

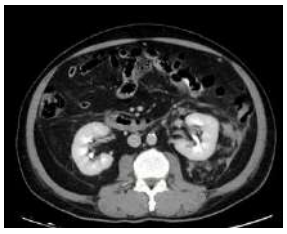
Aggressive lymphoma with bone marrow involvement was highly suspected.

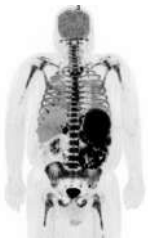
Conclusions：

For perirenal lymphadenopathy with persistent high fever, aggressive lymphoma needed to be considered.

Key words：

Perirenal lymphoma, hemophagocytic lymphohistiocytosis





病例報告 2

2-5

腹膜透析病人的沉默殺手: 腹膜橫膈膜交通

Pleuroperitoneal communication: A silent killer in peritoneal dialysis patient

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Abstracts

Background：

Pleuroperitoneal communication in peritoneal dialysis patient is a rare complication that tends
to develop 4–8 weeks after initiation of peritoneal dialysis (PD) in patients with end stage renal disease. A high index of suspicion is the key to establish an early diagnosis. We presented a 65-year-old female underwent renal transplantation in Mainland China in 2000 due to bilateral urothelial carcinoma s/p laparoscopic bilateral nephroureterectomy and progressive dyspnea after CAPD initiation since two weeks ago due to graft failure.

Methods：

Serial exams were done. Chest X-ray showed bilateral pleural effusion. Blood tests showed leukocytosis with left shift(WBC: 23770 /μL, Neu: 94%:), hypoalbuminemia (albumin: 2.5 g/dL), total protein 6000 mg/dL, elevated CRP (31.4mg/dL). CAPD fluid exam revealed high TNC level(5271/μL, Neu:95%) and culture showed Campylobacter fetus later. Right pleural effusion analysis demonstrated parapneumonic pleural effusion.(Total protein: 2300mg/dL, LDH:813 U/L, Glucose: 250 mg/dl, TNC: 11338/μL, Neu:85%). Due to high blood sugar level in pleural effusion, pleuroperitoneal communication was suspected.

Results：

Right pigtail was placed by CS doctor due to suspected empyema with high TNC level. A CT peritoneography revealed discontinued left hemidiaphragm with leakage from peritoneal cavity to left thorax.

Conclusions：

Therefore, after discussion with patient, patient was transitioned to hemodialysis via right femoral permcath temporarily after completion of antibiotic course and accepted AV fistula operation later. Patient accepted regular hemodialysis in local HD clinic smoothly till now.

Key words：

Pleuroperitoneal communication, Renal transplant, SVC syndrome, Campylobacter fetus

病例報告 2

2-6

一位二次切片結果為類澱粉沉積症之局部節段性腎絲球硬化症病人

A case of cyclosporine-resistant FSGS revealed renal amyloidosis in a second renal biopsy

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A 63-year-old man with history of nephrotic syndrome and osteogenesis imperfecta was referred to our hospital for deterioration of proteinuria under glucocorticoid treatment. The first renal biopsy revealed focal segmental glomerulosclerosis at the previous hospital in Feb. 2022 and glucocorticoid treatment was given. ANA, C3, C4 and RPR all showed negative. Urine protein /creatinine ratio was 9098 mg/g in Jan. 2023 treated with pulse Methylprednisolone. Cyclosporine was applied since Feb. 2023. However, poor response of proteinuria and general edema were noted in spite of increasing cyclosporine. Urine protein/creatinine ratio was 14389 mg/g in May 2023 and 38893 mg/g in Sep. 2023. Secondary causes of FSGS were suspected and cancer surveys were done in Nov. 2023 for glucocorticoids and cyclosporine resistance FSGS. Abnormal serum Kappa/Lambda light chain ratio 0.029 was noted. Bone marrow biopsy revealed interstitial (15%) plasmacytosis with aberrant CD56 expression and lambda light chain restriction. Light chain MGRS (monoclonal gammopathy with renal significance) was impressed. A second renal biopsy revealed positive amyloid staining by Congo red. The immunofluorescent study shows granular deposition of IgG(-), IgA(-), IgM(-), Kappa(-), Lamda(+). Systemic light-chain amyloidosis was diagnosed. Chemotherapy with Cyclophosphamide, bortezomib, dexamethasone and Thalidomide were given. Complication of herpes zoster over left C3-C4 dermatomes noted in Jan 2024. Neutropenic fever, right leg cellulitis with septic shock, acute kidney injury and acute respiratory failure were noted in Feb 2024. The patient passed away on Feb 14th 2024.
According to the literature review, plasma cell disorders may cause FSGS. Therapy for the underlying plasma cell disorder can lead to resolution of FSGS. Appropriate timing of a second kidney biopsy is important.

關鍵字:蛋白尿、輕鏈蛋白相關之類澱粉沉積症、抗藥性局部節段性腎絲球硬化症
Key word: Proteinuria, light-chain amyloidosis, glucocorticoids/cyclosporine-resistant FSGS

病例報告 2

2-7

淋巴癌所引起之免疫複合體沉積腎炎

Immune-complex mediated glomerulonephritis secondary to lymphoma

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Abstract

Immune complex glomerulonephritis is characterized by the presence of immune complexes within the glomerulus, comprising both immunoglobulin and complement components. These triggers complement system activation and inflammatory cell recruitment, leading to glomerular injury. Histologically, immune complex glomerulonephritis often presents as a injury membranoproliferative pattern. Various conditions, including chronic infections, hematologic disorders, and autoimmune diseases, have been associated with immune complex glomerulonephritis.

Here, we present the case of a 59-year-old male with a medical history of type 2 diabetes mellitus diagnosed in 2005 and hepatitis B carrier status. His kidney issues were first noted in June 2023, with reduced GFR, proteinuria, and gross hematuria. Kidney ultrasound showed normal-sized kidneys with no structural lesions. Laboratory tests revealed the intermittent presence of ANA, anti-dsDNA, PR3-ANCA, and RF. Due to persistent decline in GFR, a kidney biopsy was performed in February 2024, suggesting immune complex-mediated glomerulonephritis. Given the underlying hepatitis B carrier status, immunohistochemical studies for HBsAg and HBc Ag were done with negative result, along with undetectable HBV viral load in blood test. Autoantibody screening for ant-Sm/SSA/SSB was also negative. To rule out hematologic malignancy, bone marrow biopsy and whole-body CT were conducted. Bone marrow study showed hypocellularity with no signs of leukemia or lymphoma. CT scan revealed multiple diffusely enlarged lymph nodes in various regions. Core-needle biopsy of a left neck lymph node confirmed marginal zone lymphoma. The patient was subsequently referred to the Hematology clinic for further lymphoma treatment.

This case underscores the diagnosis and management of immune complex-mediated glomerulonephritis and emphasizes the importance of a thorough work-up for accurate diagnosis of underlying diseases, enabling timely initiation of appropriate management.

關鍵字: 免疫複合體沉積腎炎、淋巴癌、自體免疫。
Keyword: immune-complex mediated glomerulonephritis, lymphoma, autoimmune.

病例報告 2

2-8

C3 glomerulonephritis 伴隨快速進行性腎絲球腎炎

C3 glomerulonephritis with rapid progressive glomerulonephritis

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C3 glomerulopathies represent a collection of rare kidney diseases characterized by complement dysregulation, leading to complement C3 deposition in kidney biopsy samples. Currently, there is no universally agreed-upon optimal treatment approach for C3 glomerulonephritis.

This is a case of a 68-year-old male with a medical history significant for chronic kidney disease stage 3, presenting with sub-nephrotic proteinuria and microhematuria, diagnosed as C3 glomerulonephritis in January 2021. Additionally, he has a history of dyslipidemia, bilateral cataracts status post-phacoemulsification with intraocular lens implantation on August 14, 2023, and anxiety with chronic insomnia for 30 years. The patient has been regularly followed up for his chronic kidney disease at the outpatient department. The patient's renal abnormalities were first noted in 2020, with sub-nephrotic proteinuria and microscopic hematuria. A kidney ultrasound reported normal-sized kidneys with right renal cysts. Laboratory examinations revealed a C3 level of 79.8 mg/dL, and immunofixation electrophoresis in both blood and urine showed no monoclonal protein present. A kidney biopsy performed on January 7, 2021, revealed findings suggestive of C3 glomerulonephritis. Prednisolone 5mg twice daily was initiated in February 2021. However, the patient experienced edema, deteriorating renal function, and nephrotic syndrome since December 2023. A second kidney biopsy conducted on December 26, 2023, also revealed findings consistent with C3 glomerulonephritis but without crescent formation. Considering the rapid progression of glomerulonephritis and inadequate control of C3 glomerulonephritis, the patient was admitted for further pulse therapy and plasma exchange.

This case highlights the diagnosis and treatment of C3 glomerulonephritis, as well as the importance of a second biopsy in cases of rapidly progressive glomerulonephritis for accurate diagnosis and timely initiation of appropriate management.

Keyword: C3 glomerulonephritis, rapid progressive glomerulonephritis, complement dysregulation
關鍵字: C3 腎病變、快速進行性腎絲球腎炎、補體系統異常調控

交通資訊

日期：113 年 4 月 28 日(星期日)
地點：中國醫藥大學水湳校區卓越大樓 B2 樓
(台中市北屯區經貿路一段 100 號)

前往方式:

『公車資訊』

1. 中鹿客運 525 號，「經貿大鵬路口站」下車 步行約 6 分鐘抵達校本部。
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3. 豐原客運 850 號，「大德國中站」下車 步行約 10 分鐘抵達校本部。
4. 豐原客運 228 號，「大鵬國小站」下車 步行約 8 分鐘抵達校本部。
5. 台中客運 28 號，「大鵬國小站」下車 > 步行約 8 分鐘抵達校本部。
6. 仁友客運 32 號，「大鵬國小站」下車 步行約 8 分鐘抵達校本部。
7. 台中客運 6 號、23 號、54 號、82 號、101 號、108 號，「大德國中站」下車 步行約 10 分鐘抵達校本部。

『自行駕車』

*沿國道 3 號和國道一號前往西屯區的中清路二段/台 1 乙線 從國道一號的 174-大雅出口下交流道 走經貿路前往北屯區的大鵬路抵達水湳校本部。

校本部↔高鐵台中站公車轉乘路線

1. 校本部-逢甲大學：28、54、68、199、228 路
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3. 朝馬-高鐵台中站：151、153、155、160、161 路
4. 逢甲大學-高鐵台中站：33、160 路

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∞ 感謝上列廠商之支持 ∞

會場平面圖

台灣腎臟醫學會 113 年度春季學術演講會
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